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Assessment of Personal Care Product Exposure, Hair-Dye and Slimming Product Use and Their Associations with Blood Pb/Cd and Selected Biochemical and Hormonal Biomarkers among Women in Babylon Governorate, Iraq.

A Thesis

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1448 A.H.

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

يَرْفَعُ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ
وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ

صدق الله العلي العظيم

سورة المجادلة (الآية 11)

Dedication

I dedicate the fruit of this effort to:

To my support in life and my source of strength... My dear father. To the source of tenderness, whose prayers accompany me and illuminate my path... My beloved mother. To my pride and support in every step... My dear brothers. To my lifelong companion and the heartbeat of my soul... My beloved sister. To those who shared the details of this journey and were the best help and support... My dear friends. And to everyone who stood by me, contributed to helping me, or supported me, even with a kind word along the way.

To all of you, I dedicate this work.

ZAINAB

Acknowledgment



*In the Name of God, the Most Gracious, the Most Merciful.
Praise be to God, by whose grace and guidance I have completed
this work. As a matter of gratitude and acknowledgment, I would
like to express my sincere thanks and deepest appreciation to my
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profound impact on the completion of this research.
I would also like to express my deep gratitude to all the esteemed
doctors and professors of the department for the knowledge they
have imparted to us throughout my studies.*

ZAINAB

Summary

This study investigated the associations between cosmetics, hair dyes, and slimming-product exposure patterns and systemic biochemical, inflammatory, endocrine, nutritional, and whole-blood metal biomarkers among adult women in Babylon Governorate, Iraq. An analytical cross-sectional environmental biomonitoring study was conducted among 120 participants (mean age: 34.38 ± 7.90 years; mean BMI: 26.56 ± 3.33 kg/m²). Participants were classified into hair-dye-only (n=34), slimming-product-only (n=30), and combined hair-dye and slimming-product exposure groups (n=56). Exposure intensity was quantified using CEI, HDEI, SPEI, and PPEI.

Serum CRP and multiple biochemical, hepatic, renal, endocrine, haematological, and nutritional biomarkers were evaluated, together with whole-blood Pb and Cd concentrations. The mean CRP concentration was 6.02 ± 3.53 mg/L, while mean Pb and Cd levels were 14.3 ± 6.8 µg/L and 0.608 ± 0.763 µg/L, respectively. Statistical analysis included non-parametric comparisons, Spearman correlation, Benjamini–Hochberg false-discovery-rate correction, and multivariable regression adjusted for age, BMI, and menstrual phase.

The strongest inflammatory association was observed between cosmetic-product exposure and CRP. CEI showed a significant positive correlation with CRP ($\rho=0.278$, $p=0.002$, $q=0.013$), while PPEI showed a stronger positive correlation ($\rho=0.327$, $p<0.001$, $q=0.002$). In adjusted regression models, CEI remained positively associated with $\log(\text{CRP}+0.1)$ ($\beta=0.079$, 95% CI: 0.012–0.145, $p=0.021$), and PPEI also showed a significant association ($\beta=0.039$, 95% CI: 0.011–0.067, $p=0.007$). When exposure indices were modelled simultaneously, CEI and SPEI remained significant predictors, whereas HDEI showed no independent association.

All Pb measurements exceeded the quantification limit. Although initial analyses indicated associations between Pb/Cd and some exposure patterns, these findings were not stable after sensitivity analysis. Following exclusion of the separate 20-sample metal batch, Pb and Cd group differences became non-significant (Pb: $p=0.953$; Cd: $p=0.347$), and metal-related correlations were attenuated. Therefore, Pb and Cd findings were interpreted as exploratory observations rather than evidence of product-specific metal exposure. Additionally, no natural-versus-commercial product differences remained significant after FDR adjustment.

In conclusion, this study demonstrates that personal-care product exposure patterns, particularly cosmetic exposure quantified by CEI, are associated with variation in systemic inflammatory biomarkers among women in the studied population. However, the cross-sectional design and absence of a fully unexposed control group limit causal interpretation. Further investigations incorporating direct product analysis, environmental sampling, validated exposure assessment, repeated biomarker measurements, and longitudinal follow-up are required to clarify exposure sources and biological implications.

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List of Abbreviations and Symbols



Abbreviation	Full Term / Meaning
Adjusted R ²	Adjusted Coefficient of Determination
ALP	Alkaline Phosphatase
ALT	Alanine Aminotransferase
As	Arsenic
AST	Aspartate Aminotransferase
BD	Becton Dickinson
BMI	Body Mass Index
Cd	Cadmium
CEI	Cosmetic Exposure Index
CI	Confidence Interval
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
Cr	Chromium
CRP	C-Reactive Protein
CV	Coefficient of Variation
eGFR	Estimated Glomerular Filtration Rate
FDR	False Discovery Rate
FSH	Follicle-Stimulating Hormone
FT3	Free Triiodothyronine
FT4	Free Thyroxine
GFAAS	Graphite Furnace Atomic Absorption Spectrometry
GFR	Glomerular Filtration Rate
H	Kruskal–Wallis H Statistic
H ₂ O ₂	Hydrogen Peroxide
Hb	Haemoglobin
HDEI	Hair-Dye Exposure Index
Hg	Mercury
HNO ₃	Nitric Acid
IQR	Interquartile Range

Abbreviation	Full Term / Meaning
K ₂ EDTA	Dipotassium Ethylenediaminetetraacetic Acid
LH	Luteinizing Hormone
LOD	Limit of Detection
LOQ	Limit of Quantification
n	Analytical or Subgroup Sample Size
N	Total Sample Size
Ni	Nickel
p	Probability Value
Pb	Lead
PPD	p-Phenylenediamine
PPEI	Personal Product Exposure Index
q	False-Discovery-Rate-Adjusted p-Value
Q1	First Quartile
Q3	Third Quartile
QA	Quality Assurance
QC	Quality Control
R ²	Coefficient of Determination
SD	Standard Deviation
SNIBE	Shenzhen New Industries Biomedical Engineering Co., Ltd.
SPEI	Slimming Product Exposure Index
SPSS	Statistical Package for the Social Sciences
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
STROBE-ME	Strengthening the Reporting of Observational Studies in Epidemiology–Molecular Epidemiology
TSH	Thyroid-Stimulating Hormone
U	Mann–Whitney U Statistic
β	Regression Coefficient
ε ²	Epsilon-Squared Effect Size
ρ	Spearman Correlation Coefficient

Chapter One

Introduction

& Literature Review

1.1 INTRODUCTION

Personal-care products represent an important component of modern daily life and are widely used for hygiene, cosmetic enhancement, and body care. The extensive use of cosmetics, hair dyes, and other personal-care products has increased scientific interest regarding their contribution to human exposure to chemical substances. These products may contain various chemical constituents, including preservatives, fragrances, plasticizers, UV filters, pigments, antimicrobial agents, and trace contaminants, some of which have been investigated as potential endocrine-disrupting or biologically active compounds (Den Hond *et al.*, 2013; Aylward *et al.*, 2020).

Environmental health research increasingly recognises that human exposure is not restricted to traditional environmental pollutants but also includes consumer-product-related pathways occurring within domestic, occupational, and personal environments. Therefore, personal-care products represent an important exposure domain requiring integrated approaches that combine exposure assessment with human biomonitoring to evaluate internal chemical burdens and potential biological responses (Den Hond *et al.*, 2013; Wang *et al.*, 2024).

Human biomonitoring provides a valuable framework for assessing internal exposure through measurement of chemicals or their metabolites in biological matrices. However, biomarker alterations require cautious interpretation because biological responses may be influenced by multiple factors, including age, body composition, hormonal status, lifestyle factors, medication use, and other environmental exposures (Gallo *et al.*, 2011; Den Hond *et al.*, 2013).

In Iraq, particularly in Babylon Governorate, information remains limited regarding personal-care product exposure patterns and their potential relationships with systemic inflammatory, biochemical, endocrine, and metal-related biomarkers. Therefore, this study was designed to evaluate associations between personal-care product use patterns and biological indicators within an environmental health biomonitoring framework.

1.2 Research problem and knowledge Gap

The increasing use of personal-care products, including cosmetics, hair dyes, and slimming products, has raised concerns regarding potential human exposure to diverse chemical substances. Although these products are widely used, available evidence regarding their relationships with systemic biological responses remains limited, particularly in populations from developing regions.

Previous studies have mainly focused on individual product categories or specific chemical constituents, while fewer investigations have evaluated combined exposure patterns involving multiple personal-care product domains. In addition, the distinction between product use behaviour and actual internal chemical exposure remains a major challenge in environmental health research.

In Iraq, particularly in Babylon Governorate, data regarding personal-care product exposure patterns and their associations with inflammatory, biochemical, endocrine, nutritional, and metal-related biomarkers are scarce. Furthermore, the contribution of personal-care products to internal Pb and Cd concentrations remains difficult to determine because these metals may originate from multiple environmental sources.

Therefore, there is a need for an integrated biomonitoring approach that combines exposure assessment, biomarker evaluation, and cautious interpretation of potential environmental pathways.

1.3 Aim of the Study

The aim of this study was to investigate the associations between personal-care product exposure patterns and systemic biological responses among adult women in Babylon Governorate, Iraq, using an environmental health biomonitoring framework. The study focused on evaluating whether differences in the use patterns of cosmetics, hair dyes, and slimming products were associated with

variations in inflammatory, biochemical, endocrine, nutritional, and metal-related biomarkers.

1.4 Objectives of the Study

The objectives of this study were to:

1. Develop and apply exposure indices for cosmetics (CEI), hair dyes (HDEI), slimming products (SPEI), and combined personal-care product exposure (PPEI).
2. Evaluate the association between personal-care product exposure patterns and systemic inflammatory status using serum CRP as the primary biomarker.
3. Investigate differences in biochemical, hepatic, renal, endocrine, nutritional, and haematological biomarkers among different exposure-pattern groups.
4. Assess whole-blood Pb and Cd concentrations and explore their relationships with personal-care product exposure patterns while considering potential uncertainty in source attribution.
5. Compare biomarker profiles and metal concentrations between self-reported natural and commercial product users.
6. Provide an environmental health biomonitoring framework for understanding personal-care product exposure patterns and their potential biological relevance in the studied population.

1.5 Hypotheses

Based on the study objectives and the available scientific evidence, the following hypotheses were proposed:

H1: Higher personal-care product exposure patterns are associated with increased systemic inflammatory status, reflected by higher serum CRP concentrations.

H2: Different exposure domains, including cosmetics, hair dyes, and slimming products, are associated with distinct patterns of biochemical, hepatic, renal, endocrine, and nutritional biomarker variation.

H3: Combined personal-care product exposure, represented by PPEI, provides additional information regarding overall exposure patterns beyond individual product categories.

H4: Whole-blood Pb and Cd concentrations may vary across exposure-pattern groups; however, these relationships require cautious interpretation because potential environmental sources cannot be completely separated.

H5: Self-reported natural and commercial product use categories may differ in biomarker profiles, although product origin alone may not represent a reliable indicator of chemical exposure or safety.

1.6 Significance of the study

The significance of this study lies in addressing an important gap in environmental health research by evaluating personal-care product exposure patterns and their potential relationships with biological responses among women in Babylon Governorate, Iraq.

This study contributes to the limited biomonitoring data available from Iraq by integrating behavioural exposure assessment with measurements of inflammatory, biochemical, endocrine, nutritional, and metal-related biomarkers. The development of exposure indices, including CEI, HDEI, SPEI, and PPEI, provides a structured approach for evaluating complex and overlapping personal-care product use patterns.

Furthermore, this research provides a cautious framework for interpreting Pb and Cd measurements within a multi-source environmental exposure context. Rather than attributing metal concentrations to a specific product source, the study considers these measurements as indicators of internal metal burden requiring further investigation.

The findings may support future environmental health studies, improve understanding of consumer-product-related exposure pathways, and provide a basis for more detailed investigations involving product analysis, environmental sampling, and longitudinal biomonitoring approaches.

Literature review

1.7 Cosmetic products as personal-care exposure sources

Cosmetic products are widely used worldwide for hygiene, appearance enhancement, and personal care. Their frequent application, often involving direct and repeated contact with the skin, hair, and mucosal surfaces, makes them an important pathway for human exposure to various chemical constituents. Depending on product type and formulation, cosmetics may contain preservatives, fragrances, pigments, solvents, ultraviolet filters, plasticizers, and other functional ingredients that may contribute to internal exposure through dermal absorption, inhalation, or accidental ingestion (Nohynek *et al.*, 2010; Bom *et al.*, 2019).

Several cosmetic-related chemicals have received scientific attention because of their potential biological activity. Certain preservatives, plasticizers, and fragrance compounds have been investigated for possible endocrine-disrupting, inflammatory, or oxidative stress-related effects. However, exposure levels, absorption rates, and biological relevance vary considerably depending on chemical characteristics, frequency of use, product formulation, and individual factors (Gore *et al.*, 2015).

Human biomonitoring studies have detected metabolites of cosmetic-related chemicals in biological samples, supporting the contribution of personal-care products to overall chemical exposure. Nevertheless, biomonitoring findings should be interpreted cautiously because measured internal concentrations may reflect multiple exposure sources, including diet, household environments, and other consumer products (Den Hond *et al.*, 2013).

In this context, evaluating cosmetic exposure requires consideration of both product-use behaviour and biological indicators. Therefore, the present study incorporated a cosmetic exposure index (CEI) to characterise cumulative cosmetic-use patterns and investigate their associations with systemic biomarkers.

1.8 Hair dyes as exposure sources in scalp and salon situations

Hair dyes are among the most commonly used personal-care products, particularly among women, for changing hair colour, covering grey hair, and modifying appearance. Their frequent and repeated use has raised interest regarding potential exposure to chemical substances through direct scalp contact, dermal absorption, inhalation of volatile compounds, and occupational exposure among hairdressers (de Sanjose *et al.*, 2006).

Permanent hair dyes generally contain oxidative agents and aromatic amine compounds that participate in colour formation. Some of these chemical constituents have been investigated due to their potential biological activity, including oxidative stress induction, immune modulation, and endocrine-related effects. However, the extent of biological relevance depends on exposure intensity, product formulation, application practices, and individual susceptibility (Gago-Dominguez *et al.*, 2001; IARC, 2010).

Epidemiological studies investigating hair dye exposure have reported variable findings regarding associations with adverse health outcomes. Differences between studies may be related to variations in dye composition, frequency of use, exposure duration, and the presence of other environmental or lifestyle factors. Therefore, hair-dye exposure should be interpreted within a broader exposure assessment framework rather than as an isolated chemical exposure source (IARC, 2010).

In addition to chemical constituents, informal or poorly regulated hair-dye products may represent an additional exposure concern because of possible variations in composition and quality control. However, product use alone does not provide direct information about absorbed doses or internal chemical concentrations.

Accordingly, the present study incorporated a hair-dye exposure index (HDEI) to evaluate cumulative hair-dye use patterns and explore their associations with biological markers within a human biomonitoring framework.

1.9 Appearance-related and potentially unregulated exposure sources

Slimming products represent a diverse category of weight-management products that include dietary supplements, herbal preparations, meal-replacement products, and topical formulations. The increasing popularity of these products has raised concerns regarding potential exposure to biologically active compounds, particularly when products are obtained from informal markets or lack adequate quality control.

Unlike conventional dietary interventions, some slimming products may contain multiple ingredients with variable pharmacological activity, including botanical extracts, stimulants, hormones, or undeclared substances. These components may influence metabolic pathways and may be associated with hepatic, renal, endocrine, or cardiovascular responses depending on their composition, dose, duration of use, and individual susceptibility (Navarro *et al.*, 2017).

Herbal and dietary supplements have been increasingly recognised as potential contributors to drug-induced liver injury. However, the risk is highly variable and depends on product authentication, contamination, adulteration, and the presence of specific bioactive compounds. Therefore, biological findings associated with slimming-product use require careful interpretation and cannot be attributed to a single mechanism without chemical characterisation of the products (Navarro *et al.*, 2017; Zheng & Navarro, 2015).

In addition, oral slimming products differ from topical personal-care products because they undergo gastrointestinal absorption, metabolism, and systemic

distribution. This makes hepatic and renal biomarkers particularly relevant when evaluating potential biological responses related to slimming-product exposure.

Accordingly, the present study incorporated a slimming-product exposure index (SPEI) to evaluate exposure patterns and explore their relationships with inflammatory, biochemical, hepatic, renal, and endocrine biomarkers.

1.10 Natural vs. commercial products: interpretation and cautions

The classification of personal-care products as natural or commercial has become increasingly common among consumers and is often associated with perceptions of safety and health benefits. However, product origin or marketing category does not necessarily reflect chemical composition, exposure level, or biological safety. Differences in product formulation, manufacturing practices, and quality control are more relevant determinants of potential exposure than product labels alone (Bom *et al.*, 2019; Rastogi *et al.*, 2018).

Natural products may contain biologically active compounds and naturally occurring contaminants depending on their source, processing methods, and quality control practices. Similarly, commercial products may contain regulated ingredients with established safety evaluations; however, variations in formulation and manufacturing quality may influence exposure profiles (Nohynek *et al.*, 2010).

Previous studies have emphasized that safety assessment of personal-care products should be based on specific chemical constituents and exposure assessment rather than consumer-oriented classifications such as natural or commercial categories (Gore *et al.*, 2015). Therefore, self-reported product classification provides information regarding consumer behaviour and product preference but does not represent a direct measurement of chemical exposure or toxicity risk.

In the present study, natural versus commercial classification was considered as an exploratory comparison to evaluate whether reported product origin was associated with differences in biomarker profiles and metal concentrations.

1.11 Heavy metals in products and broader environmental exposure

Heavy metals such as lead (Pb) and cadmium (Cd) are environmental contaminants of concern because of their persistence, potential accumulation in biological systems, and adverse biological effects. The classification of personal-care products as natural or commercial has become increasingly common. These metals may occur in consumer products as intentional ingredients, impurities, or contaminants resulting from raw materials and manufacturing processes (Al-Saleh *et al.*, 2009; Bocca *et al.*, 2014).

Previous investigations have reported the presence of Pb, Cd, and other trace metals in cosmetic products and related personal-care products. Potential exposure may occur through dermal contact, accidental ingestion, or inhalation; however, the detection of metals in products does not necessarily demonstrate that these products represent the dominant source of internal human exposure (Bocca *et al.*, 2014; Sainio *et al.*, 2000).

Human exposure to Pb and Cd is influenced by multiple environmental pathways, including dietary intake, drinking water, household dust, occupational environments, and consumer products. Therefore, interpretation of whole-blood metal concentrations requires consideration of the broader environmental exposure context rather than attribution to a single source (ATSDR, 2020; Tchounwou *et al.*, 2012).

Human biomonitoring of Pb and Cd provides valuable information regarding internal metal burden and exposure patterns. Nevertheless, accurate source

attribution requires integration of biomonitoring with product analysis and environmental sampling approaches (Needham *et al.*, 2007).

In this study, whole-blood Pb and Cd concentrations were evaluated as indicators of internal metal burden and interpreted within a multi-source environmental exposure framework.

1.12 Biomonitoring of humans and the interpretation of biomarkers

Human biomonitoring (HBM) is an established approach in environmental health research that evaluates internal exposure to environmental chemicals by measuring parent compounds, metabolites, or biological responses in human samples such as blood, urine, or other biological matrices. Unlike external exposure assessment, biomonitoring provides information regarding the amount of a substance that has entered the body and may reflect the combined contribution of multiple exposure pathways (Needham *et al.*, 2007; Ganzleben *et al.*, 2017).

Biomarkers used in human biomonitoring studies can be classified into exposure biomarkers, effect biomarkers, and susceptibility biomarkers. Exposure biomarkers indicate the presence or concentration of chemicals within biological systems, whereas effect biomarkers reflect biological responses that may occur following exposure. However, biological responses are not necessarily specific to a single exposure source and may be influenced by multiple physiological and environmental factors (Gallo *et al.*, 2011).

In environmental health studies, inflammatory, biochemical, endocrine, and metabolic biomarkers are frequently used to investigate potential biological responses associated with environmental exposures. Serum C-reactive protein (CRP), for example, is widely used as an indicator of systemic inflammatory status; however, it is a non-specific marker influenced by several factors, including infection, obesity, lifestyle, hormonal status, and medication use (Pepys & Hirschfield, 2003).

Similarly, hepatic, renal, endocrine, and nutritional biomarkers provide valuable information regarding biological variation but require cautious interpretation because changes may result from multiple interacting factors rather than a single exposure source. Therefore, biomarker findings should be interpreted within an integrated exposure framework rather than as direct evidence of chemical toxicity (Ganzleben *et al.*, 2017).

In the present study, human biomonitoring was applied to evaluate inflammatory, biochemical, endocrine, nutritional, and metal-related indicators in relation to personal-care product exposure patterns. This approach enables assessment of potential biological associations while acknowledging the complexity of environmental exposures. The conceptual framework linking personal-care product domains, exposure pathways, exposure indices, biological matrices, measured outcomes, and interpretive boundaries is illustrated in Figure 1.1.

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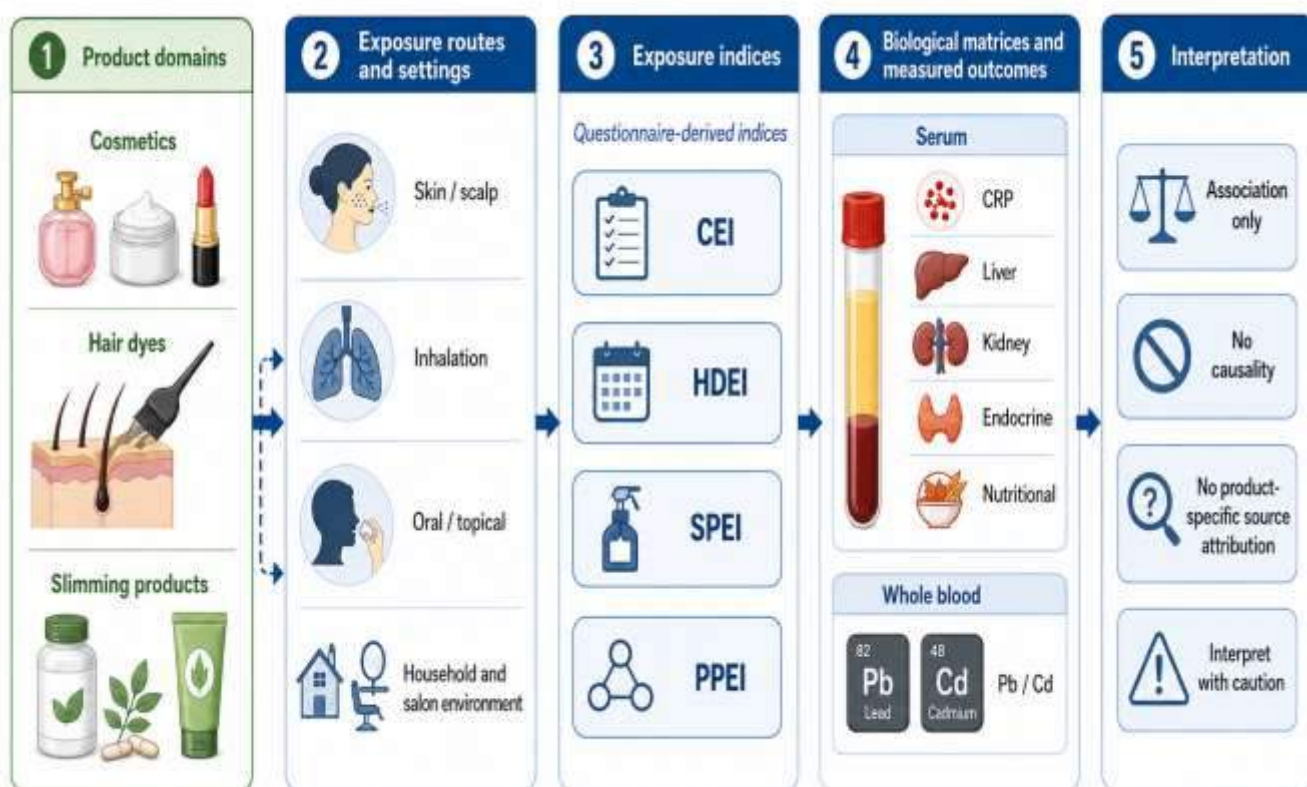


Figure 1.1 The conceptual framework linking personal-care product domains, exposure pathways, exposure indices, biological matrices, measured outcomes, and interpretive boundaries .

As shown in Figure 1.1, the separation between exposure indices and biological outcomes allows interpretation of statistical associations without assuming direct causality. Therefore, observed relationships between product-use indices and biomarkers should be considered as evidence of exposure–response patterns rather than definitive proof of product toxicity or confirmation of product-specific sources of Pb and Cd. Source attribution requires additional evidence from paired product analysis and environmental measurements (Needham *et al.*, 2007; Angerer *et al.*, 2007; CDC, 2022).

1.13 Biological Markers Relevant to the Study

Biomarkers are measurable biological indicators that provide information about physiological processes, exposure-related responses, or alterations in organ function. In environmental health research, biomarkers are widely used to investigate whether chemical or behavioural exposures are associated with measurable biological changes.

C-reactive protein (CRP) is an acute-phase inflammatory biomarker produced mainly by the liver in response to inflammatory stimuli. Although CRP is not specific to a particular exposure source, it is commonly used as an indicator of systemic inflammatory status and has been applied in studies investigating relationships between environmental exposures, lifestyle factors, and chronic low-grade inflammation (Calder *et al.*, 2013).

Hepatic biomarkers, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and bilirubin, are commonly used to evaluate alterations related to liver function. These markers are particularly relevant when assessing exposure to orally consumed products, such as some slimming products, because these products undergo absorption and metabolism through hepatic pathways. However, changes in liver enzymes should be interpreted cautiously because they may result from multiple factors, including medications, diet, metabolic conditions, and other environmental exposures (Navarro *et al.*, 2017).

Renal biomarkers, including urea, creatinine, and estimated glomerular filtration rate (eGFR), provide information regarding kidney filtration and function. They are relevant in environmental biomonitoring studies because the kidneys play an important role in the elimination of many chemicals and metabolites. Nevertheless, these indicators represent functional changes and do not independently confirm specific toxic effects without additional clinical evidence (Levey *et al.*, 2009).

Endocrine and nutritional biomarkers, including thyroid hormones, reproductive hormones, ferritin, and vitamin D, were considered relevant because hormonal regulation and nutritional status may be influenced by environmental exposures, physiological conditions, and lifestyle factors. Interpretation of these markers requires consideration of biological variability, including age, menstrual phase, diet, and medication use (Gore *et al.*, 2015).

In the present study, these biomarkers were integrated with exposure indices to evaluate potential associations between personal-care product exposure patterns and biological responses.

1.14 Exposure Indices and Assessment of Personal-Care Product Use Patterns

Assessment of personal-care product exposure is challenging because individuals are usually exposed to multiple products with different formulations, frequencies of use, and application methods. Traditional exposure classification based only on individual product use or exposed versus non-exposed categories may not adequately represent complex real-world exposure scenarios (Vrijheid, 2012; Wild, 2005).

Exposure indices provide a quantitative approach for integrating behavioural information, including product type, frequency, duration, and intensity of use, into measurable exposure indicators. Such approaches are increasingly applied in environmental epidemiology when direct measurement of all chemical constituents or absorbed doses is not feasible (Nieuwenhuijsen, 2015).

In studies involving consumer-product exposure, domain-specific indices allow assessment of different exposure sources separately while also considering combined exposure patterns. This approach is particularly relevant because cosmetics, hair dyes, and slimming products may involve different chemical compositions and exposure pathways (Den Hond *et al.*, 2013).

However, exposure indices represent estimated patterns of product use rather than direct measurements of internal chemical concentrations. Therefore, interpretation of exposure indices requires integration with human biomonitoring data and biological markers to evaluate potential relationships between external exposure patterns and internal biological responses (Needham *et al.*, 2007).

In the present study, four exposure indices were developed: cosmetic exposure index (CEI), hair-dye exposure index (HDEI), slimming-product exposure index (SPEI), and personal-care product exposure index (PPEI). These indices were applied to evaluate associations between product-use patterns and inflammatory, biochemical, endocrine, nutritional, and metal-related biomarkers.

Table 1.1. Product domains, exposure pathways, major concerns, and biomarker relevance

Product domain / index	Main exposure pathways	Major exposure concerns	Relevant biomarkers / analytical outcomes
Cosmetic products (CEI)	Dermal, inhalation, incidental oral transfer	Fragrances, preservatives, phthalates/parabens, pigments, and trace metals	CRP, endocrine markers, whole-blood Pb/Cd
Hair dyes (HDEI)	Scalp dermal contact, salon handling, wash-off residues	PPD, resorcinol, ammonia, oxidising agents, pigments, and potential metal impurities	CRP, inflammatory signals, whole-blood Pb/Cd
Slimming products (SPEI)	Oral or topical, depending on product	Herbal mixtures, adulterants, undeclared drugs, and contaminants	ALT, AST, ALP, urea, creatinine, endocrine markers, CRP
Combined exposure (PPEI)	Multiple routes and concurrent product-use behaviours	Mixed and potentially cumulative exposure patterns	Overall exposure-pattern signal, CRP, and exploratory biomarker panel; not source attribution

1.15 Environmental Pathways Other Than Direct Body Exposure

The environmental relevance of appearance-related products extends beyond direct application to the user. Cosmetics, hair dyes, and slimming products may contribute to household and environmental exposure through pathways including volatilisation, particle deposition, spills, storage, contaminated applicators, discarded packaging, and residues on textiles and household surfaces (Guo & Kannan, 2013; Csiszar *et al.*, 2016). These pathways support considering personal-care products as components of household micro-environments rather than solely as individual behavioural exposures (Weschler & Nazaroff, 2017; Steinemann, 2019).

Wastewater represents another relevant pathway, particularly for rinse-off cosmetics and hair dyes. During bathing, hair washing, and salon activities, chemical residues such as dyes, surfactants, preservatives, fragrances, pigments, and botanical compounds may enter wastewater systems. Variations in wastewater treatment efficiency may influence the environmental distribution of these substances (Daughton & Ternes, 1999; Fatta-Kassinos *et al.*, 2011).

Household dust may also act as an indirect pathway linking personal-care product use with environmental exposure. Product-derived particles and semi-volatile compounds may accumulate in indoor dust, while outdoor sources may introduce metals and combustion-related contaminants into indoor environments (Adamkiewicz *et al.*, 2011; Weschler & Nazaroff, 2017). Therefore, personal-product exposure may occur within a complex environment containing multiple concurrent sources.

Salon environments represent an additional exposure context, particularly for hair dyes and cosmetic products used repeatedly in enclosed spaces. Exposure among professional workers may differ from consumer-level exposure; therefore, occupational salon exposure should be considered as a potential confounding factor when interpreting biomonitoring findings (Symanzik *et al.*, 2022; Kezic *et al.*, 2022; Kaikiti *et al.*, 2022).

Product storage conditions, packaging, and supply-chain quality may further influence potential exposure, particularly for imported or poorly regulated products. Variations in labelling, ingredient information, and quality control highlight the importance of product authentication and future market surveillance, especially in regions where formal and informal markets coexist (Roy *et al.*, 2023; Ketan *et al.*, 2024; European Commission, 2024).

1.16 Behavioural, Social, and Gender-Related Exposure Considerations

Personal-care product use among women is influenced by various behavioural and social factors, including cultural practices, age, marital and employment status, income, media influence, and product availability. Cosmetics, hair dyes, and slimming products may be used for aesthetic preferences, social occasions, occupational requirements, grey-hair coverage, body-image concerns, or peer influence. These factors contribute to exposure patterns by affecting product frequency, duration, source, and combinations of use (Rodgers *et al.*, 2024; Rosenberg *et al.*, 2026).

Socioeconomic factors may also influence personal-care product selection and exposure characteristics. Differences in income, access to regulated products, and purchasing from formal or informal markets may affect product availability and potential exposure profiles. However, product price, brand status, or marketing category alone cannot be considered reliable indicators of safety or hazard without chemical characterisation (Rodgers *et al.*, 2024; Rosenberg *et al.*, 2026).

Body-image perceptions and social-media influences may contribute particularly to the use of slimming products. These products are often marketed as natural, herbal, or safe alternatives, which may influence consumer perceptions and patterns of use. Therefore, exposure assessment should consider not only product use but

also route, frequency, duration, and reliability of reported information (Brasil et al., 2024; LaMotte *et al.*, 2024; Lai *et al.*, 2024; El-Hadidy *et al.*, 2025).

Hair-dye use is also influenced by age, personal preferences, and social context. Differences between salon and home application may affect exposure conditions, including preparation methods, application duration, ventilation, scalp contact, and professional handling. These variations support the use of a quantitative hair-dye exposure index (HDEI) rather than a simple user/non-user classification (Symanzik *et al.*, 2022; Kaikiti *et al.*, 2022; Uter *et al.*, 2023).

1.17 Rationale for Conservative Interpretation and Multiple-Testing Control

Environmental biomonitoring studies involving multiple exposure indicators and biological outcomes require careful interpretation because observed associations may reflect complex interactions between exposure patterns, individual characteristics, and other environmental factors. Questionnaire-derived exposure indices represent behavioural patterns of product use, whereas biomarkers such as CRP, biochemical parameters, and whole-blood Pb/Cd concentrations represent biological responses or internal burden indicators. Therefore, these measures may identify exposure-related patterns but cannot independently establish product toxicity, absorbed dose, or specific source attribution (WHO Regional Office for Europe, 2023; Nakayama *et al.*, 2023).

The evaluation of multiple biomarkers and exposure variables increases the possibility of false-positive statistical findings. Accordingly, reliable interpretation requires appropriate control of multiple comparisons, consideration of effect direction and biological plausibility, and clear distinction between consistent findings and exploratory observations (Sarkar & Nandi, 2024; Westphal & Zapf, 2024).

1.18 Critical Summary of Selected Literature

The reviewed literature demonstrates that personal-care products can be investigated within an environmental health framework by integrating product-use behaviour, exposure pathways, human biomonitoring, and cautious interpretation of source attribution. Previous studies have provided evidence regarding chemical constituents in cosmetics, hair dyes, and slimming products, while biomonitoring investigations have demonstrated that internal biological measurements can reflect exposure-related variations without necessarily identifying specific sources.

Table 1.2 summarises selected studies that contributed to the design and interpretation of the present thesis. The reviewed evidence highlights three complementary levels of understanding. First, product-based studies demonstrate that personal-care products may contain metals, preservatives, or other chemical constituents of potential concern. Second, human biomonitoring studies indicate that biological markers may vary with exposure patterns but require careful interpretation because internal measurements may reflect multiple exposure sources. Third, reporting frameworks such as STROBE and STROBE-ME provide guidance for transparent reporting of observational biomarker studies.

The present thesis integrates these evidence domains by combining behavioural exposure assessment, biomarker evaluation, and internal metal-burden measurements. However, these components are interpreted separately to avoid unsupported conclusions regarding product-specific toxicity or direct source attribution.

Table 1.2 Critical summary of selected literature and its relevance to the present thesis

Author / year	Country or scope	Study design / sample / material	Principal finding	Relevance and main limitation for the present thesis
A. Empirical, intervention, and biomonitoring studies				
Guo & Kannan (2013)	United States	Analysis of personal-care products	Phthalates and parabens were detected across several product categories.	Supports the environmental-product exposure rationale for cosmetics; product-level detection does not establish internal dose in the present participants.
Dewalque et al. (2014)	Belgium	Urinary biomonitoring of parabens, benzophenone-3, and phthalates	Human biomonitoring was able to characterise exposure to common personal-care chemicals.	Supports biomonitoring and matrix-specific interpretation; urinary organic biomarkers reflect a different matrix and exposure window from serum CRP and whole-blood metals.
Harley et al. (2016)	United States	HERMOSA intervention among adolescent girls	Reducing use of selected personal-care products lowered urinary biomarkers of some phthalates, parabens, and phenols.	Supports product-use behaviour as a meaningful exposure domain; the adolescent intervention and urinary biomarkers are not directly equivalent to adult serum CRP or whole-blood metals.
Al-Azzawi & Al-Hili (2017)	Iraq	Facial cosmetics obtained from local markets	Cadmium and chromium were detected in some facial cosmetic products.	Provides local Iraqi evidence supporting cosmetic-metal surveillance; it is a product-contamination study rather than human biomonitoring evidence.
Khazaal et al. (2018)	Iraq	Lipstick brands sold in local markets	Lead and chromium were detected in selected lipstick brands.	Supports the local plausibility of cosmetic metal contamination; it does not show that participants' whole-blood Pb/Cd originated from lipstick use.

Author / year	Country or scope	Study design / sample / material	Principal finding	Relevance and main limitation for the present thesis
Alam et al. (2019)	Bangladesh	Selected cosmetic products assessed for heavy metals and health risk	Heavy metals were detected in commonly used cosmetic products, with concentrations varying by product.	Supports the plausibility of product contamination; product measurements alone cannot identify the source of whole-blood metal burden.
Berger et al. (2019)	United States	HERMOSA product-use questionnaire linked to urinary biomarkers	Reported product use predicted selected urinary phthalate, paraben, and phenol concentrations.	Supports CEI as a behavioural ranking score; it does not validate CEI as a direct chemical dose or as a predictor of CRP or Pb/Cd.
Khalili et al. (2019)	Iran	Chemical hair-dye products assessed for heavy metals and dermal exposure risk	Heavy metals were evaluated in hair dyes in relation to potential dermal exposure.	Supports examining HDEI in relation to whole-blood Pb/Cd; product analysis cannot establish the source of participants' blood metal concentrations.
Mohammed et al. (2023)	Iraq	Heavy-metal assessment of lipsticks sold in Iraqi markets	Several metals were measured and evaluated in lipstick products.	Supports the local rationale for investigating Pb/Cd burden; product contamination and human internal burden answer different questions.
Yousif et al. (2024)	Iraq	Environmental assessment of heavy metals in road dust	Road dust contained heavy metals and was identified as a potential environmental-health concern.	Supports road dust as a plausible alternative source of Pb/Cd; road-dust exposure was not measured directly in the present participants.

B. Reviews, reporting guidance, and conceptual frameworks

Author / year	Country or scope	Study design / sample / material	Principal finding	Relevance and main limitation for the present thesis
Wild (2005)	International	Conceptual exposome framework	The exposome concept emphasises cumulative environmental exposures over the life course.	Supports the broader mixed-exposure framing; it does not directly validate the questionnaire-derived exposure indices used in this study.
von Elm et al. (2007)	International	STROBE reporting guideline for observational studies	Defines essential reporting items for cross-sectional, case-control, and cohort studies.	Supports transparent reporting of design, participant selection, variables, bias, statistical methods, and limitations; it is reporting guidance rather than exposure evidence.
Gallo et al. (2011)	International	STROBE-ME extension for molecular epidemiology and biomarker studies	Emphasises transparent reporting of biological matrices, laboratory methods, biomarkers, confounding, and missing data.	Supports the thesis reporting structure and interpretive safeguards; it does not provide evidence of product toxicity.
Den Hond et al. (2015)	European / international	Human biomonitoring framework and interpretation	Human biomonitoring integrates multiple exposure pathways but does not automatically identify exposure sources.	Supports interpreting CRP and Pb/Cd as biological-response or internal-burden indicators; source attribution requires complementary product and environmental testing.
Zheng & Navarro (2015)	Not country-specific	Clinical hepatology review of herbal, dietary, and weight-loss supplements	Selected weight-loss and dietary supplements have been implicated in reports of liver injury.	Supports exploration of liver markers in relation to SPEI; clinical case evidence does not establish a population-level causal effect.

Author / year	Country or scope	Study design / sample / material	Principal finding	Relevance and main limitation for the present thesis
Mukkanna et al. (2017)	Not country-specific	Clinical review of p-phenylenedia mine allergy	PPD is an important hair-dye sensitiser and may cause allergic reactions.	Supports cautious interpretation of inflammatory plausibility; the evidence mainly concerns sensitisation and local allergic effects.
Attard & Attard (2022)	International	Review chapter on heavy metals in cosmetics	Summarizes the occurrence, sources, and potential health relevance of heavy metals in cosmetic products.	Supports the cosmetic-metal rationale; as a secondary source, it cannot establish internal burden or source attribution in the current participants.
He et al. (2022)	International	Review of hair-dye ingredients and health-risk evidence	Oxidative and non-oxidative hair dyes contain diverse ingredients with different exposure concerns.	Supports HDEI as a separate exposure domain; review evidence does not demonstrate absorbed dose or systemic effects in the present cohort.
Gyamfi et al. (2024)	International	Systematic review of heavy-metal contamination in cosmetics	Multiple metals were reported across diverse cosmetic categories, with substantial variation between products and settings.	Strengthens the product-contamination rationale; heterogeneous product studies cannot by themselves explain participants' whole-blood Pb/Cd.
Phan et al. (2025)	International	Systematic review and meta-analysis of natural weight-loss products	Natural or herbal weight-loss products may contain undeclared pharmaceuticals or adulterants.	Supports SPEI and caution in natural-versus-commercial comparisons; the products used by the present participants were not chemically verified.

C. Thesis and dissertation comparators

Moraa (2014)	Kenya	Master's thesis on aloe-vera	Selected heavy metals were	Supports the value of local market surveillance and
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		branded skin cosmetics	measured in locally marketed skin-cosmetic products.	cautious interpretation of natural or branded products; evidence remains product-based.
Rusmadi (2016)	Malaysia	Master's thesis on skin-lightening products and young adult women	Nickel, cadmium, and lead were evaluated in cosmetic products with dermal health-risk interpretation.	Useful as a structural and methodological comparator for cosmetic-metal assessment; not treated as primary evidence for the present biological associations.
Gkionakis (2020)	Greece	MSc dissertation on human biomonitoring and health-impact assessment	Human biomonitoring was organised around exposure pathways, biological matrices, and biomarkers of exposure or effect.	Provides a methodological comparator for separating behavioural indices, biological responses, and internal burden; not primary evidence for toxicity.

CHAPTER TWO

MATERIALS & METHODS

2.1 Equipment and instruments

Table 2.1 Equipment and instruments used in the study

No	Device name	Company	Country
1	Sterile syringes and vacutainer holder (adapter)	Weigao Group / Becton Dickinson (BD)	China / USA
2	Serum separator tubes and K ₂ EDTA whole-blood tubes	Kindly Group / Becton Dickinson (BD)	China / USA
3	Centrifuge	Sigma Laborzentrifugen GmbH	Germany
4	Sensitive balance	Sartorius	Germany
5	Cobas c311 clinical chemistry analyser MAGLUMI 600 chemiluminescence immunoassay analyser Sysmex XS-1000i haematology analyser	Roche Diagnostics Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE) Sysmex Corporation	Switzerland China Japan
6	novAA 400 P atomic absorption spectrometer operated in graphite-furnace mode (GFAAS)	Analytik Jena	Germany

2.2 Kits and chemicals

Table 2.2 Kits and chemicals used in the study

No.	Kit / Material	Company	Country
1	Routine clinical chemistry reagents: ALT, AST, ALP, urea, creatinine, total bilirubin, albumin, serum iron, and CRP	Roche Diagnostics	Switzerland
2	TSH	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
3	Free T3 (FT3)	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
4	Free T4 (FT4)	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
5	LH	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
6	FSH	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
7	Estradiol	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
8	Cortisol	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
9	Vitamin D	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China
10	Ferritin	Shenzhen New Industries Biomedical Engineering Co., Ltd. (SNIBE)	China

No.	Kit / Material	Company	Country
11	CBC/haemoglobin reagents	Sysmex Corporation	Japan
12	Pb calibration standard	Sigma-Aldrich (Merck KGaA)	Germany
13	Cd calibration standard	Sigma-Aldrich (Merck KGaA)	Germany
14	Concentrated nitric acid (HNO ₃ , 65%)	Laboratory-grade, local supplier	Iraq
15	Hydrogen peroxide (H ₂ O ₂ , 30%)	Laboratory-grade, local supplier	Iraq
16	Nitric acid washing solution (HNO ₃ , 10%)	Laboratory-grade, local supplier	Iraq
17	Instrument blank solution (HNO ₃ , 5%)	Laboratory-grade, local supplier	Iraq
18	Deionised water and acid-washed Teflon/PTFE digestion vessels	Laboratory-grade, local supplier	Iraq



Figure 2.1 Study workflow of participant recruitment, sample collection, laboratory analysis, and interpretation.

2.3 Study design

This analytical cross-sectional environmental-biomonitoring study examined associations between cosmetic-product, hair-dye, and slimming-product use and systemic biomarker variation among women aged 20–50 years in Babylon Governorate, Iraq. Exposure information and biological outcomes were measured at one time point between October 2025 and January 2026; temporality and causality therefore cannot be inferred. Because no independent unexposed group was recruited, the analyses represent within-population exposure-pattern comparisons and adjusted associations rather than exposed-versus-unexposed causal contrasts.

The analytical framework was documented before the final statistical analysis to reduce post hoc interpretation and improve transparency. The methodological structure followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement and the STROBE-ME extension for molecular epidemiology (Gallo et al., 2011; von Elm et al., 2007), which guided the definition of participants, classification of exposure, interpretation of biomarkers, reporting of laboratory quality control, statistical analysis, and reporting of limitations.

A major design principle was to disentangle product-use behaviour from measured biological markers. According to the questionnaire, cosmetic-product use, hair-dye use and slimming-product use were included as behavioural exposure domains, while CRP and routine biochemical markers were measured in serum and Pb/Cd were measured in digested whole-blood samples as internal metal-burden variables.

The study was hypothesis-generating. It examined associations between product-use patterns and inflammatory, biochemical, endocrine, nutritional, and metal-burden markers without asserting product causality or definitive source attribution.

2.4 Study setting

The study was conducted in Babylon Governorate, Iraq, between October 2025 and January 2026. Participant recruitment and routine laboratory analyses were conducted through Al-Qasim General Hospital and Imam Al-Sadiq Hospital, Babylon Health Directorate. Whole-blood Pb and Cd analyses were performed in a private laboratory within the governorate.

Cosmetic products, hair dyes, and slimming products were obtained from pharmacies, markets, beauty salons, online suppliers, and informal sellers; these sources may differ in labelling quality, storage conditions, authenticity, and contamination risk. Participants may also have encountered non-product sources such as road and household dust, variable drinking-water quality, traffic-related particles, and household-generator emissions (Khudhair et al., 2025; Ngai et al., 2022; Yousif et al., 2024). Product use was therefore treated as one component of a mixed exposure environment.

Venous blood samples were collected and processed into serum and whole-blood fractions according to the intended analytical purpose. Serum samples were used for the assessment of CRP and selected biochemical, endocrine, haematological, and nutritional biomarkers. Biochemical analyses were performed using automated clinical chemistry methods, while endocrine markers were evaluated using immunoassay-based procedures. CRP was selected as the primary inflammatory biomarker because of its sensitivity to systemic inflammatory variation, while recognising its non-specific nature (Pearson *et al.*, 2003; Ferguson *et al.*, 2014).

Whole-blood samples collected in K₂EDTA tubes were analysed for Pb and Cd concentrations using graphite furnace atomic absorption spectrometry (GFAAS). The analytical procedure included sample preparation, calibration using certified standards, and quality-control assessment through blanks, detection limits, and quantification limits. GFAAS is widely used for trace-metal determination in biological matrices because of its high sensitivity and suitability for low-concentration measurements (WHO Regional Office for Europe, 2023; Gyamfi *et al.*, 2024).

The generated laboratory data were integrated with questionnaire-derived exposure indices (CEI, HDEI, SPEI, and PPEI). Statistical analysis included non-parametric group comparisons, Dunn pairwise testing, Spearman correlation analysis, Benjamini–Hochberg false-discovery-rate adjustment, and multivariable regression modelling of log(CRP + 0.1). These procedures were selected to account for non-normal biomarker distributions, multiple comparisons, and potential confounding factors (Benjamini & Hochberg, 1995).

2.5 Study population and analytical sample

The analysis-ready dataset included 120 adult female participants aged 20–50 years from Babylon Governorate. All participants were women, and no participant outside this age range was included.

A convenience sampling strategy was used to recruit participants from Al-Qasim General Hospital, Imam Al-Sadiq Hospital, related laboratory settings, and community sources, with an effort to include women with diverse cosmetic-product, hair-dye, and slimming-product use behaviours.

The final dataset consisted of three mutually exclusive exposure-pattern groups: hair dye only (n=34), slimming product only (n=30), and hair dye plus slimming product (n=56). In overlapping exposure counts, 90 women reported hair-dye use and 86 reported slimming-product use. Cosmetic-product use was recorded separately, because it overlapped with these groups in most participants and was therefore treated as an overlapping product-use variable rather than an independent mutually exclusive category.

The final convenience sample comprised 120 participants and was used for the planned exploratory environmental-biomonitoring analyses, including non-parametric group comparisons, Spearman correlations, and limited adjusted regression models. No formal claim of definitive statistical power, clinical-risk estimation, or causal inference was made. Interpretation therefore emphasised effect direction, FDR-adjusted findings, regression consistency, and biological plausibility.

Table 2.3 Categories of exposure patterns and methodological interpretation

Exposure pattern/domain	Operational definition	n	Methodological interpretation
Hair dye only	Hair-dye use without slimming-product use; cosmetic use could overlap.	34	Mutually exclusive exposure-pattern group; not an unexposed control.
Slimming product only	Slimming-product use without hair-dye use; cosmetic use could overlap.	30	Mutually exclusive exposure-pattern group; not an unexposed control.
Hair dye + slimming	Concurrent hair-dye and slimming-product use; cosmetic use could overlap.	56	Combined exposure-pattern group representing multiple product domains.
Cosmetic-product use	Recorded separately as an overlapping domain.	108 yes; 10 no; 2 missing/unclear	Analysed mainly through CEI rather than as a fourth mutually exclusive group.
Total analytical sample	Adult women aged 20–50 years from Babylon Governorate.	120	Cross-sectional within-population comparison; no independent unexposed control group.

2.6 Ethical approval and informed consent

Ethical approval and administrative permission were obtained from the Babylon Health Directorate (Approval No. 4366, 14 September 2025). Recruitment and clinical-laboratory linkage were conducted through Al-Qasim General Hospital and Imam Al-Sadiq Hospital. Written informed consent was obtained from all participants before

questionnaire completion and venous blood sampling. All participant recruitment and blood sampling occurred after the documented ethical-approval date.

Personal identifiers were separated from the analytical dataset before analysis. Participants were informed about the study purpose, the voluntary nature of participation, confidentiality safeguards, and their right to withdraw from the study at any time. Laboratory results were used for research purposes only and not for clinical diagnosis of product toxicity, heavy-metal poisoning, renal disease, hepatic disease, or endocrine disease.

2.7 Inclusion and exclusion criteria

Participants were eligible if they were adult women aged 20–50 years from Babylon Governorate with available questionnaire information for the product-exposure domains and laboratory data for inflammatory, biochemical, and heavy-metal outcomes. The final analytic dataset included 120 women with adequate exposure and outcome information to support exposure-pattern grouping and product-exposure index calculation.

Before blood sampling, all participants completed a structured questionnaire to screen for potential confounding conditions. Women reporting smoking, chronic diseases, recent acute or subclinical infection, medication use that could influence biochemical or inflammatory markers, pregnancy, lactation, active malignancy, or severe occupational metal exposure were excluded from the study.

Menstrual-phase fluctuations, major dietary variation, and physical-activity variation were also considered during pre-sampling screening and interpretation. These criteria were used to reduce non-product-related influences on CRP, endocrine markers, liver/kidney markers, and blood Pb/Cd.

The screening for exclusion criteria was based on the structured pre-sampling questionnaire and available clinical/laboratory records. This screening reduced the influence of smoking, chronic disease, medication use, and recent infection on the measured biomarkers.

Table 2.4 Inclusion and exclusion logic for the analytical dataset

Criterion domain	Included / excluded condition	Reason
Eligibility	Adult female participant aged 20–50 years from Babylon Governorate	Defines the target population and local environmental context.
Exposure data	Recorded information on cosmetic products, hair dyes, or slimming products	Required for product-domain classification and exposure index calculation.
Laboratory data	Available CRP, biochemical, and metal-burden data	This is required for biomonitoring interpretation.
Inflammatory validity	Active smoking, recent acute/subclinical infections, chronic inflammatory disorders, and immunomodulatory medication use screened and strictly excluded prior to sampling	Eliminates confounding elevations in serum CRP levels, ensuring baseline inflammatory signals reflect appearance-related product exposure patterns.
Organ-function validity	Known chronic hepatic or renal disease considered exclusion or interpretive flag	These conditions may confound the interpretation of liver and kidney biomarkers.
Metal-source validity	Severe or direct occupational heavy-metal exposure (e.g., industrial fields) identified via screening and excluded.	Distinguishes consumer-level personal product exposure profiles from baseline industrial or high-dose occupational environmental body burdens
Slimming-product validity	Prescribed medical slimming drugs identified separately	Prescription drugs differ from unregulated or consumer slimming products.

2.8 Questionnaire-based exposure assessment

Exposure data were obtained using a structured questionnaire covering demographic information, anthropometric variables, product-use behaviour, product origin, duration and frequency of use, route of exposure, and selected potential confounders. Rather than relying solely on binary yes-or-no responses, the questionnaire collected information on exposure frequency, duration, route, and participant-reported product origin to support the construction of structured behavioural exposure indices.

The questionnaire was adapted from published biomonitoring and cosmetic-exposure assessment tools, including HERMOSA and related product-use instruments (Berger et al., 2019; Harley et al., 2016), and was modified for the Iraqi context following consultation with local environmental-health experts. Before full data collection, it was pilot-tested to assess clarity, comprehensibility, and cultural appropriateness.

The pilot assessment evaluated questionnaire clarity and cultural suitability but did not constitute a full psychometric validation study. Test–retest reliability, product-diary validation, photographic product-inventory comparison, and biomarker-based convergent validation were not performed. Questionnaire variables and the derived exposure indices were therefore interpreted as structured behavioural ranking measures rather than validated estimates of absorbed chemical dose.

For cosmetic products, the questionnaire collected information on the number of products used, frequency of application, duration of use, leave-on versus rinse-off status where available, product source, and participant-reported origin. Cosmetic products included skin-care preparations, make-up, lip products, perfumes, deodorants, sunscreens, creams, lotions, and related personal-care items. The CEI calculation used the number of products used per day and the duration of use in years; the remaining variables were retained for descriptive analyses.

For hair dyes, the questionnaire recorded hair-dye use, dye type, participant-reported product origin, use of natural or henna-based preparations, use of commercial preparations, frequency and duration of dyeing, salon versus home application where available, and likely scalp contact. Hair dyes were treated as a separate exposure domain because their formulations, scalp-contact patterns, aromatic-amine precursors, oxidising

agents, and salon-related exposure pathways differ from those of general cosmetic products. The HDEI calculation used annual dyeing frequency, duration of use in years, and the scalp-contact score; dye type, origin, and place of application were retained as descriptive variables.

For slimming products, the questionnaire recorded product type, route of exposure, frequency and duration of use, product source, label status where available, and whether the product was reported as topical, oral, herbal or natural, commercial or prepared, or mixed. This distinction was maintained because topical and oral slimming products involve different exposure routes and may have different relevance to hepatic, renal, endocrine, and inflammatory biomarkers. The SPEI calculation used the route score, weekly use frequency, and duration of use in months; product type, source, label status, and origin were retained as descriptive variables.

Menstrual-phase information was recorded through the structured questionnaire. Menstrual phase was classified as follicular or ovulatory according to the participant-reported cycle day and the predefined classification rule used in the study questionnaire. Blood sampling was not standardised to a fixed menstrual-cycle day or a single cycle phase. Menstrual phase was therefore included as a categorical covariate in the adjusted CRP regression models, with the follicular phase used as the reference category. Hormonal biomarkers, particularly LH, FSH, estradiol, and cortisol, were interpreted as exploratory because single measurements may be influenced by cycle timing and other physiological factors.

The questionnaire captured product-use behaviour and participant-reported product categories but did not chemically verify product ingredients, contaminants, adulterants, or authenticity. Questionnaire-derived exposure variables were therefore interpreted as behavioural exposure indicators rather than direct measures of absorbed chemical dose or product-specific toxicity.

Table 2.5 Product-domain questionnaire items, main exposure routes, and key methodological concerns

Product domain	Key questionnaire items	Main route
Cosmetic products	Products/day; years of use; frequency; leave-on/rinse-off; source; reported origin	Dermal, inhalation, oral transfer
Hair dyes	Dye type; natural/henna or commercial; frequency/year; years of use; salon/home; scalp contact	Scalp dermal contact, salon handling, wash-off residues
Slimming products	Type; route; frequency; duration; source; label status; natural/herbal or commercial/prepared	Topical or oral depending on product

2.9 Product exposure indices (CEI, HDEI, SPEI, PPEI)

Four questionnaire-derived exposure indices were developed to quantify different domains of appearance-related product use: the Cosmetic Exposure Index (CEI), Hair-Dye Exposure Index (HDEI), Slimming Product Exposure Index (SPEI), and Personal Product Exposure Index (PPEI). PPEI was considered a secondary combined exposure measure and was not used as a replacement for the independent interpretation of CEI, HDEI, and SPEI.

The indices were calculated using spreadsheet-based equations as follows:

1. Cosmetic Exposure Index (CEI)

$$CEI = \frac{N_c \times D_c}{10}$$

Where:

N_c = Number of cosmetic products used per day

D_c = Duration of cosmetic-product use (years)

10 = Scaling factor used only for numerical rescaling purposes

2. Hair-Dye Exposure Index (HDEI)

$$\text{HDEI} = \frac{\text{Fh} \times \text{Dh} \times \text{Sc}}{10}$$

Where:

Fh = Hair-dyeing frequency (applications per year)

Dh = Duration of hair-dye use (years)

Sc = Scalp-contact score

The scalp-contact score was coded as:

Scalp-contact category Score

No scalp contact	0
Low scalp contact	1
Moderate scalp contact	2
High scalp contact	3

3. Slimming Product Exposure Index (SPEI)

$$\text{SPEI} = \frac{\text{Rs} \times \text{Fs} \times \text{Ds}}{10}$$

Where:

Rs = Slimming-product route score

Fs = Frequency of slimming-product use (uses per week)

Ds = Duration of slimming-product use (months)

The slimming-product route score was coded as:

Exposure route	Score
No slimming-product use	0
Topical use	1

Exposure route	Score
Oral use	2
Combined topical and oral use	3

4. Personal Product Exposure Index (PPEI)

$$\text{PPEI} = \text{CEI} + \text{HDEI} + \text{SPEI}$$

Where:

PPEI represents the overall appearance-related product-use pattern by combining the three domain-specific exposure indices.

The division factor (**10**) was applied only to standardise the numerical values of the indices and does not represent a physical exposure unit, absorbed chemical dose, or internal biomarker concentration. Additional questionnaire variables, including weekly cosmetic application frequency, leave-on/rinse-off classification, product source and origin, hair-dye type and application location, and slimming-product type, source, label status, and origin, were retained for descriptive and subgroup analyses but were not included as independent components in the index equations.

The duration unit was selected deliberately for each exposure domain rather than standardised to a single time scale. Years were retained for CEI and HDEI because cosmetic-product and hair-dye use commonly extends over long periods, whereas months were retained for SPEI because slimming-product use was generally shorter and more episodic. Annual dyeing frequency was therefore combined with duration in years, while weekly slimming-product frequency was combined with duration in months. These domain-specific units were used only to construct relative behavioural ranking scores. Although each component was numerically rescaled by division by 10, CEI, HDEI, and SPEI were not interpreted as equivalent physical exposure units, and PPEI was retained only as a secondary composite summary rather than a measure of cumulative absorbed dose.

CEI, HDEI, SPEI, and PPEI were interpreted as relative ranking tools within the study population and not as direct measures of absorbed dose, toxicokinetic burden,

verified ingredient concentration, or product contamination. Because these indices are formative behavioural scores derived from selected questionnaire variables, internal-consistency statistics alone would not establish validity. In the absence of test-retest or external validation data, possible exposure misclassification is acknowledged and may contribute to underestimation or instability of exploratory associations.

Exposure-index calculations were completed before outcome analysis according to predefined scoring rules retained in the analysis workbook and data dictionary.

CEI, HDEI, SPEI, and PPEI were calculated before biomarker outcomes were inspected. Participants with incomplete component data were handled according to the predefined analysis-workbook rule and were not reclassified according to observed biomarker results. This approach prevented outcome-driven scoring and preserved reproducibility.

The formulas are reported to make the scoring logic transparent and reproducible. The indices remain behavioural ranking scores and are not interpreted as measures of absorbed chemical dose.

2.10 Natural versus commercial product classification

The product-origin variable was analysed only as an exploratory comparison within the applicable product domains. This comparison was included to test whether participant-reported market-origin categories correlated with biomarker patterns, not to evaluate inherent safety of natural or commercial products. Because product composition was not chemically verified, any observed differences may reflect user characteristics, purchasing channels, salon practices, storage conditions, product authenticity, or concurrent environmental exposures rather than product origin itself.

To reduce misclassification, unclear or mixed natural-versus-commercial product categories were not forced into binary groups unless the classification was supported by a predefined rule. In the final analysis dataset, the direct comparison included 20 natural/henna and 55 commercial hair dye users and 19 natural/herbal and 51 commercial/prepared slimming product users.

Natural and commercial categories were based on participant reports, product sources, and label information, where available. They were not based on laboratory

authentication of product ingredients; therefore, natural-versus-commercial results were interpreted as product-origin pattern comparisons rather than validated safety classifications.

In the local market context, natural/herbal products included henna-based hair dyes, herbal slimming teas, plant oil mixtures, and home-prepared creams, whereas commercial/prepared products referred to branded items sold in pharmacies, cosmetic shops, salons, or other formal retail channels.

Table 2.6 Natural and commercial product-origin groups for hair dyes and slimming products

Product domain	Natural group	Commercial group	Interpretation
Hair dyes	Natural/henna users (n=20)	Commercial hair-dye users (n=55)	Exploratory comparison; mixed/unclear categories not forced into either group.
Slimming products	Natural/herbal users (n=19)	Commercial/prepared users (n=51)	Exploratory comparison; labels and sources not chemically verified.

2.11 Blood sampling and laboratory procedures

Laboratory procedures were standardised to minimise pre-analytical and analytical variability in specimen collection, serum separation, dedicated whole-blood handling for metal analysis, storage, instrumental measurement, and assay-specific quality-control procedures. Venous blood specimens were processed for inflammatory, haematological, hepatic, renal, endocrine, nutritional, and Pb/Cd metal-burden analyses.

Morning venous blood was collected from each participant by trained laboratory personnel using sterile single-use equipment. A dedicated 5-mL whole-blood specimen for Pb and Cd analysis was collected into a K₂EDTA tube separately from the serum and routine laboratory specimens. The K₂EDTA tube was mixed immediately after collection by approximately eight gentle inversions to prevent clot formation and ensure adequate anticoagulant mixing.

Whole-blood metal specimens were maintained at 2–8°C and analysed within 24–72 hours. When delayed analysis was required, samples were stored at –20°C and were not subjected to more than one freeze–thaw cycle. Serum specimens were allowed to clot for approximately 20–30 minutes, centrifuged at 3000 rpm for 10 minutes, and analysed

on the same day when possible. Participants fasted before assays for which fasting was required by the laboratory protocol.

Routine biochemical measurements, including ALT, AST, ALP, urea, creatinine, total bilirubin, albumin, serum iron, and CRP, were performed using a Cobas c311 automated clinical chemistry analyser (Roche Diagnostics). CRP was measured on the same platform by a turbidimetric immunoassay. TSH, FT3, FT4, LH, FSH, estradiol, cortisol, vitamin D, and ferritin were measured using a MAGLUMI 600 chemiluminescence immunoassay analyser (Shenzhen New Industries Biomedical Engineering Co., Ltd. [SNIBE], China). Haemoglobin and complete blood-count parameters were measured using a Sysmex XS-1000i automated haematology analyser (Sysmex Corporation, Japan). Two levels of internal quality-control material were used for each assay; within-run and between-run coefficients of variation were maintained below 10%, and calibration or analysis was repeated when quality-control results were outside the predefined acceptable range. All routine biochemical, immunoassay, and haematological measurements were conducted in the same laboratory.

Whole-blood Pb and Cd were measured using a novAA 400 P atomic absorption spectrometer (Analytik Jena, Germany) operated in graphite-furnace atomic absorption spectrometry mode with Zeeman background correction. Separate element-specific hollow-cathode lamps were used at 283.3 nm for Pb and 228.8 nm for Cd. Seven-point calibration curves were prepared at 0, 1, 2, 5, 10, 20, and 40 µg/L for Pb and at 0, 0.2, 0.5, 1, 2, 4, and 8 µg/L for Cd. The fitted equations were $y = 0.01799x + 0.00016$ for Pb and $y = 0.08494x + 0.00028$ for Cd, with $R^2 = 0.99999$ for both elements. Calibration verification was performed using 25 µg/L Pb and 5 µg/L Cd standards and was accepted when recovery was within 90–110% of the nominal concentration. The element-specific calibration curves, analytical calibration characteristics, and a representative technical schematic of the novAA 400 P instrument model are documented in Appendix G.

A 0.5-mL aliquot of whole blood was transferred into Teflon/PTFE digestion vessels that had been pre-cleaned with 10% HNO₃ for at least 4 hours. Three millilitres of concentrated HNO₃ (65%) were added, and the samples were allowed to pre-digest overnight. The samples were then heated on a hotplate at 90°C for 20 minutes, after which 1.5 mL of H₂O₂ (30%) was added dropwise. Digestion was continued at 130°C for 90

minutes until a clear solution was obtained. After cooling, each digest was made up to a final volume of 20 mL with deionised water. The corresponding dilution factor of 40 was predefined in the instrument protocol, and the software reported the final participant-level whole-blood concentration after dilution and back-calculation (Barbosa *et al.*, 2015; Nardi *et al.*, 2015; ATSDR, 2022).

A method blank was included in each analytical batch by subjecting deionised water to the same acid-digestion procedure as the blood samples, including 3 mL HNO₃ (65%), 1.5 mL H₂O₂ (30%), heating at 90°C for 20 minutes and 130°C for 90 minutes, and adjustment to a final volume of 20 mL. An instrumental blank consisting of 5% HNO₃ was analysed at the beginning of each run and after every 10–15 samples to monitor carryover and instrumental contamination. One participant sample was analysed in duplicate within each batch; observed relative differences did not exceed 8%. Whole-blood matrix-spike recoveries ranged from 95% to 105%, and a certified whole-blood reference material for Pb and Cd produced results within ±10% of the certified target values (ISO, 2017; CDC, 2022).

Table 2.7 Key laboratory procedures and quality-control parameters

Laboratory item	Methodological specification
Metal-specimen collection	Dedicated 5 mL whole blood in a separate K ₂ EDTA tube; mixed immediately by approximately eight gentle inversions.
Sampling time and fasting	Morning sampling; fasting applied to assays requiring fasting.
Serum processing	Clotting for 20–30 min; centrifugation at 3000 rpm for 10 min; same-day analysis when possible.
Whole-blood storage	2–8°C for 24–72 h; –20°C for delayed analysis; no more than one freeze–thaw cycle.
Routine laboratory platforms	Cobas c311 for clinical chemistry and turbidimetric CRP; MAGLUMI 600 for CLIA-based hormones, ferritin, and vitamin D; Sysmex XS-1000i for CBC/haemoglobin.
Routine-assay QC	Two-level internal QC; within-run and between-run CV <10%; recalibration or repeat analysis when QC failed.

Laboratory item	Methodological specification
Metal digestion and instrumental analysis	0.5 mL whole blood; 3 mL HNO ₃ 65%; overnight pre-digestion; 90°C for 20 min; 1.5 mL H ₂ O ₂ 30% added dropwise; 130°C for 90 min; final volume 20 mL; novAA 400 P GFAAS.
Metal-analysis QC	Seven-point GFAAS calibration: Pb 0–40 µg/L and Cd 0–8 µg/L; R ² = 0.99999; calibration verification at 25 µg/L Pb and 5 µg/L Cd (90–110%); method and instrumental blanks; one duplicate per batch (observed difference ≤8%); spike recovery 95–105%; certified whole-blood reference material within ±10%; Pb LOD/LOQ 0.093/0.281 µg/L (0.0000093/0.0000281 mg/dL) and Cd LOD/LOQ 0.021/0.063 µg/L; two Cd values below LOQ evaluated by LOQ/√2 sensitivity analysis.

For biochemical outcomes, CRP was considered the primary inflammatory biomarker. Secondary exploratory outcomes included haemoglobin, ferritin, serum iron, ALT, AST, ALP, bilirubin, albumin, urea, creatinine, eGFR, TSH, free T3, free T4, LH, FSH, estradiol, cortisol, and vitamin D. These variables were interpreted as systemic biochemical status markers rather than product-specific toxicity indicators.

For heavy metals, only Pb and Cd were retained as final whole-blood variables. Pb was reported in mg/dL and Cd in µg/L throughout the analysis, tables, figures, and appendices. The instrument software reported final participant-level whole-blood concentrations after applying the predefined dilution factor. Cu and Hg were excluded from the final dataset and were not considered in the primary interpretation. The analytical characteristics and interpretive roles of all measured biomarker domains, including inflammatory, biochemical, endocrine, nutritional, and metal variables, are summarised in Table 2.8.

Table 2.8 Summary of biomarker domains, analytical reporting, and interpretive implications

Laboratory item	Analytical reporting	Interpretive implication
Biological matrix	Serum for routine biochemical and hormonal markers; anticoagulated whole blood for CBC and for Pb/Cd after acid digestion	Results are interpreted according to the biological matrix used.
Biochemical biomarkers	CRP primary; liver, renal, endocrine, haematological, and nutritional markers exploratory	The broad panel requires multiple-testing adjustment and cautious interpretation.
Heavy metals	Pb retained and reported in mg/dL; Cd retained and reported in µg/L.	Whole-blood internal-burden indicators, not proof of a specific product source.
Quality control	Calibration, calibration verification, method and instrumental blanks, duplicate precision, spike recovery, certified reference material, and method LOD/LOQ were applied	Supports analytical validity, traceability, and reproducible interpretation.

2.12 Heavy-metal reporting units and quality control

Whole-blood Pb was recorded and analysed in mg/dL, whereas whole-blood Cd was recorded and analysed in µg/L. These units were retained consistently in the analysis dataset, thesis tables, figures, appendices, and statistical outputs.

The instrument protocol included the digestion-related dilution factor of 40 and automatically back-calculated the final participant-level whole-blood concentrations. Consequently, no additional digestion or dilution factor was applied during data cleaning or statistical analysis.

Product-source attribution for Pb and Cd remained conservative. Because the cosmetics, hair dyes, and slimming products used by participants were not chemically analysed and environmental media were not sampled, whole-blood Pb/Cd values were interpreted as internal metal-burden indicators of uncertain source rather than product-source markers. Product-index correlations with Pb/Cd were therefore treated as secondary exploratory signals that may reflect shared environmental pathways, market-

source patterns, diet, water, household dust, road dust, occupational exposure, or unmeasured confounding. The final metal variables, reporting units, analytical status, and interpretation framework are summarised in Table 3.10.

Comparisons with external biomonitoring literature were made only after confirming that the reported units were compatible with the units used in the present dataset.

Table 2.9 Metal variables, reporting units, and final analytical actions

Metal variable	Reporting-unit rule	Analytical action
Pb	Whole-blood Pb was recorded and analysed in mg/dL throughout the dataset, statistical outputs, tables, figures, and appendices.	Final variable: whole-blood Pb (mg/dL).
Cd	Whole-blood Cd was recorded and analysed in µg/L throughout the dataset, statistical outputs, tables, figures, and appendices.	Final variable: whole-blood Cd (µg/L).
Variable-level unit control	The final Pb and Cd columns were checked for consistent unit labels, numeric format, and participant alignment.	Prevents mixed-unit interpretation and participant-level mismatch.
Dilution/back-calculation	The factor of 40 was predefined in the GFAAS protocol and applied automatically by the instrument software.	No additional dilution or digestion factor was reapplied during analysis.
Participant-level audit	Participant identifier, final concentration, reporting unit, analytical-status information, and classification relative to LOD/LOQ were retained for all 120 participants.	Provides traceability and supports the below-LOQ sensitivity analysis for two Cd observations.
Source-attribution boundary	Whole-blood Pb/Cd values were treated as internal metal-burden indicators of uncertain source.	Prevents unsupported attribution to a specific cosmetic, hair-dye, or slimming product.

2.13 Data management and cleaning

Data management and quality control included checking participant identifiers, missing fields, non-numeric entries, decimal-format inconsistencies, categorical coding, unit consistency, sampling dates, and biologically implausible values. Numeric variables

were range-checked, and potential outliers were investigated using tables, histograms, and box plots. Implausible values were cross-checked against the original laboratory reports and resolved or set to missing as appropriate. During the final sampling-date audit, the month had been entered incorrectly as September for six participants. The corrected dates were P057 (4 October 2025), P019 (6 October 2025), P031 (7 October 2025), P091 (8 October 2025), P058 (9 October 2025), and P064 (12 October 2025). After this correction, all sampling dates occurred after ethical approval. No missing values remained for the core biochemical, metal, and exposure-index variables in the final analysis dataset. Domain-specific natural-versus-commercial analyses included only participants with an eligible and clearly classified product-origin category. Audit files linking original values, cleaned variables with verified unit labels, and data-cleaning decisions were retained to preserve traceability and reproducibility.

No values were imputed. Each table reports the relevant analytical sample size, and the final analysis workbook retains raw variables, cleaned variables with verified unit labels, exclusion flags, unit-audit notes, and model-specific sample sizes. During the final integrity audit, participant P001 was corrected to CEI/HDEI/SPEI/PPEI = 5/3/0/8 using the original questionnaire fields. The mutually exclusive exposure groups were then verified and retained consistently as hair dye only ($n = 34$), slimming product only ($n = 30$), and hair dye plus slimming product ($n = 56$). All dependent descriptive statistics, Spearman correlations, BH-FDR values, and CRP regression models were rerun.

2.14 Statistical analysis

Several biomarkers and exposure indices demonstrated skewed distributions; therefore, the primary analyses were conducted using non-parametric statistical methods, supplemented by multivariable linear regression models using transformed CRP values. The distribution of continuous variables was assessed using the Shapiro–Wilk test, which is widely used for evaluating deviations from normality, particularly in small-to-moderate sample sizes (Shapiro & Wilk, 1965; Razali & Wah, 2011).

Variables that approximately followed a normal distribution were summarised as mean $\pm$ standard deviation (SD), whereas non-normally distributed variables were presented as

median [interquartile range (Q1–Q3)]. Categorical variables were reported as frequencies and percentages.

All statistical analyses were performed using IBM SPSS Statistics version 26 and Python version 3.11. The Python workflow included procedures for data cleaning, statistical analysis, false-discovery-rate correction, regression modelling, and figure generation. All analytical decisions, variable coding, and statistical workflows were documented to improve reproducibility and transparency.

2.14.1 Kruskal–Wallis test

Overall differences among the three exposure-pattern groups were assessed using the Kruskal–Wallis test. This non-parametric alternative to one-way analysis of variance (ANOVA) was selected because several biomarker variables did not satisfy assumptions of normality and equal variance.

The Kruskal–Wallis test evaluates whether independent groups originate from the same distribution based on ranked observations rather than raw measurements (Kruskal & Wallis, 1952).

The null hypothesis was:

H0: The distributions of all groups are equal

A p-value < 0.05 indicated evidence of a statistically significant difference among groups. Because the Kruskal–Wallis test identifies only the presence of an overall difference and does not determine which groups differ, significant results were followed by Dunn’s multiple comparison test (Dunn, 1964).

2.14.2 Dunn pairwise rank comparison

When the Kruskal–Wallis test showed statistical significance, Dunn’s post hoc test was performed to identify specific differences between exposure-pattern groups.

The three pairwise comparisons included:

- Hair-dye only versus slimming-product only.
- Hair-dye only versus combined exposure.
- Slimming-product only versus combined exposure.

Because multiple pairwise comparisons increase the probability of false-positive results, p-values were adjusted using the Benjamini–Hochberg false-discovery-rate (FDR) procedure (Benjamini & Hochberg, 1995).

Adjusted values were interpreted as:

- $q < 0.05$: statistically significant after correction.
- $q \geq 0.05$: not statistically significant after controlling for multiple comparisons.

2.14.3 Mann–Whitney U test

The Mann–Whitney U test was used for comparisons between two independent groups, particularly for natural-versus-commercial product-origin comparisons.

This test evaluates whether observations from one group tend to have higher or lower ranks than observations from another group without requiring normally distributed data (Mann & Whitney, 1947).

The null hypothesis was:

H0: The two groups have the same distribution

A p-value below 0.05 indicated evidence of a statistically significant difference between the two groups.

2.14.4 Spearman rank correlation analysis

Associations between CEI, HDEI, SPEI, PPEI, and biochemical or metal outcomes were assessed using **Spearman’s rank correlation coefficient (ρ)**.

Spearman correlation was selected because it evaluates monotonic relationships and does not require normally distributed variables (Spearman, 1904).

The coefficient ranges from:

$$-1 \leq \rho \leq +1$$

where:

- Positive values indicate that higher exposure-index values are associated with higher biomarker values.
- Negative values indicate an inverse relationship.
- Values close to zero indicate weak or absent monotonic association.

Because 88 correlations were tested (four exposure indices × 22 outcomes), the Benjamini–Hochberg FDR correction was applied, with statistical significance defined as $q < 0.05$ (Benjamini & Hochberg, 1995).

2.14.5 Effect-size estimation

Statistical significance alone does not describe the magnitude of observed differences; therefore, effect sizes were calculated.

For Kruskal–Wallis comparisons, epsilon-squared (ϵ^2) was reported as an estimate of the proportion of variability explained by group differences.

For two-group comparisons and Dunn pairwise comparisons, the rank-biserial correlation coefficient was used as a non-parametric effect-size measure.

The coefficient was calculated as:

$$rrb = \frac{2U}{n_1 n_2} - 1$$

Where:

- **U** = Mann–Whitney U statistic.
- **n₁** = sample size of the first group.
- **n₂** = sample size of the second group.

Positive values indicate higher values in the first named group, whereas negative values indicate higher values in the second named group. Values approaching zero indicate minimal group separation (Kerby, 2014).

2.14.6 Multivariable linear regression analysis

Multivariable linear regression models were used to evaluate adjusted associations between exposure indices and serum CRP concentration.

Because CRP values showed right-skewed distribution, the dependent variable was transformed as:

$\log(\text{CRP}+0.1)$

Separate regression models were constructed for:

- CEI.
- HDEI.
- SPEI.
- PPEI.

Additionally, mutually adjusted component models included CEI, HDEI, and SPEI simultaneously. PPEI was not included in the same model with its components because it represents their mathematical sum and would introduce redundancy.

All adjusted models included age, BMI, and menstrual phase as covariates. Regression coefficients were interpreted as cross-sectional associations rather than causal effects.

2.14.7 Regression diagnostics

Regression assumptions were evaluated using several diagnostic procedures.

Coefficient of determination (R^2) was used to describe the proportion of variation in CRP explained by predictors, while adjusted R^2 was reported because it accounts for the number of variables included in the model.

Multicollinearity was assessed using the variance inflation factor (VIF). Higher VIF values indicate increased correlation among predictors and potential instability of regression coefficients (O'Brien, 2007).

Residual variance was evaluated using the Breusch–Pagan test for heteroscedasticity (Breusch & Pagan, 1979), while independence of residuals was assessed using the Durbin–Watson statistic (Durbin & Watson, 1950).

Potentially influential observations were evaluated using Cook's distance, with $4/N$ used as a screening threshold and values approaching 1.0 considered indicative of substantial influence (Cook, 1977).

Sensitivity analyses were conducted using heteroscedasticity-consistent HC3 standard errors and exclusion of observations exceeding the Cook's distance screening threshold.

2.14.8 Multiple-testing control

Because multiple biomarker comparisons increase the probability of false-positive findings, the Benjamini–Hochberg false-discovery-rate procedure was applied across the relevant statistical families.

The correction was applied to:

- 22 omnibus biomarker comparisons.
- 88 exposure-index correlation analyses.
- Dunn pairwise comparisons within each biomarker.

This approach controls the expected proportion of false discoveries among statistically significant findings (Benjamini & Hochberg, 1995).

Table 2.10 Main research questions, exposure variables, outcomes, and planned statistical analyses

Research question	Independent variable	Outcome	Primary test/model	Reporting
Cosmetic exposure and inflammation	CEI	CRP	Spearman; adjusted linear regression on $\log(\text{CRP} + 0.1)$	ρ , β , 95% CI, p, FDR q; association wording only.
Hair-dye exposure and biomarkers	HDEI / hair-dye category	CRP, Pb, Cd, exploratory biomarkers	Spearman; Kruskal–Wallis followed by Dunn–BH pairwise comparisons; Mann–Whitney as appropriate	median [Q1–Q3], omnibus p/FDR q, Dunn–BH adjusted pairwise p, effect direction; metal findings retain sensitivity caution
Slimming-product exposure and biomarkers	SPEI / slimming category	CRP, ALT, AST, urea, creatinine, hormones, Pb, Cd	Spearman; Kruskal–Wallis followed by Dunn–BH pairwise comparisons; Mann–Whitney; adjusted regression if justified	ρ , p, FDR q, and cautious cross-sectional interpretation.
Overall product exposure	PPEI	CRP and biomarker panel	Spearman; adjusted regression on $\log(\text{CRP} + 0.1)$	overall exposure-pattern signal; secondary analysis.

Research question	Independent variable	Outcome	Primary test/model	Reporting
Natural vs commercial origin	Product-origin category	Biochemical and metal markers	Mann–Whitney U; FDR adjustment	exploratory comparison; no causal or safety claim.
Sample adequacy and group balance	Final exposure-pattern groups and product indices	Participant characteristics and group counts	Descriptive adequacy check; no causal power claim	N per group, imbalance note, and conservative interpretation.
Missing-data handling	All exposure and biomarker variables	Model-specific analytical sample	Adjusted linear regression with variance-inflation, residual, homoscedasticity, and influence diagnostics; HC3 and Cook’s-distance sensitivity analyses	β , 95% CI, p, R^2 /adjusted R^2 , maximum VIF, Breusch–Pagan p, residual-normality result, Cook’s-distance screening, and robust/sensitivity interpretation
Regression model assessment	CEI, HDEI, SPEI, PPEI, age, BMI, and menstrual phase	$\log(\text{CRP} + 0.1)$	Adjusted linear regression; R^2 and adjusted R^2 used to summarise model fit	β , 95% CI, p, approximate percentage change in $\text{CRP} + 0.1$, R^2 , and adjusted R^2 ; exploratory association wording only.
Pb/Cd attribution boundary	Whole-blood Pb (mg/dL) and Cd ($\mu\text{g/L}$) variables	Internal metal burden	Correlation/group comparison only	Report as uncertain-source internal burden, not product-specific exposure source.

2.15 Bias, confounding, and interpretive safeguards

Potential selection bias may have arisen from the convenience-sampling strategy and recruitment settings. Information and exposure-classification biases may also have occurred because product-use variables were questionnaire-derived.

Information bias may have occurred because product-use data were questionnaire-derived and may have been affected by recall error, uncertain product labels, variable product definitions, and mixed product use. Misclassification is especially possible for natural/herbal versus commercial/prepared slimming products because product labels have not been chemically verified.

Important potential confounders included age, BMI, menstrual phase, recent infection, medication use, oral contraceptive or hormonal treatment, pregnancy/lactation status, occupation, salon work, diet, physical activity, passive smoke exposure, household dust, generator exposure, road proximity, drinking-water source, and other environmental metal sources. Covariates included in the final adjusted models were limited to variables available in the final dataset. Unmeasured confounders were explicitly treated as limitations.

Throughout the thesis, causal language was avoided. Findings were described as associations, exposure-pattern signals, or hypothesis-generating observations rather than as product-caused biomarker changes.

2.16 Control-Group Limitations and Interpretive Boundaries

Because the study did not recruit a fully independent unexposed control group, the study cannot distinguish absolute exposure effects from baseline characteristics of the sampled population. The comparisons are therefore interpreted as gradients or patterns of reported product use within the available female participant sample, not as proof that product users differ causally from non-users.

All regression, correlation, and group-comparison results were interpreted as cross-sectional exposure-pattern associations. They do not establish temporal sequence, reverse-causation direction, product-specific toxicity, or direct Pb/Cd source attribution. This interpretive boundary was applied consistently in the Summary, Results, and Discussion.

Future studies should recruit a matched minimally exposed or non-user comparison group from the same geographic area, collect paired product samples, household-dust samples, drinking-water and food information, and repeat biomarker measurements over time. Such a design would allow stronger evaluation of temporality, source attribution, and dose-response relationships.

ChapterThree

Results & Discussion

RESULTS

This chapter presents the findings obtained from the final study dataset and discusses their biological and environmental health implications. The results are organized according to the main objectives of the study, including the characterization of participant exposure patterns, assessment of whole-blood lead and cadmium concentrations, evaluation of biochemical and hormonal biomarkers, and investigation of the relationships between exposure indices and measured health outcomes.

Statistical analyses were interpreted using the adjusted results after false discovery rate (FDR) correction where applicable, while additional exploratory analyses are provided in the supplementary appendices. Whole-blood lead (Pb) and cadmium (Cd) concentrations were considered indicators of internal metal burden and were not interpreted as direct evidence of contamination from specific cosmetic, hair-dye, or slimming products.

3.1 Whole-blood Lead and Cadmium Concentrations

Whole-blood lead (Pb) and cadmium (Cd) concentrations were determined for all participants included in the final analytical dataset (N=120). Lead concentrations exceeded the analytical quantification limit in all samples, while cadmium was detected in all participants, with the majority of measurements being above the quantification threshold. Therefore, both metals were considered reliable indicators of internal metal burden and were included in subsequent statistical analyses.

The calibration curves demonstrated excellent linearity for both Pb and Cd measurements. The Pb calibration curve showed an R^2 value of 0.9999, with LOD and LOQ values of 0.093 and 0.281 $\mu\text{g/L}$, respectively. For Cd, the calibration curve showed an R^2 value of 0.9999, with LOD and LOQ values of 0.021 and 0.063 $\mu\text{g/L}$, respectively (Appendix G, Figure G.1).

As presented in Table 3.1, the measured whole-blood Pb concentrations ranged from 1.10 to 32.77 $\mu\text{g/L}$, with a mean concentration of $14.29 \pm 6.82 \mu\text{g/L}$. Cadmium

concentrations ranged from 0.021 to 3.970 µg/L, with a mean concentration of 0.608 ± 0.763 µg/L. These findings describe the background distribution of internal metal exposure among the studied population before evaluating their associations with exposure patterns and biological markers.

Table 3.1. Distribution of whole-blood lead and cadmium concentrations among study participants(N=120)

Metal	N	Mean ± SD (µg/L)	Median [Q1–Q3]	Range
Pb	120	14.29 ± 6.82	14.91 [10.10–18.87]	1.10–32.77
Cd	120	0.608 ± 0.763	0.373 [0.261–0.477]	0.021–3.970

The detection of measurable whole-blood lead (Pb) and cadmium (Cd) concentrations among all study participants indicates the presence of internal exposure to trace metals within the studied population. In the present study, Pb concentrations ranged from 1.10 to 32.77 µg/L, whereas Cd concentrations ranged from 0.021 to 3.970 µg/L. These findings should be interpreted as indicators of internal metal burden rather than direct evidence of contamination from specific cosmetic, hair-dye, or slimming products, because blood metal concentrations reflect the cumulative contribution of multiple environmental and lifestyle-related exposure sources (Sullivan *et al.*, 2023).

Lead and cadmium are non-essential toxic metals with no known physiological role in humans. Even at relatively low exposure levels, chronic accumulation of these metals may contribute to biological disturbances through oxidative stress induction, inflammatory activation, mitochondrial dysfunction, and disruption of cellular signalling pathways (Howard et al., 2024; Qu & Zheng, 2024).

The presence of detectable Pb levels in the current population is consistent with previous biomonitoring studies showing that low-level lead exposure remains widespread in general populations despite reductions in environmental lead sources. Blood lead measurement is considered an appropriate biomarker for evaluating recent and ongoing exposure because lead circulates in blood and reflects internal exposure status during the relevant exposure window (Sullivan *et al.*, 2023). However, the absence of a specific exposure source in

the current design prevents attributing the detected Pb concentrations specifically to cosmetic products, hair dyes, or slimming products.

Similarly, measurable Cd concentrations among participants may reflect exposure from multiple environmental sources, including diet, tobacco smoke, occupational environments, and contaminated consumer products. Cadmium has a long biological persistence and accumulates mainly in renal tissues, where chronic exposure has been associated with oxidative stress, renal dysfunction, and inflammatory responses (Badawi *et al.*, 2024; Qu & Zheng, 2024).

In the present study, although Pb and Cd showed significant differences among exposure-pattern groups in the primary analysis, these differences were not maintained after sensitivity analysis excluding the separate metal batch. Therefore, these findings should be considered exploratory rather than evidence of a confirmed association between personal-care product exposure patterns and blood metal concentrations. This interpretation is consistent with current recommendations in human biomonitoring studies, which emphasize that metal biomarkers should be interpreted within the context of analytical reliability, exposure timing, and potential confounding factors (Viegas *et al.*, 2024).

3.2 Participant Characteristics and Exposure-Index Distribution

The demographic characteristics and calculated exposure indices of the study participants are summarized in Table 3.2. These descriptive parameters provide an overview of the study population and demonstrate the variability in exposure intensity related to cosmetic products, hair dyes, and slimming products. The exposure indices (CEI, HDEI, SPEI, and PPEI) were used to quantify cumulative patterns of product use and were subsequently evaluated in relation to biochemical markers and metal burden indicators.

The study included 120 adult women with a mean age of 34.38 ± 7.90 years and a mean BMI of 26.56 ± 3.33 kg/m². The calculated exposure indices showed considerable variation among participants, reflecting differences in the frequency, duration, and combined use of personal-care products.

Table 3.2. Demographic characteristics, exposure indices, and inflammatory marker distribution among study participants (N=120)

Variable	N	Mean $\pm$ SD	Median [Q1–Q3]	Range
Age (years)	120	34.38 $\pm$ 7.90	34 [26.75–41.25]	20–50
BMI (kg/m²)	120	26.56 $\pm$ 3.33	26.85 [24.05–28.60]	20–34.10
CEI	120	3.69 $\pm$ 2.41	3.60 [2–5]	0–12
HDEI	120	5.60 $\pm$ 5.85	4.00 [0.15–9.60]	0–26
SPEI	120	4.59 $\pm$ 3.60	5.40 [0–8.40]	0–12.60
PPEI	120	13.88 $\pm$ 7.66	13 [8–17.55]	2.10–38.60
CRP (mg/L)	120	6.02 $\pm$ 3.53	5.88 [3.54–7.91]	0.20–13.97

The calculated exposure indices (CEI, HDEI, SPEI, and PPEI) provided a quantitative representation of personal-care product use patterns among the studied women. Unlike simple user/non-user classification, cumulative exposure indices incorporate differences in frequency, duration, and combined use of products, allowing a more realistic evaluation of real-world exposure scenarios. Similar approaches have been increasingly applied in environmental health research because individuals are commonly exposed to mixtures of chemicals from multiple consumer products rather than isolated compounds (Senta et al., 2020).

In the current study, the variability observed in CEI, HDEI, SPEI, and PPEI values indicates substantial differences in personal-care product exposure intensity among participants. This variation is expected because cosmetic and personal-care product use is influenced by behavioural, cultural, socioeconomic, and lifestyle factors. Previous studies have demonstrated that frequent use of cosmetics and personal-care products can increase internal exposure to several chemical constituents, including phthalates, parabens,

bisphenols, and other endocrine-active compounds, which may be absorbed through dermal contact and subsequently detected in biological matrices (Senta et al., 2020; Voutsas et al., 2025).

The mean BMI observed among participants indicated that the study population included women with different body composition profiles. BMI is an important consideration in biomonitoring studies because adiposity may influence the distribution, metabolism, and biological effects of some environmental chemicals. In addition, obesity-related low-grade inflammation may contribute to variations in inflammatory biomarkers such as CRP, making adjustment for BMI necessary in subsequent regression models.

The use of combined exposure indices such as PPEI represents an important strength of the current study because exposure to personal-care products rarely occurs independently. Individuals may simultaneously use cosmetics, hair dyes, and slimming products, resulting in potential combined exposure to multiple chemical classes. Recent research has emphasized that mixture-based exposure assessment provides a more realistic framework than evaluating single compounds separately, as real-life consumer exposure occurs through complex chemical mixtures (Jobe, 2026).

However, the interpretation of exposure indices should consider that they are based on self-reported product-use information rather than direct chemical measurement of products. Therefore, these indices represent behavioural exposure intensity and do not directly quantify the absorbed dose of specific chemicals. This limitation highlights the need for future studies combining questionnaire-based exposure assessment with chemical analysis of products and biological monitoring of specific metabolites.

The distribution of exposure indices among study participants is further illustrated in Figure 3.1. The median and interquartile ranges demonstrate the variability in cumulative exposure levels related to cosmetic products, hair dyes, and slimming products. PPEI showed the highest overall exposure variability, reflecting the combined contribution of different personal-care product exposures.

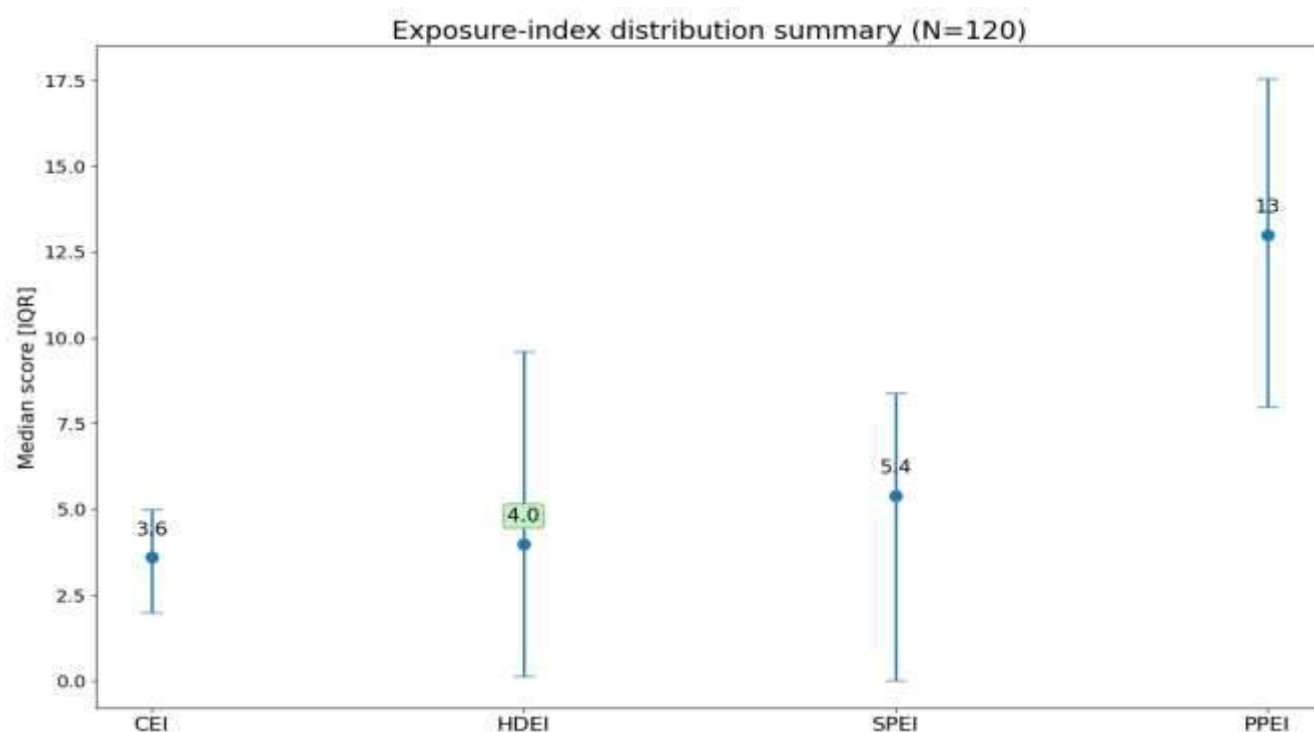


Figure 3.1 Median and interquartile range of CEI, HDEI, SPEI, and PPEI exposure indices (N=120).

3.3 Exposure Pattern Distribution

Participants were categorized according to their reported patterns of hair-dye and slimming-product use to evaluate differences in biological markers across exposure profiles. As shown in Table 3.3, participants were classified into three mutually exclusive exposure groups: hair-dye users only, slimming-product users only, and combined hair-dye and slimming-product users. Cosmetic product use was considered an overlapping exposure variable because most participants reported cosmetic use regardless of their primary exposure classification; therefore, it was not included as a separate exposure group.

Among the 120 participants, the combined hair-dye and slimming-product exposure group represented the largest proportion of the study population, followed by the hair-dye-only group and the slimming-product-only group.

Table 3.3. Distribution of participants according to exposure patterns and personal-care product use (N=120)

Exposure pattern		N
Hair dye only		34
Slimming product only		30
Hair dye + slimming product		56

The distribution of exposure indices is further illustrated in Figure 3.2. The distribution of exposure-pattern groups is illustrated in Figure 3.2

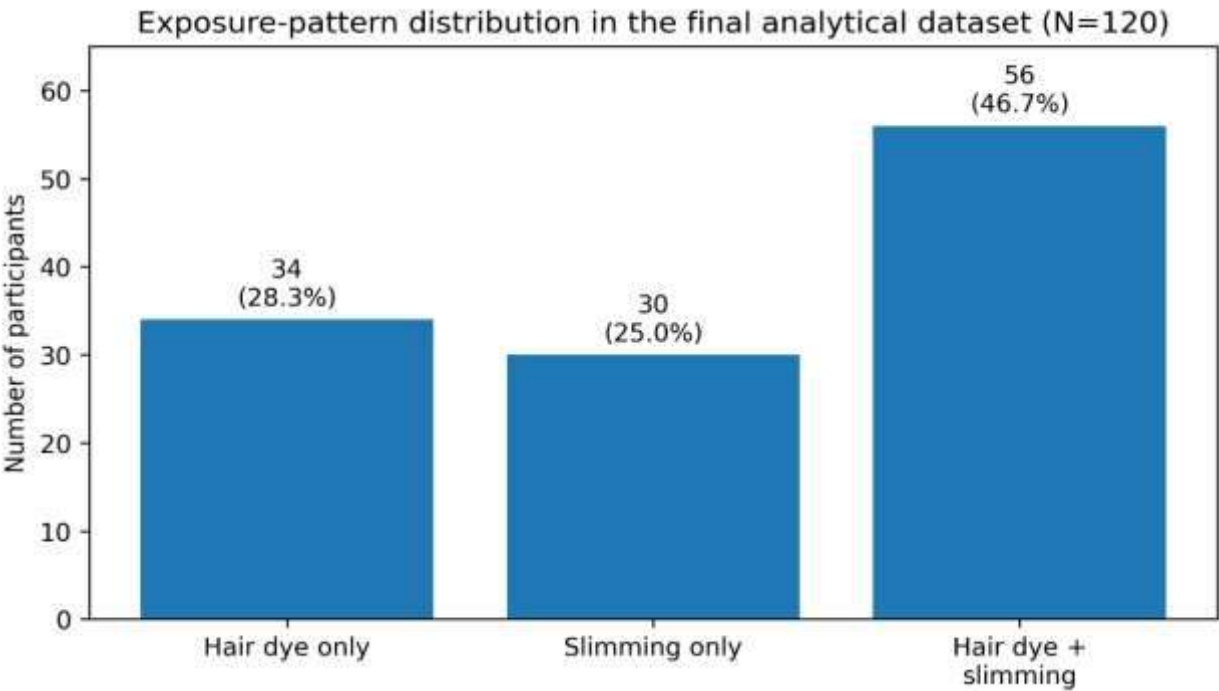


Figure 3.2 Distribution of exposure-pattern groups in the final analytical dataset(N=120).

The distribution of exposure patterns in the present study demonstrated that combined exposure to hair dyes and slimming products represented the most frequent exposure profile among participants. This finding indicates that personal-care product exposure commonly occurs as a multiple-product pattern rather than as an isolated single-product exposure.

The predominance of combined exposure is biologically relevant because individuals are frequently exposed to mixtures of chemical compounds originating from different

consumer products. Hair dyes, cosmetics, and slimming products may contain diverse chemical components, including colouring agents, preservatives, fragrances, botanical extracts, and other bioactive substances. Therefore, evaluating combined exposure patterns may provide a more realistic representation of real-life exposure scenarios compared with assessment of individual products alone (Nohynek *et al.*, 2010; Bom *et al.*, 2019).

Previous biomonitoring studies have emphasized that simultaneous exposure to multiple personal-care products can increase the diversity of chemical exposures detected in biological samples. The combined use of several products may also complicate the identification of individual contributors because chemicals from different sources may share similar biological pathways, including oxidative stress, endocrine modulation, and inflammatory signalling (Braun *et al.*, 2014).

In the current study, exposure groups were defined according to reported product use and were used as behavioural exposure categories rather than direct measurements of absorbed chemical doses. Therefore, the higher proportion of combined exposure should be interpreted as an indication of common product-use behaviour rather than evidence of increased toxicity in this group. Further studies incorporating chemical analysis of individual products and specific biomarkers of exposure are required to clarify the contribution of each product category.

3.3.1 Product-Type and Menstrual-Phase Distributions

The distribution of product types and menstrual-cycle phases among participants is presented in Table 3.4. These characteristics were included to provide a detailed description of exposure patterns and to support statistical adjustment where appropriate.

Among hair-dye users, commercial permanent dyes represented the most frequently reported category, followed by natural/henna and commercial semi-permanent dyes. Slimming-product use showed greater heterogeneity, including capsules/tablets, powders, herbal preparations, topical creams/gels, and mixed forms. These classifications were based on participant-reported product use and represent exposure categories rather than chemically confirmed product compositions.

Regarding menstrual-cycle phase, most participants were classified within the follicular phase, while a smaller proportion were in the ovulatory phase at the time of sample collection.

Table 3.4 Product-type and menstrual-phase distributions used for exposure description and adjustment

Variable	Category	Count
Hair-dye type	Natural/henna	20
Hair-dye type	Commercial permanent	40
Hair-dye type	Not applicable: non-user	15
Hair-dye type	Missing/unclear among records	15
Hair-dye type	Commercial semi-permanent	15
Hair-dye type	Mixed natural/commercial	6
Hair-dye type	Mixed	9
Slimming-product type	Not applicable: non-user	19
Slimming-product type	Missing/unclear among records	15
Slimming-product type	Herbal tea/powder	7
Slimming-product type	Capsule/tablet	32
Slimming-product type	Powder	13
Slimming-product type	Topical cream/gel	6
Slimming-product type	Herbal tea	12
Slimming-product type	Mixed	12
Slimming-product type	Mixed topical + oral	4
Menstrual phase	Follicular	89
Menstrual phase	Ovulatory	31

The distribution of product categories provides important contextual information regarding the nature of exposure among the studied participants. Commercial permanent hair dyes represented the most frequently reported hair-dye category, suggesting that long-

lasting colouring products are commonly preferred among users. This pattern may be related to their prolonged colour retention and convenience compared with temporary alternatives.

Hair dyes contain multiple chemical constituents, including oxidative colouring agents, aromatic amines, and other reactive compounds. Although modern formulations are regulated for safety, repeated and prolonged exposure has been investigated because some ingredients may participate in oxidative stress pathways or act as potential sensitizers (Nohynek *et al.*, 2010; Bolt & Golka, 2007).

Regarding slimming products, capsules/tablets represented the most common category, followed by powders and herbal preparations. The preference for oral slimming products is important because systemic exposure may differ from topical products due to gastrointestinal absorption and metabolism. Previous studies have highlighted that herbal and weight-loss products may show considerable variability in composition, quality control, and possible contamination, which may influence their biological effects (Posadzki *et al.*, 2013).

The distribution of menstrual-cycle phases showed that most participants were sampled during the follicular phase. This information is relevant because hormonal fluctuations during the menstrual cycle may influence inflammatory markers, endocrine measurements, and metabolic parameters. Therefore, menstrual phase was included as a covariate in adjusted statistical models to reduce potential biological variability.

Overall, these findings emphasize the importance of considering product type and physiological status when evaluating associations between personal-care product exposure and biological outcomes. However, the reported product categories represent self-reported usage patterns and do not provide direct information regarding chemical composition or absorbed doses.

3.4 Descriptive statistics of biochemical and hormonal markers

The descriptive characteristics of the biochemical, renal, hepatic, endocrine, nutritional, and haematological biomarkers measured among the study participants are presented in Table 3.5. These parameters represent the secondary biological outcomes evaluated to explore potential associations between personal-care product exposure patterns and systemic physiological responses.

The measured biomarkers demonstrated variable distributions among participants; therefore, both mean $\pm$ standard deviation and median with interquartile range were provided to describe normally and non-normally distributed variables, respectively. The complete descriptive profile provides the baseline distribution of biological markers before assessing differences between exposure-pattern groups and their relationships with exposure indices.

Table 3.5. Descriptive statistics of biochemical, renal, hepatic, endocrine, nutritional, and haematological biomarkers among study participants (N=120)

Biomarker	n	Mean $\pm$ SD	Median [Q1-Q3]	Min	Max
Hb (g/dL)	120	12.54 $\pm$ 1.13	12.60 [11.70-13.40]	9.50	14.90
Ferritin (μ g/L)	120	65.18 $\pm$ 49.12	55.90 [30-86.50]	3.30	230
Serum iron (μ g/dL)	120	88.51 $\pm$ 24.01	89.80 [69.50-103.35]	29	149.10
ALT (U/L)	120	17.87 $\pm$ 8.89	16.85 [10-24.23]	4.30	40.50
AST (U/L)	120	22.15 $\pm$ 11.85	20.45 [14.88-25.85]	8	94.30
ALP (U/L)	120	71.89 $\pm$ 19.51	71.70 [58.60-83.65]	28	117.10
Bilirubin (mg/dL)	120	0.65 $\pm$ 0.21	0.66 [0.51-0.78]	0.21	1.19
Albumin (g/dL)	120	4.38 $\pm$ 0.27	4.39 [4.21-4.57]	3.78	4.93
Urea (mg/dL)	120	26.39 $\pm$ 8.85	26.20 [21.18-32.82]	2.30	48.90
Creatinine (mg/dL)	120	0.70 $\pm$ 0.17	0.69 [0.60-0.82]	0.30	1.04

Biomarker	n	Mean ± SD	Median [Q1-Q3]	Min	Max
eGFR (mL/min/1.73 m²)	120	111.26 ± 7.49	111.65 [105.57-116.22]	96.80	129.70
TSH (μIU/mL)	120	1.95 ± 1.05	1.68 [1.24-2.29]	0.60	6.02
Free T3 (pg/mL)	120	3.16 ± 0.33	3.16 [2.98-3.39]	2.28	3.82
Free T4 (ng/dL)	120	1.13 ± 0.19	1.12 [0.98-1.27]	0.75	1.50
LH (mIU/mL)	120	11.23 ± 11.13	7.59 [4.32-13.96]	0.23	67.50
FSH (mIU/mL)	120	9.12 ± 8.93	7.42 [4.96-10.04]	0.30	68.80
Estradiol (pg/mL)	120	78.73 ± 67.43	54.05 [34.08-100]	4.60	421.20
Cortisol (μg/dL)	120	13.76 ± 10.26	12.70 [7.79-16.23]	4.64	86.20
Vitamin D (ng/mL)	120	16.80 ± 10.20	16.05 [8.97-23.62]	1.90	41.30

The descriptive analysis of biochemical, renal, hepatic, endocrine, nutritional, and inflammatory biomarkers provides an overview of the physiological status of the studied population before assessing associations with exposure patterns. The variability observed among these biomarkers reflects the complex interaction between individual characteristics, lifestyle factors, environmental exposures, and physiological regulation.

The measured inflammatory marker CRP showed variability among participants, which is expected because systemic inflammation is influenced by multiple factors, including adiposity, metabolic status, infections, and environmental exposures. Low-grade chronic inflammation has been increasingly recognized as a potential biological pathway linking environmental chemical exposure with adverse health outcomes (Calder *et al.*, 2013).

The analytical sensitivity assessment showed that Pb measurements were above the quantification limit in all samples, while two Cd measurements were detected between the LOD and LOQ. These values were retained in the primary analysis with sensitivity analysis performed using LOQ/√2 substitution.

The distribution of liver-function markers, including ALT, AST, ALP, bilirubin, and albumin, demonstrated differences between individuals but generally reflected preserved hepatic function within the studied population. Nevertheless, evaluation of liver-related biomarkers is important because some personal-care product constituents and slimming-product ingredients have been investigated for their potential hepatotoxic effects through oxidative stress, mitochondrial dysfunction, and inflammatory mechanisms (Navarro *et al.*, 2017).

Renal markers, including urea, creatinine, and eGFR, were assessed because the kidneys represent a major target organ for several environmental toxicants, particularly heavy metals such as cadmium. Although the descriptive findings alone cannot establish exposure-related impairment, monitoring renal biomarkers provides an important indication of potential physiological alterations associated with environmental and consumer-product exposures (Satarug *et al.*, 2020).

Hormonal and nutritional markers were included because endocrine regulation and micronutrient status may influence inflammatory responses and metabolic health. Vitamin D deficiency, which is common in many populations, has been associated with altered immune regulation and inflammatory processes, while hormonal fluctuations may contribute to variability in endocrine biomarkers among women (Bikle, 2020).

Overall, the descriptive profile establishes the biological background of the study population and provides the basis for subsequent analyses examining whether specific exposure patterns are associated with changes in these biomarkers.

3.5 Biomarker Differences Across Exposure-Pattern Groups

To evaluate whether biological markers differed according to reported exposure patterns, participants were compared across three groups: hair-dye users only, slimming-product users only, and combined hair-dye and slimming-product users. Because several biomarkers showed non-normal distributions, differences among groups were assessed using the Kruskal–Wallis test followed by Dunn’s post hoc comparisons with Benjamini–Hochberg adjustment.

As shown in Table 3.6, several biomarkers remained significantly different among exposure-pattern groups after false discovery rate correction. The most consistent differences were observed in markers related to liver function (ALT), renal function (urea, creatinine, and eGFR), endocrine markers (TSH, LH, FSH, and cortisol), ferritin, and vitamin D levels.

The observed differences should be interpreted as associations with exposure patterns rather than evidence of causation because of the cross-sectional design of the study. Whole-blood Pb and Cd differences observed in the primary analysis were considered exploratory findings, as these associations were not maintained after batch-exclusion sensitivity analysis.

Table 3.6. Biomarkers showing significant differences across exposure-pattern groups after FDR adjustment

Biomarker	Hair dye only	Slimming only	Hair dye + slimming	H	p	FDR q	ϵ^2
Ferritin ($\mu\text{g/L}$)	Hair dye only n=34, median=35.2	Slimming only n=30, median=50	Hair dye + slimming n=56, median=63.1	10.45	0.005	0.011	0.072
ALT (U/L)	Hair dye only n=34, median=11.3	Slimming only n=30, median=18.8	Hair dye + slimming n=56, median=21.8	17.22	<0.001	<0.001	0.130
Urea (mg/dL)	Hair dye only n=34, median=23.4	Slimming only n=30, median=25.7	Hair dye + slimming n=56, median=29.3	13.64	0.001	0.003	0.099
Creatinine (mg/dL)	Hair dye only n=34, median=0.6	Slimming only n=30, median=0.71	Hair dye + slimming n=56, median=0.75	19.16	<0.001	<0.001	0.147
eGFR (mL/min/1.73 m²)	Hair dye only n=34, median=115	Slimming only n=30, median=115	Hair dye + slimming n=56, median=107	20.52	<0.001	<0.001	0.158
TSH ($\mu\text{IU/mL}$)	Hair dye only n=34, median=1.38	Slimming only n=30, median=1.85	Hair dye + slimming n=56, median=1.78	10.10	0.006	0.012	0.069
LH (mIU/mL)	Hair dye only n=34, median=14.5	Slimming only n=30, median=6.65	Hair dye + slimming n=56, median=6.1	16.85	<0.001	<0.001	0.127
FSH (mIU/mL)	Hair dye only n=34, median=6.01	Slimming only n=30, median=8.35	Hair dye + slimming n=56, median=8.16	12.40	0.002	0.004	0.089

Biomarker	Hair dye only	Slimming only	Hair dye + slimming	H	p	FDR q	ϵ^2
Cortisol ($\mu\text{g/dL}$)	Hair dye only n=34, median=7.68	Slimming only n=30, median=13.25	Hair dye + slimming n=56, median=13.8	18.62	<0.001	<0.001	0.142
Vitamin D (ng/mL)	Hair dye only n=34, median=7.55	Slimming only n=30, median=19.8	Hair dye + slimming n=56, median=17.7	27.98	<0.001	<0.001	0.222
Pb ($\mu\text{g/L}$)	Hair dye only n=34, median=5.90	Slimming only n=30, median=15.94	Hair dye + slimming n=56, median= 16.48	25.34	<0.001	<0.001	0.200
Cd ($\mu\text{g/L}$)	Hair dye only n=34, median=0.885	Slimming only n=30, median=0.301	Hair dye + slimming n=56, median=0.348	22.74	<0.001	<0.001	0.177

Note. The p and FDR q values in Table 3.6 are omnibus Kruskal–Wallis results. Significant group pairs were identified using Dunn tests with Benjamini–Hochberg adjustment across the three pairwise comparisons within each biomarker (Appendix C.1). After excluding the separate 20-sample metal batch, the omnibus group effects were no longer significant for Pb (H = 0.096, p = 0.953) or Cd (H = 2.117, p = 0.347); consequently, the primary metal pairwise results are non-robust exploratory observations.

The Dunn–BH comparisons showed that the hair-dye-only group had lower ferritin than the combined group; lower ALT, creatinine, TSH, FSH, cortisol, and vitamin D than both slimming-product groups; and higher LH than both slimming-product groups. Urea was higher and eGFR was lower in the combined group than in each single-exposure group. In the primary full-sample metal analysis, Pb was lower and Cd was higher in the hair-dye-only group than in both other groups, but these metal contrasts were not retained

in the batch-exclusion sensitivity analysis. The slimming-only and combined groups did not differ significantly for the remaining post hoc comparisons.

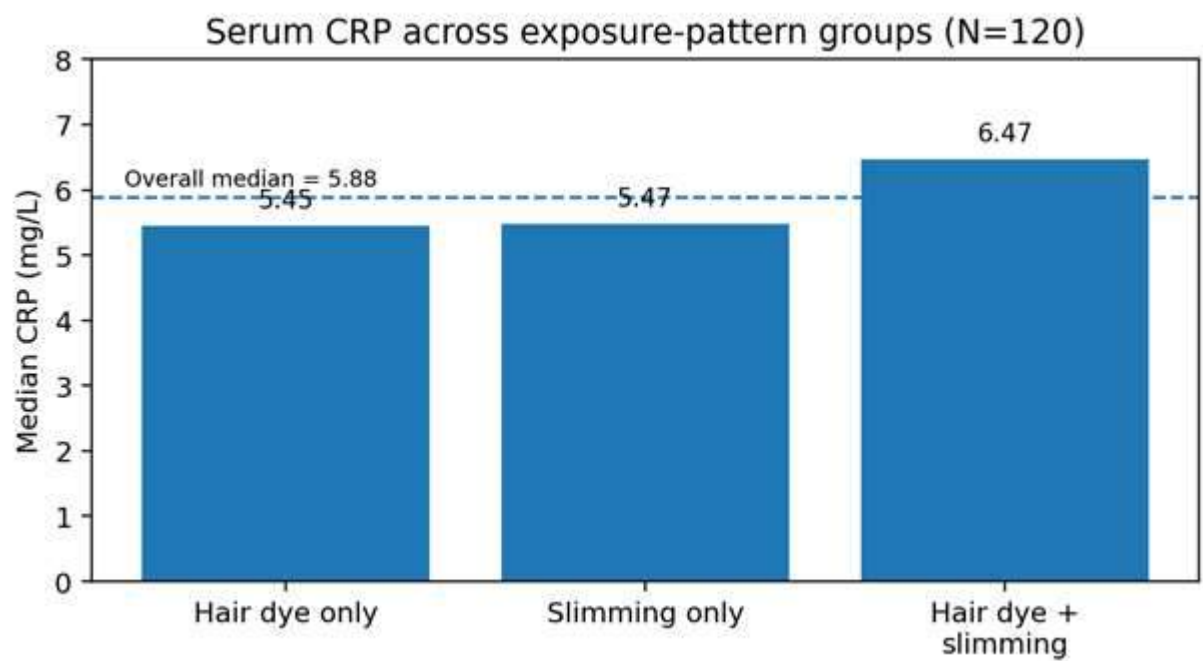


Figure 3.3 Median serum CRP levels across the three exposure-pattern groups with overall median reference line (N=120).

3.5.1 Liver Function Marker (ALT)

In the present study, ALT levels differed significantly among exposure-pattern groups, with higher median values observed in the combined hair-dye and slimming-product group compared with the hair-dye-only group. ALT is widely used as an indicator of hepatocellular injury because it is released into circulation following hepatocyte damage.

The observed association between exposure pattern and ALT may be biologically plausible because slimming products, particularly herbal preparations, have previously been linked with liver injury through mechanisms involving hepatotoxic compounds, contamination, adulteration, and oxidative stress-mediated cellular damage (Navarro *et al.*, 2017). Several reports have documented cases of herbal and dietary supplement-induced liver injury, although susceptibility varies considerably between individuals.

Hair-dye exposure has also been investigated in relation to oxidative stress because some dye constituents, particularly aromatic amines and oxidative colouring agents, may generate reactive oxygen species under certain conditions (Bolt & Golka, 2007). However, the absence of a significant difference between all exposure categories after controlling for multiple factors indicates that the observed ALT variation should be interpreted as an association rather than direct evidence of hepatotoxicity.

The current findings are consistent with studies suggesting that repeated exposure to multiple consumer products may contribute to biological stress responses, but they also emphasize the importance of considering other factors such as diet, medication use, obesity, and lifestyle characteristics.

3.5.2 Renal Function Markers (Urea, Creatinine, and eGFR)

In the present study, renal function indicators, including urea, creatinine, and estimated glomerular filtration rate (eGFR), showed significant differences across exposure-pattern groups. These findings suggest that differences in personal-care product exposure patterns may be associated with variations in renal-related biomarkers; however, they should be interpreted cautiously because renal function is influenced by multiple factors, including age, hydration status, dietary habits, medication use, metabolic conditions, and environmental exposures.

The kidney is considered a major target organ for several environmental toxicants because of its role in filtration and elimination of circulating compounds. Heavy metals, particularly cadmium, have been extensively studied for their nephrotoxic potential. Chronic cadmium exposure may lead to accumulation in renal tubular cells, oxidative stress generation, tubular dysfunction, and alterations in filtration capacity (Satarug *et al.*, 2020). Although the current study did not demonstrate robust exposure-group differences in Pb and Cd after sensitivity analysis, the assessment of renal markers remains biologically relevant when evaluating populations exposed to multiple environmental and consumer-product sources.

The observed differences in creatinine and eGFR between exposure groups may reflect variations in physiological status rather than direct renal toxicity. Previous studies have shown that lifestyle factors, body composition, inflammation, and nutritional status can influence renal biomarkers independently of chemical exposure (Levey *et al.*, 2020). Therefore, the current findings should be considered as indicators of possible biological variation associated with exposure patterns rather than evidence of kidney injury.

The relationship between slimming-product exposure and renal biomarkers requires particular attention because some herbal and weight-loss products may contain bioactive compounds, contaminants, or undeclared ingredients that can affect renal function in susceptible individuals (Posadzki *et al.*, 2013). Nevertheless, direct chemical analysis of the products and longitudinal follow-up are required to determine whether these associations represent true exposure-related effects.

3.5.3 Endocrine Markers (TSH, LH, FSH, and Cortisol)

In the present study, several endocrine biomarkers showed significant differences across exposure-pattern groups, suggesting that personal-care product exposure patterns may be associated with variations in hormonal profiles. Endocrine markers are particularly relevant in environmental health research because many chemicals present in consumer products have been investigated for their potential endocrine-disrupting properties.

The observed differences in thyroid-stimulating hormone (TSH) among exposure groups may be biologically plausible because some environmental chemicals, including certain endocrine-disrupting compounds, have been reported to interfere with thyroid hormone regulation through alterations in hormone synthesis, transport, metabolism, or receptor activity (Diamanti-Kandarakis *et al.*, 2009). However, thyroid function is influenced by multiple factors, including nutritional status, autoimmune conditions, medications, and iodine availability; therefore, these findings require cautious interpretation.

Differences in reproductive hormones, including luteinizing hormone (LH) and follicle-stimulating hormone (FSH), may reflect variations in endocrine regulation among

exposure groups. Previous experimental and epidemiological studies have suggested that exposure to some cosmetic-related chemicals, such as phthalates and certain preservatives, may influence reproductive endocrine pathways through estrogenic or anti-androgenic mechanisms (Gore *et al.*, 2015). Nevertheless, the current study cannot establish whether the observed hormonal differences are directly attributable to product exposure because of the cross-sectional design.

Cortisol showed differences between exposure patterns, which may indicate a possible relationship between product-use behaviour and stress-related physiological responses. Cortisol is regulated by the hypothalamic–pituitary–adrenal axis and can be affected by psychological stress, inflammation, sleep patterns, obesity, and environmental factors. Previous studies have suggested that exposure to endocrine-active chemicals may interact with stress-response pathways; however, the biological significance of small hormonal variations requires further investigation (Kortenkamp, 2007).

Overall, the endocrine findings support the hypothesis that personal-care product exposure patterns may be associated with hormonal variability. However, these results should be interpreted as biological associations rather than evidence of endocrine toxicity, and future studies incorporating specific chemical biomarkers and longitudinal hormonal measurements are required.

3.5.4 Ferritin and Vitamin D (Nutritional and Metabolic Markers)

In the present study, ferritin and vitamin D concentrations differed significantly across exposure-pattern groups, suggesting that differences in personal-care product exposure patterns may be associated with variations in nutritional and metabolic-related biomarkers. However, these biomarkers are influenced by multiple physiological factors and should not be interpreted as direct indicators of chemical toxicity.

Ferritin is widely used as an indicator of body iron storage; however, it also functions as an acute-phase protein that increases during inflammatory conditions. Therefore, changes in ferritin concentrations may reflect alterations in iron metabolism, inflammatory status, or both. Previous studies have demonstrated that environmental exposures capable of

inducing oxidative stress and inflammation may influence iron homeostasis through regulation of inflammatory pathways and altered iron metabolism (Ganz & Nemeth, 2015).

The differences observed in ferritin among exposure groups may therefore represent variations in systemic inflammatory status rather than a direct effect of personal-care product exposure. This interpretation is consistent with the current study findings, where exposure indices showed associations with inflammatory markers, particularly CRP.

Vitamin D levels also differed between exposure-pattern groups. Vitamin D plays an important role in immune regulation, inflammatory control, and metabolic homeostasis. Reduced vitamin D status has been associated with increased inflammatory activity and altered immune responses in several populations (Bikle, 2020). However, vitamin D concentration is strongly influenced by sun exposure, dietary intake, body composition, age, and lifestyle factors, which may contribute to the observed differences.

Although some environmental chemicals have been suggested to interfere with vitamin D metabolism through endocrine-related pathways, the current study does not provide evidence for a direct causal relationship between product exposure and vitamin D alteration. The observed differences should therefore be interpreted as biological associations requiring confirmation through longitudinal studies and more specific exposure measurements.

3.5.5 Whole-blood Lead and Cadmium Differences Across Exposure Groups

In the present study, whole-blood lead (Pb) and cadmium (Cd) concentrations showed significant differences among exposure-pattern groups in the primary analysis. However, these associations were not maintained after the batch-exclusion sensitivity analysis, indicating that the observed differences should be interpreted cautiously and considered exploratory findings rather than confirmed exposure-related effects.

Lead and cadmium are widely recognized environmental toxicants with established biological effects involving oxidative stress, inflammation, and disruption of cellular functions. Therefore, evaluating blood concentrations of these metals remains important

in populations with potential exposure to multiple environmental and consumer-product sources (Tchounwou *et al.*, 2012; Genchi *et al.*, 2020).

The initial differences observed between exposure groups may suggest a possible relationship between personal-care product exposure patterns and internal metal burden. Some cosmetic products, pigments, and consumer products have previously been investigated as potential sources of trace metal exposure, particularly when quality control is inadequate or products contain contaminated raw materials (Bocca *et al.*, 2014). However, because the current study did not include direct chemical analysis of the investigated products, a specific source attribution cannot be established.

The lack of persistence after sensitivity analysis highlights the importance of analytical reliability and batch effects in human biomonitoring studies. Variations related to sample processing, analytical batches, or measurement conditions can influence trace-metal results, particularly when concentrations are relatively low. Therefore, the current findings support continued monitoring of Pb and Cd exposure but do not provide sufficient evidence to confirm a direct association with hair-dye or slimming-product use.

These findings emphasize the importance of combining questionnaire-based exposure assessment with direct product analysis and repeated biological measurements in future studies to better characterize potential metal exposure pathways.

3.6 Natural versus Commercial Product Comparisons

To investigate whether the reported origin of personal-care products was associated with differences in biological outcomes, comparisons were performed between natural/herbal and commercial/prepared product categories for hair dyes and slimming products. The evaluated outcomes included inflammatory, hepatic, renal, endocrine, nutritional, and metal-burden indicators.

As presented in Table 3.7, none of the evaluated biomarkers showed statistically significant differences between natural and commercial product users after false discovery rate correction. These findings indicate that the reported product-origin categories alone

were not sufficient to explain variations in biological markers within the studied population.

However, these results should be interpreted cautiously because product classification was based on participant-reported information and did not include chemical characterization of the product formulations. (full results in Appendix D).

Table 3.7. Comparison of biological markers between natural/herbal and commercial/prepared product users

Comparison	Biomarker	Natural n	Natural median	Commercial n	Commercial median	p	FDR q	Rank- biserial correlation
Hair dye: natural vs commercial	CRP (mg/L)	20	6.15	55	5.47	0.461	0.812	0.113
	ALT (U/L)	20	10.85	55	15.40	0.480	0.812	-0.108
	Urea (mg/dL)	20	26.25	55	25.40	0.886	0.974	0.023
	Creatinine (mg/dL)	20	0.70	55	0.62	0.399	0.812	0.128
	eGFR (mL/min/1.73 m ²)	20	113.05	55	111.30	0.314	0.812	0.154
	Cortisol (µg/dL)	20	9.47	55	10.96	0.335	0.812	-0.147
	Vitamin D (ng/mL)	20	13.20	55	14.80	0.628	0.862	-0.075
	Pb (µg/L)	20	14.72	55	14.06	0.806	0.933	0.038
	Cd (µg/L)	20	0.434	55	0.404	0.469	0.812	0.111
Slimming products: natural/herbal vs commercial/prepared	CRP (mg/L)	19	4.89	51	6.07	0.484	0.778	-0.110
	ALT (U/L)	19	21.60	51	19.10	0.672	0.900	-0.067
	Urea (mg/dL)	19	32.90	51	27.40	0.232	0.778	0.188
	Creatinine (mg/dL)	19	0.78	51	0.71	0.792	0.917	0.042
	eGFR (mL/min/1.73 m ²)	19	111.70	51	110.90	0.300	0.778	0.163
	Cortisol (µg/dL)	19	14.90	51	13.70	0.279	0.778	0.170

Comparison	Biomarker	Natural n	Natural median	Commercial n	Commercial median	p	FDR q	Rank- biserial correlation
	Vitamin D (ng/mL)	19	16.70	51	18.90	0.736	0.900	-0.054
	Pb (µg/L)	19	14.93	51	15.47	0.916	0.963	-0.018
	Cd (µg/L)	19	0.313	51	0.348	0.530	0.778	-0.099

Note. Rank-biserial correlation was oriented so that positive values indicate higher values in the first-named natural/herbal group and negative values indicate higher values in the second-named commercial/prepared group.

In the present study, no significant differences were observed between natural/herbal and commercial/prepared product users after false discovery rate correction across the evaluated biochemical, hormonal, inflammatory, and metal-related biomarkers. These findings indicate that product classification based solely on being “natural” or “commercial” may not adequately explain biological variability among users.

The absence of significant differences between product categories does not necessarily indicate that both categories have identical chemical compositions or biological effects. Natural products may contain bioactive plant-derived compounds, whereas commercial products may contain synthetic ingredients, preservatives, colouring agents, or other additives. In addition, variability in manufacturing quality, contamination, and product standardization can influence the chemical characteristics of both categories (Posadzki *et al.*, 2013).

Previous studies have highlighted that the term “natural” does not automatically represent a safer exposure category. Herbal and natural products may contain variable concentrations of active compounds and may occasionally be affected by contamination, adulteration, or differences in preparation methods (Posadzki *et al.*, 2013). Similarly, commercial products are subjected to regulatory requirements, but differences in formulation and frequency of use may influence the magnitude of exposure.

The current findings may also be explained by the relatively broad classification of product origin. Categorizing products as natural or commercial does not capture specific chemical constituents, concentrations, duration of use, or cumulative exposure intensity. Therefore, the absence of significant differences after FDR adjustment should be interpreted as a lack of evidence for a measurable difference between these broad categories rather than evidence of complete safety or equivalence.

Future investigations combining detailed chemical characterization of products with specific biomarkers of exposure may provide a more accurate assessment of whether particular product ingredients contribute to biological changes.

3.7 Spearman Correlations Between Exposure Indices and Biomarkers

Spearman correlation analysis was performed to evaluate the relationships between cumulative exposure indices and biological markers. Because multiple comparisons were assessed, significance was interpreted after Benjamini–Hochberg false discovery rate correction. The significant correlations retained after adjustment are summarized in Table 3.8, while the complete exploratory correlation matrix is provided in Appendix E.

The strongest and most consistent inflammatory association was observed between cosmetic exposure index (CEI) and serum CRP concentration, where higher CEI values were associated with increased CRP levels. Similarly, the combined personal-care product exposure index (PPEI) showed a positive association with CRP, suggesting a relationship between cumulative product exposure patterns and systemic inflammatory status.

Several renal and endocrine associations were also observed. Higher hair-dye exposure index (HDEI) was negatively correlated with eGFR and positively correlated with cortisol concentrations. Slimming-product exposure index (SPEI) demonstrated significant associations with multiple biomarkers, including creatinine, vitamin D, LH, cortisol, and ALT.

Correlations involving whole-blood Pb and Cd were interpreted cautiously. Although some associations were observed in the primary analysis, they were not maintained after

sensitivity analysis excluding the separate metal batch; therefore, they were considered exploratory rather than confirmed exposure–metal relationships.

Table 3.8 FDR-significant Spearman correlations between exposure indices and biomarkers

Exposure index	Outcome	Spearman ρ	p	FDR q
SPEI	Pb ($\mu\text{g/L}$)	0.472	<0.001	<0.001
PPEI	eGFR (mL/min/1.73 m^2)	-0.461	<0.001	<0.001
HDEI	eGFR (mL/min/1.73 m^2)	-0.431	<0.001	<0.001
SPEI	Cd ($\mu\text{g/L}$)	-0.417	<0.001	<0.001
SPEI	Vitamin D (ng/mL)	0.414	<0.001	<0.001
SPEI	Creatinine (mg/dL)	0.395	<0.001	<0.001
PPEI	Creatinine (mg/dL)	0.348	<0.001	0.001
SPEI	LH (mIU/mL)	-0.344	<0.001	0.001
PPEI	Cortisol ($\mu\text{g/dL}$)	0.335	<0.001	0.002
PPEI	CRP (mg/L)	0.327	<0.001	0.002
SPEI	Cortisol ($\mu\text{g/dL}$)	0.298	<0.001	0.008
PPEI	ALT (U/L)	0.291	0.001	0.009
PPEI	Pb ($\mu\text{g/L}$)	0.278	0.002	0.013
CEI	CRP (mg/L)	0.278	0.002	0.013
PPEI	Vitamin D (ng/mL)	0.273	0.003	0.015
PPEI	Urea (mg/dL)	0.267	0.003	0.018
PPEI	Cd ($\mu\text{g/L}$)	-0.251	0.006	0.030
PPEI	Ferritin ($\mu\text{g/L}$)	0.242	0.008	0.038
SPEI	ALT (U/L)	0.239	0.009	0.038
HDEI	Cortisol ($\mu\text{g/dL}$)	0.238	0.009	0.038

The present study identified several significant associations between exposure indices and biological markers, indicating that different personal-care product exposure patterns may have distinct biological relationships. The negative correlation between hair-dye exposure index (HDEI) and estimated glomerular filtration rate (eGFR) suggests a possible association between higher cumulative hair-dye exposure and reduced renal filtration capacity.

This finding is biologically plausible because some chemical constituents associated with personal-care products have been investigated for their potential effects on renal function through oxidative stress, inflammation, and endocrine-related mechanisms. However,

kidney function is influenced by multiple physiological and environmental factors, including age, body composition, hydration status, medication use, and dietary factors. Therefore, this association should be interpreted as a potential biological relationship rather than evidence of direct renal toxicity.

The positive correlation between HDEI and cortisol may indicate a relationship between cumulative hair-dye exposure and stress-related physiological responses. Cortisol is regulated by the hypothalamic–pituitary–adrenal axis and can be influenced by inflammatory status, psychological stress, metabolic conditions, and environmental exposures. Previous studies have suggested that endocrine-disrupting chemicals may interact with hormonal regulation pathways, although the magnitude and clinical relevance of these effects remain under investigation (Gore *et al.*, 2015).

Similarly, slimming-product exposure index (SPEI) showed significant associations with several biomarkers, including ALT, creatinine, cortisol, vitamin D, and LH. These findings suggest that higher slimming-product exposure may be associated with broader biological variation. This observation is consistent with previous reports indicating that weight-loss products, particularly herbal preparations, may contain biologically active compounds with variable effects depending on formulation, dosage, and individual susceptibility (Navarro *et al.*, 2017).

The association between SPEI and ALT is particularly relevant because some herbal and dietary supplements have been linked to liver injury through mechanisms involving oxidative stress, immune-mediated responses, or the presence of contaminants and undeclared ingredients. Nevertheless, because product composition was not chemically verified in the current study, these findings should be interpreted cautiously.

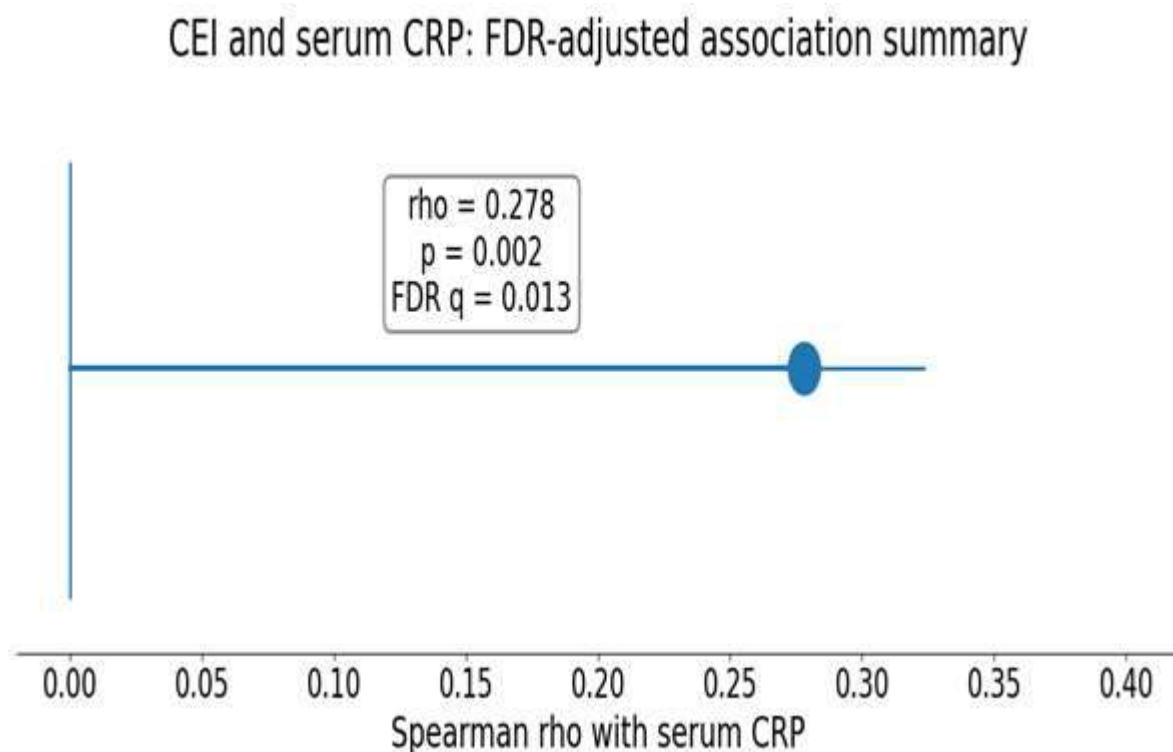


Figure3.4 FDR-adjusted Spearman association summary for CEI and serum CRP

The positive correlation between cosmetic exposure index (CEI) and serum CRP represents one of the most important findings of the current study. Higher CEI values were associated with increased CRP concentrations, suggesting that greater cumulative exposure to cosmetic products may be related to increased systemic inflammatory status.

This association is supported by previous evidence indicating that some chemicals commonly used in personal-care products, including preservatives, plasticizers, and fragrance-related compounds, may influence inflammatory pathways through oxidative stress generation and immune modulation (Gore *et al.*, 2015).

Chronic low-grade inflammation is considered an important biological pathway linking environmental chemical exposure with adverse health outcomes. Increased oxidative stress may activate inflammatory signalling pathways, leading to increased production of inflammatory mediators, including CRP (Calder *et al.*, 2013).

The present finding should nevertheless be interpreted within the limitations of the study design. The CEI represents reported product-use intensity rather than direct measurement of absorbed chemical exposure. Therefore, the observed association does not establish causality but suggests that cumulative cosmetic exposure patterns may be associated with variation in systemic inflammatory status.

The persistence of the CEI–CRP relationship after adjustment in multivariable regression analysis strengthens the evidence that cosmetic exposure patterns may contribute independently to variation in inflammatory markers; however, longitudinal studies and chemical biomonitoring are required to confirm this relationship.

3.8 Multivariable Regression Analysis

Multivariable linear regression analysis was performed to determine whether exposure indices remained independently associated with systemic inflammatory status after adjustment for potential confounding variables. The dependent variable was the logarithmically transformed CRP concentration [$\log(\text{CRP} + 0.1)$], while age, BMI, and menstrual phase were included as covariates in all models.

In separate adjusted models, both CEI and PPEI demonstrated significant positive associations with $\log(\text{CRP} + 0.1)$, indicating that higher cumulative exposure to personal-care products was associated with increased inflammatory marker levels after controlling for demographic and physiological factors.

When exposure indices were entered simultaneously in the mutually adjusted model, CEI and SPEI remained significantly associated with $\log(\text{CRP} + 0.1)$, whereas HDEI no longer showed an independent association. These findings suggest that different categories of personal-care product exposure may contribute differently to variation in systemic inflammatory status; however, the cross-sectional design limits causal interpretation.

Table 3.9. Multivariable linear regression models for log(CRP + 0.1) adjusted for age, BMI, and menstrual phase

Model	Predictor	β	95% CI	P	Approximate change in CRP + 0.1	R ²	Adjusted R ²
Model 1 CEI adjusted	CEI	0.079	0.012 to 0.145	0.021	8.19	0.122	0.091
Model 2 HDEI adjusted	HDEI	0.012	-0.023 to 0.047	0.504	1.20	0.083	0.051
Model 3 SPEI adjusted	SPEI	0.045	-0.001 to 0.092	0.057	4.64	0.108	0.077
Model 4 PPEI adjusted	PPEI	0.039	0.011 to 0.067	0.007	3.94	0.136	0.106
Model 5 combined indices adjusted	CEI	0.088	0.022 to 0.154	0.009	9.17	0.166	0.122
Model 5 combined indices adjusted	HDEI	0.021	-0.014 to 0.055	0.237	2.08	0.166	0.122
Model 5 combined indices adjusted	SPEI	0.053	0.007 to 0.099	0.025	5.42	0.166	0.122
Model 6 combined + whole-blood Pb/Cd	CEI	0.088	0.022 to 0.153	0.009	9.16	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	HDEI	0.023	-0.011 to 0.057	0.188	2.30	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	SPEI	0.062	0.009 to 0.115	0.023	6.39	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	Pb (per 10 µg/L)	0.187	-0.092 to 0.466	0.187	20.58	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	Cd (per 1 µg/L)	0.2505	0.0005 to 0.5005	0.0495	28.47	0.197	0.139

Note. The dependent variable was log-transformed CRP [log(CRP + 0.1)]. Models were adjusted for age, BMI, and menstrual phase. β represents the regression coefficient; $p < 0.05$ was considered statistically significant.

The multivariable regression analysis was performed to determine whether exposure indices remained independently associated with systemic inflammatory status after adjustment for potential confounding factors. The persistence of significant associations after controlling for age, BMI, and menstrual phase strengthens the evidence that specific personal-care product exposure patterns may contribute to variation in CRP concentrations.

In the separate adjusted models, both the cosmetic exposure index (CEI) and the combined personal-care product exposure index (PPEI) showed positive associations with $\log(\text{CRP} + 0.1)$. These findings suggest that higher cumulative exposure to personal-care products may be associated with increased systemic inflammatory activity. The association between cumulative chemical exposure and inflammation is biologically plausible because several compounds present in consumer products have been investigated for their ability to induce oxidative stress, activate inflammatory signalling pathways, and influence immune regulation (Gore *et al.*, 2015).

In the mutually adjusted model, CEI and SPEI remained significantly associated with $\log(\text{CRP} + 0.1)$, whereas HDEI did not retain statistical significance. The persistence of CEI suggests that cosmetic product exposure may represent an important contributor to inflammatory variation in this population. Meanwhile, the continued association of SPEI may reflect the influence of slimming-product exposure, particularly because some weight-loss products contain biologically active ingredients that may affect metabolic and inflammatory pathways (Navarro *et al.*, 2017).

The loss of significance for HDEI after mutual adjustment may indicate that the effect of hair-dye exposure overlaps with other exposure categories or that its independent contribution to inflammatory variation is weaker. This finding highlights the importance of evaluating multiple simultaneous exposures rather than interpreting each product category in isolation.

Although regression analysis provides stronger evidence than simple correlations because it accounts for important covariates, the cross-sectional nature of the study prevents establishing temporal relationships or causality. Therefore, these findings should be interpreted as adjusted associations between exposure patterns and inflammatory status. Longitudinal studies incorporating repeated exposure assessment and chemical biomarkers are required to confirm whether these exposure patterns contribute directly to chronic inflammatory changes.

3.9 Strengths of the Study

- **Comprehensive exposure framework:**

The study evaluated three major personal-care product domains, including cosmetics, hair dyes, and slimming products, allowing a broader assessment of real-world exposure patterns.

- **Multiple exposure indices:**

The development of CEI, HDEI, SPEI, and PPEI provided domain-specific and combined exposure assessments rather than relying on a simple exposed versus unexposed classification.

- **Integrated biomonitoring approach:**

The study combined inflammatory, biochemical, hormonal, nutritional, and metal-burden biomarkers to provide a multidimensional evaluation of biological responses associated with personal-care product exposure patterns.

- **Robust statistical methodology:**

The analytical workflow included appropriate non-parametric testing, false discovery rate (FDR) correction, Dunn–Bonferroni pairwise comparisons, and multivariable regression models adjusted for relevant covariates.

- **Careful interpretation of Pb and Cd findings:**

The study applied unit harmonisation and sensitivity analyses, allowing Pb and Cd results to be interpreted cautiously as internal metal-burden indicators rather than direct evidence of product contamination.

- **Exposure-pattern rather than single-product assessment:**

The study considered overlapping product-use behaviours, reflecting more realistic human exposure scenarios.

3. 10 Summary of Main Findings

The present study identified several important associations between personal-care product exposure patterns and biological indicators among adult women. Whole-blood Pb and Cd concentrations demonstrated measurable internal metal burdens within the studied population; however, metal differences between exposure groups were considered exploratory because they were not retained after sensitivity analysis.

Exposure indices showed considerable variability among participants, with combined hair-dye and slimming-product use representing the largest exposure pattern. Several biochemical, renal, endocrine, and nutritional biomarkers differed across exposure groups, particularly ALT, urea, creatinine, eGFR, cortisol, ferritin, and vitamin D.

Correlation analyses demonstrated significant associations between cumulative exposure indices and selected biological markers, most notably the positive relationships between CEI/PPEI and CRP. Multivariable regression analysis further indicated that some exposure indices remained independently associated with systemic inflammatory status after adjustment for age, BMI, and menstrual phase.

Overall, these findings suggest that patterns of personal-care product exposure may be associated with measurable biological variations; however, longitudinal studies and direct chemical characterization of products are required to establish causal relationships.

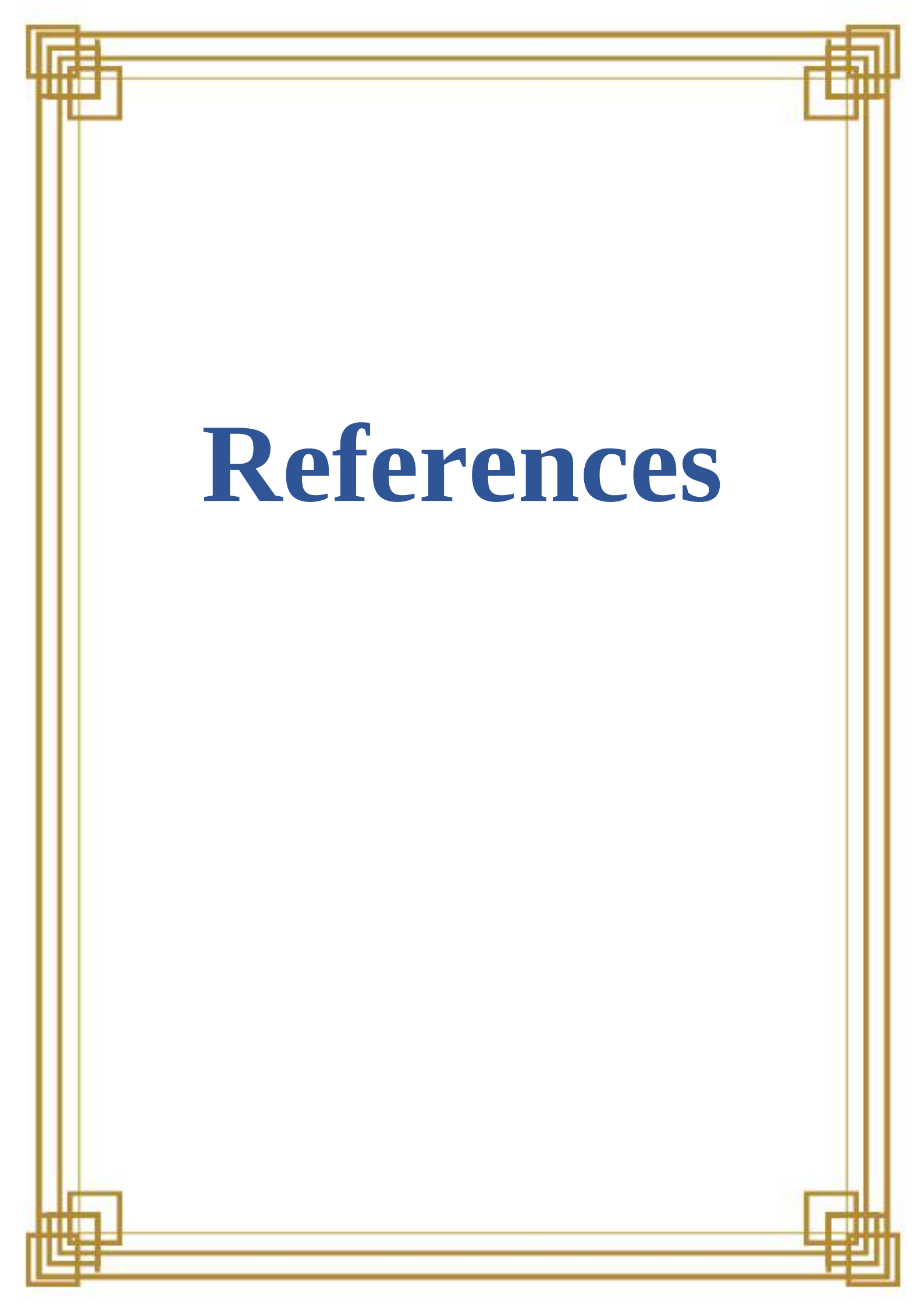
CONCLUSIONS& RECOMMENDATIONS

3.11 Conclusions

1. The study demonstrated that personal-care product exposure patterns were associated with variations in systemic biomarkers among Iraqi women. The most consistent finding was the positive association between the cosmetic exposure index (CEI) and serum CRP, suggesting a potential relationship between cumulative cosmetic exposure and low-grade systemic inflammation.
2. Combined exposure assessment using PPEI provided additional information regarding overall personal-care product exposure; however, it should be interpreted as a composite indicator rather than an independent exposure domain because it integrates multiple product-use categories.
3. The observed associations between exposure patterns and biochemical, renal, hepatic, endocrine, and nutritional biomarkers indicate biological variability associated with personal-care product use patterns. However, due to the cross-sectional design, these findings represent associations rather than evidence of causality or product-induced toxicity.
4. Whole-blood Pb and Cd concentrations reflected internal metal burden among participants. However, the lack of robust associations after sensitivity analysis indicates that these metals should be considered exploratory indicators with uncertain sources rather than confirmed evidence of product-related contamination.
5. No significant differences remained between natural and commercial product users after statistical adjustment, indicating that product origin alone cannot be considered a reliable indicator of safety or hazard.
6. Overall, this study provides baseline biomonitoring evidence regarding personal-care product exposure patterns and biological responses in an Iraqi population. The findings support further investigation using direct chemical analysis, environmental assessment, and longitudinal study designs.

3.12 Recommendations

- Conduct chemical analysis of participant-used cosmetics, hair dyes, and slimming products to identify potential contaminants and chemical exposure sources.
- Include urinary biomarkers of personal-care product chemicals, such as phthalate, paraben, phenol, triclosan, and benzophenone metabolites, to validate questionnaire-based exposure assessment.
- Integrate human biomonitoring with environmental sampling, including household dust, salon environments, water, soil, and food sources, to identify different exposure pathways.
- Perform future studies with larger sample sizes, prospective designs, repeated biomarker measurements, and appropriate low-exposure or unexposed comparison groups.
- Separate slimming products according to route of exposure (oral versus topical) and evaluate product composition, authentication, dosage, and duration of use.
- Classify hair-dye exposure according to product type (natural henna, black henna, permanent dyes, semi-permanent dyes) and include application frequency and occupational exposure factors.
- Investigate the relationship between changes in product-use patterns and subsequent changes in inflammatory, hepatic, renal, and endocrine biomarkers through longitudinal studies.



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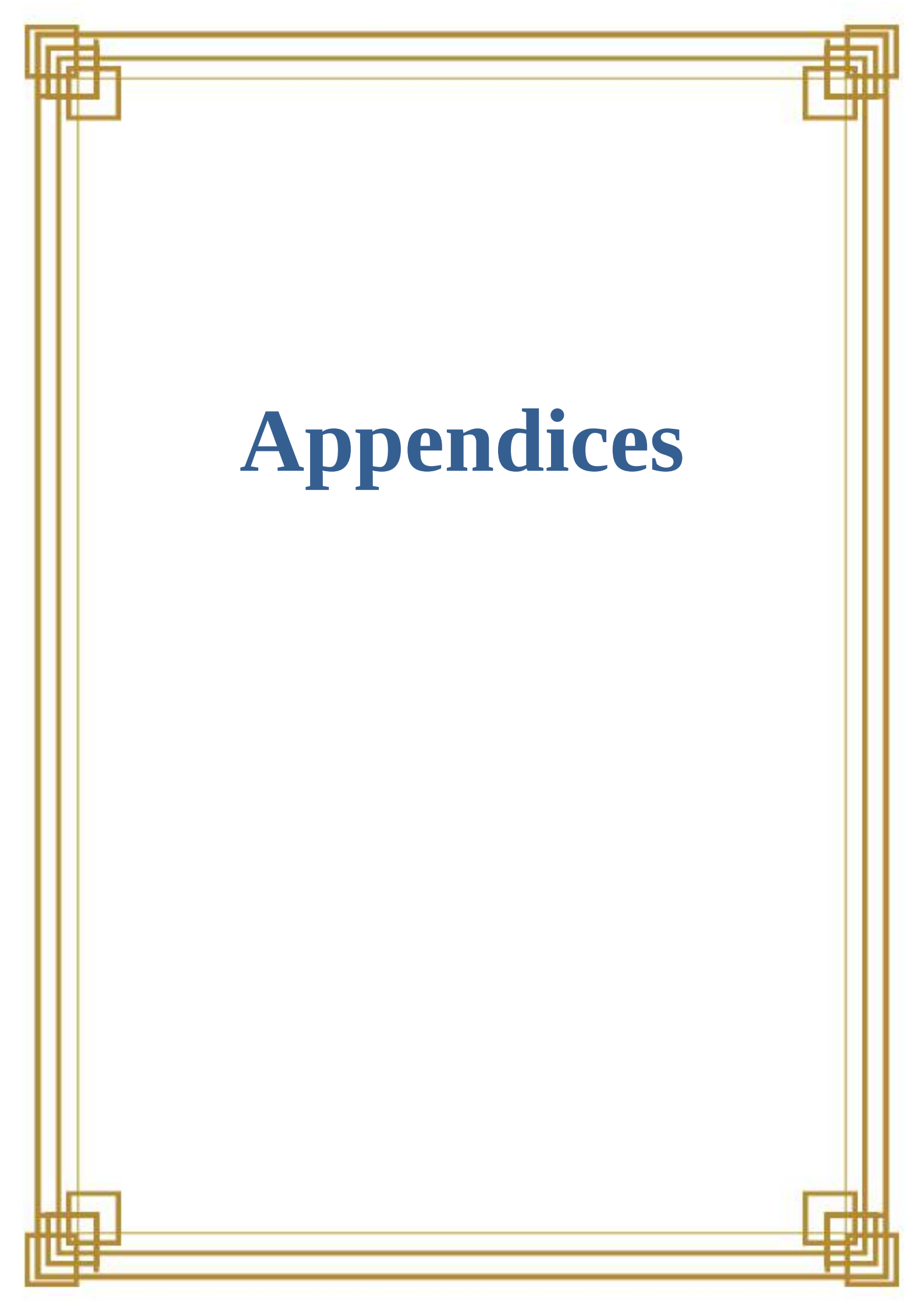
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Appendices

Appendix A

Questionnaire Used for Data Collection

This appendix presents the structured questionnaire used for assessment of personal-care product exposure patterns, including cosmetic products, hair dyes, and slimming-product use among study participants.

The questionnaire collected demographic information, anthropometric variables, product-use behaviour, product origin, duration and frequency of use, exposure routes, and related lifestyle factors.

Demographic and participant information

Questionnaire item	Participant response
Participant ID	_____
Recruitment Source	_____
Age years	_____
BMI kg m2	_____
Residence Area	_____
Occupation	_____

Potential confounding factors

Questionnaire item	Participant response
Salon Worker	_____
Passive Smoking	_____
Recent Infection 4weeks	_____

Medication Use	_____
AntiInflammatory or Steroid	_____
Pregnant or Lactating	_____
Menstrual Phase	_____
Oral Contraceptive Use	_____

Cosmetic product exposure

Questionnaire item	Participant response
Cosmetic User	_____
Cosmetic Main Type	_____
Cosmetic Products Per Day	_____
Cosmetic Frequency Days Week	_____
Cosmetic Duration Years	_____
Cosmetic Source	_____
LeaveOn Products Count	_____
RinseOff Products Count	_____

Hair dye exposure

Questionnaire item	Participant response
Hair Dye User	_____

Hair Dye Type	_____
Dye Frequency Per Year	_____
Dye Duration Years	_____
Dye Application Place	_____
Scalp Contact Score	_____

Slimming product exposure

Questionnaire item	Participant response
Slimming Product User	_____
Slimming Product Type	_____
Slimming Route Score	_____
Slimming Frequency Per Week	_____
Slimming Duration Months	_____
Slimming Source	_____
Labelled Ingredients	_____

STATISTICAL OUTPUT APPENDICES

The following appendices present the complete primary N = 120 statistical outputs retained for transparency and reproducibility. All group counts are consistently defined as hair dye only (n = 34), slimming product only (n = 30), and hair dye plus slimming product (n = 56), and Appendices E and F reflect the corrected P001 indices. Pb/Cd rows remain primary full-sample outputs using instrument-reported values. The two Cd observations between the LOD and LOQ were additionally tested at $LOQ/\sqrt{2}$; this changed the full-model Cd p value from 0.0495 to 0.0501 without materially changing descriptive or rank-based results. The batch-exclusion sensitivity results are reported in Sections 4.5, 4.7, 4.8, and 4.18 and supersede any strong interpretation of appendix metal p or q values.

Appendix C. Full Kruskal-Wallis biomarker comparisons across exposure-pattern groups.

Biomarker	Hair dye only	Slimming only	Hair dye + slimming	H	p	FDR q	ϵ^2	FDR significant
CRP (mg/L)	Hair dye only n=34, median=5.445	Slimming only n=30, median=5.47	Hair dye + slimming n=56, median=6.47	4.05	0.132	0.194	0.017	No
Hb (g/dL)	Hair dye only n=34, median=12.8	Slimming only n=30, median=12.65	Hair dye + slimming n=56, median=12.2	2.06	0.358	0.463	0.000	No
Ferritin (µg/L)	Hair dye only n=34, median=35.2	Slimming only n=30, median=50.05	Hair dye + slimming n=56, median=63.1	10.45	0.005	0.011	0.072	Yes
Serum iron (µg/dL)	Hair dye only n=34, median=90.9	Slimming only n=30, median=87	Hair dye + slimming n=56, median=88.65	0.76	0.684	0.752	0.000	No
ALT (U/L)	Hair dye only n=34, median=11.35	Slimming only n=30, median=18.8	Hair dye + slimming n=56, median=21.8	17.22	<0.001	<0.001	0.130	Yes
AST (U/L)	Hair dye only n=34, median=20.45	Slimming only n=30, median=19.2	Hair dye + slimming n=56, median=20.7	0.66	0.719	0.754	0.000	No
ALP (U/L)	Hair dye only n=34, median=67.85	Slimming only n=30, median=73.25	Hair dye + slimming n=56, median=76.2	5.72	0.057	0.090	0.032	No
Bilirubin (mg/dL)	Hair dye only n=34, median=0.655	Slimming only n=30, median=0.685	Hair dye + slimming n=56, median=0.655	0.04	0.981	0.981	0.000	No
Albumin (g/dL)	Hair dye only n=34, median=4.35	Slimming only n=30, median=4.395	Hair dye + slimming n=56, median=4.415	1.62	0.446	0.545	0.000	No
Urea (mg/dL)	Hair dye only n=34, median=23.4	Slimming only n=30, median=25.7	Hair dye + slimming n=56, median=29.3	13.64	0.001	0.003	0.099	Yes

Biomarker	Hair dye only	Slimming only	Hair dye + slimming	H	p	FDR q	ϵ^2	FDR significant
Creatinine (mg/dL)	Hair dye only n=34, median=0.6	Slimming only n=30, median=0.71	Hair dye + slimming n=56, median=0.75	19.16	<0.001	<0.001	0.147	Yes
eGFR (mL/min/1.73 m ²)	Hair dye only n=34, median=114.8	Slimming only n=30, median=114.8	Hair dye + slimming n=56, median=106.95	20.52	<0.001	<0.001	0.158	Yes
TSH (μ IU/mL)	Hair dye only n=34, median=1.38	Slimming only n=30, median=1.85	Hair dye + slimming n=56, median=1.78	10.10	0.006	0.012	0.069	Yes
Free T3 (pg/mL)	Hair dye only n=34, median=3.09	Slimming only n=30, median=3.21	Hair dye + slimming n=56, median=3.16	3.43	0.180	0.247	0.012	No
Free T4 (ng/dL)	Hair dye only n=34, median=1.19	Slimming only n=30, median=1.075	Hair dye + slimming n=56, median=1.125	1.24	0.539	0.624	0.000	No
LH (mIU/mL)	Hair dye only n=34, median=14.5	Slimming only n=30, median=6.65	Hair dye + slimming n=56, median=6.095	16.85	<0.001	<0.001	0.127	Yes
FSH (mIU/mL)	Hair dye only n=34, median=6.005	Slimming only n=30, median=8.345	Hair dye + slimming n=56, median=8.16	12.40	0.002	0.004	0.089	Yes
Estradiol (pg/mL)	Hair dye only n=34, median=78.85	Slimming only n=30, median=56.9	Hair dye + slimming n=56, median=48.8	5.85	0.054	0.090	0.033	No
Cortisol (μ g/dL)	Hair dye only n=34, median=7.685	Slimming only n=30, median=13.25	Hair dye + slimming n=56, median=13.8	18.62	<0.001	<0.001	0.142	Yes
Vitamin D (ng/mL)	Hair dye only n=34, median=7.55	Slimming only n=30, median=19.8	Hair dye + slimming n=56, median=17.7	27.98	<0.001	<0.001	0.222	Yes
Pb (μ g/L)	Hair dye only n=34, median=5.90	Slimming only n=30, median=15.94	Hair dye + slimming n=56, median=16.48	25.34	<0.001	<0.001	0.200	Yes
Cd (μ g/L)	Hair dye only n=34, median=0.885	Slimming only n=30, median=0.301	Hair dye + slimming n=56, median=0.348	22.74	<0.001	<0.001	0.177	Yes

Note. Results represent primary N = 120 omnibus Kruskal–Wallis comparisons across the three exposure-pattern groups. Every FDR-significant omnibus result was followed by Dunn pairwise tests with Benjamini–Hochberg adjustment across the three group pairs within that biomarker; detailed results follow in Appendix C.1. The Pb and Cd rows are retained for reproducibility, but neither omnibus group effect remained significant after exclusion of the separate 20-sample metal batch (Pb p = 0.953; Cd p = 0.347).

Appendix C.1. Dunn–Benjamini–Hochberg pairwise comparisons following FDR-significant Kruskal–Wallis results.

Biomarker	Group comparison	Median 1	Median 2	Dunn z	BH-adjusted p	Rank-biserial r
Ferritin (µg/L)	Hair dye only vs Slimming only	35.2	50.05	-1.791	0.110	-0.244
Ferritin (µg/L)	Hair dye only vs Hair dye + slimming	35.2	63.1	-3.232	0.004	-0.416
Ferritin (µg/L)	Slimming only vs Hair dye + slimming	50.05	63.1	-1.122	0.262	-0.137
ALT (U/L)	Hair dye only vs Slimming only	11.35	18.8	-2.671	0.011	-0.416
ALT (U/L)	Hair dye only vs Hair dye + slimming	11.35	21.8	-4.115	<0.001	-0.504
ALT (U/L)	Slimming only vs Hair dye + slimming	18.8	21.8	-0.997	0.319	-0.148
Urea (mg/dL)	Hair dye only vs Slimming only	23.4	25.7	-0.136	0.892	0.006
Urea (mg/dL)	Hair dye only vs Hair dye + slimming	23.4	29.3	-3.179	0.004	-0.414
Urea (mg/dL)	Slimming only vs Hair dye + slimming	25.7	29.3	-2.905	0.006	-0.365
Creatinine (mg/dL)	Hair dye only vs Slimming only	0.6	0.71	-2.913	0.005	-0.392
Creatinine (mg/dL)	Hair dye only vs Hair dye + slimming	0.6	0.75	-4.323	<0.001	-0.559
Creatinine (mg/dL)	Slimming only vs Hair dye + slimming	0.71	0.75	-0.930	0.353	-0.104
eGFR (mL/min/1.73 m ²)	Hair dye only vs Slimming only	114.8	114.8	-0.226	0.821	-0.048
eGFR (mL/min/1.73 m ²)	Hair dye only vs Hair dye + slimming	114.8	106.95	3.686	<0.001	0.473
eGFR (mL/min/1.73 m ²)	Slimming only vs Hair dye + slimming	114.8	106.95	3.792	<0.001	0.488
TSH (µIU/mL)	Hair dye only vs Slimming only	1.38	1.85	-2.252	0.036	-0.318
TSH (µIU/mL)	Hair dye only vs Hair dye + slimming	1.38	1.78	-3.103	0.006	-0.396
TSH (µIU/mL)	Slimming only vs Hair dye + slimming	1.85	1.78	-0.488	0.625	-0.058
LH (mIU/mL)	Hair dye only vs Slimming only	14.5	6.65	3.434	<0.001	0.493
LH (mIU/mL)	Hair dye only vs Hair dye + slimming	14.5	6.095	3.747	<0.001	0.475
LH (mIU/mL)	Slimming only vs Hair dye + slimming	6.65	6.095	-0.201	0.841	-0.030
FSH (mIU/mL)	Hair dye only vs Slimming only	6.005	8.345	-3.046	0.003	-0.433
FSH (mIU/mL)	Hair dye only vs Hair dye + slimming	6.005	8.16	-3.133	0.003	-0.400
FSH (mIU/mL)	Slimming only vs Hair dye + slimming	8.345	8.16	0.362	0.717	0.053

Biomarker	Group comparison	Median 1	Median 2	Dunn z	BH-adjusted p	Rank-biserial r
Cortisol (µg/dL)	Hair dye only vs Slimming only	7.685	13.25	-2.518	0.018	-0.393
Cortisol (µg/dL)	Hair dye only vs Hair dye + slimming	7.685	13.8	-4.309	<0.001	-0.528
Cortisol (µg/dL)	Slimming only vs Hair dye + slimming	13.25	13.8	-1.352	0.176	-0.194
Vitamin D (ng/mL)	Hair dye only vs Slimming only	7.55	19.8	-4.503	<0.001	-0.596
Vitamin D (ng/mL)	Hair dye only vs Hair dye + slimming	7.55	17.7	-4.767	<0.001	-0.632
Vitamin D (ng/mL)	Slimming only vs Hair dye + slimming	19.8	17.7	0.404	0.686	0.088
Pb (µg/L)	Hair dye only vs Slimming only	5.90	15.94	-3.986	<0.001	-0.596
Pb (µg/L)	Hair dye only vs Hair dye + slimming	5.90	16.48	-4.740	<0.001	-0.588
Pb (µg/L)	Slimming only vs Hair dye + slimming	15.94	16.48	-0.142	0.887	-0.029
Cd (µg/L)	Hair dye only vs Slimming only	0.885	0.301	4.451	<0.001	0.602
Cd (µg/L)	Hair dye only vs Hair dye + slimming	0.885	0.347	3.845	<0.001	0.508
Cd (µg/L)	Slimming only vs Hair dye + slimming	0.301	0.347	-1.233	0.218	-0.189

Note. Pairwise adjustment was applied separately within each biomarker. A positive rank-biserial r indicates that the first-named group tended to have higher values. Primary Pb/Cd pairwise findings are non-robust because the corresponding omnibus effects were not retained after batch exclusion.

Appendix D.1. Full hair-dye natural-versus-commercial exploratory comparisons.

Biomarker	Natural n	Natural median	Commercial n	Commercial median	p	FDR q	Rank-biserial correlation
CRP (mg/L)	20	6.15	55	5.47	0.461	0.812	0.113
Hb (g/dL)	20	11.95	55	12.70	0.054	0.677	-0.293
Ferritin (µg/L)	20	56.65	55	55.6	0.606	0.862	0.079
Serum iron (µg/dL)	20	90.80	55	91.10	0.986	0.986	0.004
ALT (U/L)	20	10.85	55	15.40	0.480	0.812	-0.108
AST (U/L)	20	18.50	55	21.50	0.645	0.862	-0.071
ALP (U/L)	20	73.60	55	69.20	0.746	0.912	0.050
Bilirubin (mg/dL)	20	0.55	55	0.66	0.062	0.677	-0.285
Albumin (g/dL)	20	4.37	55	4.37	0.986	0.986	0.004
Urea (mg/dL)	20	26.25	55	25.40	0.886	0.974	0.023
Creatinine (mg/dL)	20	0.70	55	0.62	0.399	0.812	0.128
eGFR (mL/min/1.73 m ²)	20	113.05	55	111.30	0.314	0.812	0.154
TSH (µIU/mL)	20	1.60	55	1.61	0.666	0.862	0.066
Free T3 (pg/mL)	20	3.06	55	3.14	0.210	0.772	-0.191
Free T4 (ng/dL)	20	1.03	55	1.14	0.181	0.772	-0.204
LH (mIU/mL)	20	10.20	55	7.58	0.211	0.772	0.191
FSH (mIU/mL)	20	6.50	55	7.60	0.454	0.812	-0.115
Estradiol (pg/mL)	20	45.90	55	62.30	0.165	0.772	-0.212
Cortisol (µg/dL)	20	9.47	55	10.96	0.335	0.812	-0.147
Vitamin D (ng/mL)	20	13.20	55	14.80	0.628	0.862	-0.075
Pb (µg/L)	20	14.72	55	14.06	0.806	0.933	0.038
Cd (µg/L)	20	0.434	55	0.404	0.469	0.812	0.111

Appendix D.2. Full slimming-product natural-versus-commercial exploratory comparisons.

Biomarker	Natural n	Natural median	Commercial n	Commercial median	p	FDR q	Rank-biserial correlation
CRP (mg/L)	19	4.89	51	6.07	0.484	0.778	-0.110
Hb (g/dL)	19	12.40	51	12.60	0.963	0.963	-0.008
Ferritin (µg/L)	19	59.10	51	72.40	0.162	0.778	-0.220
Serum iron (µg/dL)	19	91.30	51	85.30	0.348	0.778	0.148
ALT (U/L)	19	21.60	51	19.10	0.672	0.900	-0.067
AST (U/L)	19	21.70	51	20.50	0.711	0.900	0.059
ALP (U/L)	19	75.50	51	75.40	0.345	0.778	0.149
Bilirubin (mg/dL)	19	0.62	51	0.71	0.025	0.553	-0.351
Albumin (g/dL)	19	4.38	51	4.37	0.921	0.963	-0.017
Urea (mg/dL)	19	32.90	51	27.40	0.232	0.778	0.188
Creatinine (mg/dL)	19	0.78	51	0.71	0.792	0.917	0.042
eGFR (mL/min/1.73 m ²)	19	111.70	51	110.90	0.300	0.778	0.163
TSH (µIU/mL)	19	2.06	51	1.78	0.150	0.778	0.226
Free T3 (pg/mL)	19	3.18	51	3.20	0.472	0.778	-0.114
Free T4 (ng/dL)	19	1.11	51	1.10	0.439	0.778	0.122
LH (mIU/mL)	19	6.85	51	6.89	0.355	0.778	-0.146
FSH (mIU/mL)	19	7.98	51	8.17	0.309	0.778	-0.160
Estradiol (pg/mL)	19	44.80	51	50.10	0.509	0.778	-0.104
Cortisol (µg/dL)	19	14.90	51	13.70	0.279	0.778	0.170
Vitamin D (ng/mL)	19	16.70	51	18.90	0.736	0.900	-0.054
Pb (µg/L)	19	15.47	51	15.47	0.916	0.963	-0.018
Cd (µg/L)	19	0.313	51	0.348	0.530	0.778	-0.099

Note. Rank-biserial correlation was oriented consistently throughout the thesis: positive values indicate higher values in the first-named natural/herbal group, whereas negative values indicate higher values in the commercial/prepared group. The p and FDR q values are unchanged by this direction-standardisation.

Appendix E.1. Full Spearman correlations for CEI.

Biomarker	rho	p	FDR q	FDR significant
CRP (mg/L)	0.278	0.002	0.013	Yes
Hb (g/dL)	-0.009	0.918	0.968	No
Ferritin (µg/L)	-0.052	0.571	0.749	No
Serum iron (µg/dL)	-0.032	0.732	0.866	No
ALT (U/L)	0.052	0.575	0.749	No
AST (U/L)	-0.103	0.263	0.484	No
ALP (U/L)	0.054	0.559	0.749	No
Bilirubin (mg/dL)	-0.102	0.269	0.484	No
Albumin (g/dL)	0.067	0.465	0.730	No
Urea (mg/dL)	0.121	0.186	0.390	No
Creatinine (mg/dL)	0.111	0.229	0.457	No
eGFR (mL/min/1.73 m ²)	-0.099	0.282	0.488	No
TSH (µIU/mL)	-0.010	0.911	0.968	No
Free T3 (pg/mL)	0.061	0.510	0.737	No
Free T4 (ng/dL)	0.094	0.310	0.514	No
LH (mIU/mL)	0.099	0.283	0.488	No
FSH (mIU/mL)	-0.045	0.624	0.796	No
Estradiol (pg/mL)	0.020	0.825	0.919	No
Cortisol (µg/dL)	-0.132	0.149	0.338	No
Vitamin D (ng/mL)	0.005	0.957	0.968	No
Pb (µg/L)	-0.059	0.519	0.737	No
Cd (µg/L)	-0.007	0.942	0.968	No

Appendix E.2. Full Spearman correlations for HDEI.

Biomarker	Rho	p	FDR q	FDR significant
CRP (mg/L)	0.198	0.030	0.106	No
Hb (g/dL)	-0.037	0.688	0.865	No
Ferritin (µg/L)	0.158	0.085	0.241	No
Serum iron (µg/dL)	0.054	0.559	0.749	No
ALT (U/L)	0.154	0.092	0.249	No
AST (U/L)	-0.060	0.512	0.737	No
ALP (U/L)	0.103	0.265	0.484	No
Bilirubin (mg/dL)	-0.029	0.754	0.873	No
Albumin (g/dL)	0.011	0.909	0.968	No
Urea (mg/dL)	0.185	0.043	0.139	No
Creatinine (mg/dL)	0.073	0.429	0.690	No
eGFR (mL/min/1.73 m ²)	-0.431	<0.001	<0.001	Yes
TSH (µIU/mL)	0.064	0.485	0.737	No
Free T3 (pg/mL)	0.033	0.720	0.866	No
Free T4 (ng/dL)	0.104	0.258	0.484	No
LH (mIU/mL)	-0.006	0.948	0.968	No
FSH (mIU/mL)	0.033	0.717	0.866	No
Estradiol (pg/mL)	-0.098	0.289	0.488	No
Cortisol (µg/dL)	0.238	0.009	0.038	Yes
Vitamin D (ng/mL)	0.008	0.933	0.968	No
Pb (µg/L)	0.051	0.579	0.749	No
Cd (µg/L)	0.026	0.777	0.877	No

Appendix E.3. Full Spearman correlations for SPEI.

Biomarker	rho	p	FDR q	FDR significant
CRP (mg/L)	0.154	0.093	0.249	No
Hb (g/dL)	-0.062	0.498	0.737	No
Ferritin (µg/L)	0.204	0.026	0.098	No
Serum iron (µg/dL)	0.027	0.773	0.877	No
ALT (U/L)	0.239	0.009	0.038	Yes
AST (U/L)	-0.106	0.250	0.484	No
ALP (U/L)	0.128	0.162	0.348	No
Bilirubin (mg/dL)	-0.053	0.564	0.749	No
Albumin (g/dL)	0.014	0.881	0.968	No
Urea (mg/dL)	0.144	0.117	0.287	No
Creatinine (mg/dL)	0.395	<0.001	<0.001	Yes
eGFR (mL/min/1.73 m ²)	-0.198	0.030	0.106	No
TSH (µIU/mL)	0.184	0.044	0.139	No
Free T3 (pg/mL)	0.213	0.020	0.083	No
Free T4 (ng/dL)	0.000	1.000	1.000	No
LH (mIU/mL)	-0.344	<0.001	0.001	Yes
FSH (mIU/mL)	0.207	0.023	0.094	No
Estradiol (pg/mL)	-0.129	0.161	0.348	No
Cortisol (µg/dL)	0.298	<0.001	0.008	Yes
Vitamin D (ng/mL)	0.414	<0.001	<0.001	Yes
Pb (µg/L)	0.472	<0.001	<0.001	Yes
Cd (µg/L)	-0.417	<0.001	<0.001	Yes

Appendix E.4. Full Spearman correlations for PPEI.

Biomarker	rho	p	FDR q	FDR significant
CRP (mg/L)	0.327	<0.001	0.002	Yes
Hb (g/dL)	-0.061	0.511	0.737	No
Ferritin (µg/L)	0.242	0.008	0.038	Yes
Serum iron (µg/dL)	0.034	0.709	0.866	No
ALT (U/L)	0.291	0.001	0.009	Yes
AST (U/L)	-0.148	0.106	0.266	No
ALP (U/L)	0.149	0.105	0.266	No
Bilirubin (mg/dL)	-0.072	0.431	0.690	No
Albumin (g/dL)	0.031	0.738	0.866	No
Urea (mg/dL)	0.267	0.003	0.018	Yes
Creatinine (mg/dL)	0.348	<0.001	0.001	Yes
eGFR (mL/min/1.73 m ²)	-0.461	<0.001	<0.001	Yes
TSH (µIU/mL)	0.174	0.057	0.172	No
Free T3 (pg/mL)	0.142	0.121	0.289	No
Free T4 (ng/dL)	0.115	0.213	0.436	No
LH (mIU/mL)	-0.184	0.044	0.139	No
FSH (mIU/mL)	0.162	0.078	0.228	No
Estradiol (pg/mL)	-0.132	0.150	0.338	No
Cortisol (µg/dL)	0.335	<0.001	0.002	Yes
Vitamin D (ng/mL)	0.273	0.003	0.015	Yes
Pb (µg/L)	0.278	0.002	0.013	Yes
Cd (µg/L)	-0.251	0.006	0.030	Yes

Appendix F.1. Regression assumption and influence diagnostics.

Model	Maximum VIF	Shapiro–Wilk p	Breusch–Pagan p	Durbin–Watson	Maximum Cook’s D	n with Cook’s D > 4/N
Model 1 CEI adjusted	1.04	<0.001	0.483	1.93	0.086	7
Model 2 HDEI adjusted	1.65	<0.001	0.422	1.89	0.166	8
Model 3 SPEI adjusted	1.13	<0.001	0.252	1.84	0.090	6
Model 4 PPEI adjusted	1.88	<0.001	0.306	1.98	0.097	8
Model 5 combined indices adjusted	1.91	<0.001	0.350	1.99	0.128	8
Model 6 combined + whole-blood Pb/Cd	1.92	<0.001	0.248	1.93	0.187	9

Note. VIF values below 5 indicate no concerning multicollinearity. Breusch–Pagan p values above 0.05 do not indicate heteroscedasticity. Residual Shapiro–Wilk p values were <0.001 in every model. No Cook’s distance exceeded 1.0; 4/N was used only as a sensitivity-screening threshold.

Appendix F.2. HC3 robust standard errors and Cook's-distance influence sensitivity for primary predictors.

Model	Predictor	β	OLS p	HC3 95% CI	HC3 p	Cook-exclusion p (n)
Model 1 CEI adjusted	CEI	0.079	0.021	0.012 to 0.146	0.021	0.007 (n=113)
Model 2 HDEI adjusted	HDEI	0.012	0.504	-0.029 to 0.053	0.570	0.091 (n=112)
Model 3 SPEI adjusted	SPEI	0.045	0.057	-0.001 to 0.092	0.057	0.093 (n=114)
Model 4 PPEI adjusted	PPEI	0.039	0.007	0.012 to 0.066	0.005	0.002 (n=112)
Model 5 combined indices adjusted	CEI	0.088	0.009	0.023 to 0.152	0.008	0.003 (n=112)
Model 5 combined indices adjusted	HDEI	0.021	0.237	-0.017 to 0.058	0.281	0.153 (n=112)
Model 5 combined indices adjusted	SPEI	0.053	0.025	0.005 to 0.101	0.031	0.038 (n=112)
Model 6 combined + whole-blood Pb/Cd	CEI	0.088	0.009	0.023 to 0.152	0.008	0.006 (n=111)
Model 6 combined + whole-blood Pb/Cd	HDEI	0.023	0.188	-0.016 to 0.061	0.244	0.019 (n=111)
Model 6 combined + whole-blood Pb/Cd	SPEI	0.062	0.023	0.002 to 0.122	0.042	0.006 (n=111)
Model 6 combined + whole-blood Pb/Cd	Pb per 10 $\mu\text{g/L}$	1.87	1.87	-2.45 to 6.19	3.93	3.47 (n=111)
Model 6 combined + whole-blood Pb/Cd	Cd per 1 $\mu\text{g/L}$	0.251	0.050	-0.007 to 0.508	0.057	0.092 (n=111)

Note. HC3 coefficients equal the OLS point estimates but use heteroscedasticity-consistent standard errors. Cook-exclusion models removed observations with Cook's distance $> 4/N$ for that specific model. CEI and PPEI were stable; SPEI remained supported in the mutually adjusted models but not as a single index; HDEI was model-sensitive; Pb and Cd were not robust.

Appendix F.3. Full multivariable regression output including covariates.

Model	Predictor	β	95% CI	p	Approximate % Change in CRP + 0.1	R ²	Adjusted R ²
Model 1 CEI adjusted	CEI	0.079	0.012 to 0.145	0.021	8.19	0.122	0.091
Model 1 CEI adjusted	Age (years)	0.011	-0.009 to 0.032	0.262	1.16	0.122	0.091
Model 1 CEI adjusted	BMI (kg/m ²)	0.051	0.004 to 0.099	0.035	5.25	0.122	0.091
Model 1 CEI adjusted	Menstrual phase: ovulatory	-0.248	-0.605 to 0.109	0.171	-21.97	0.122	0.091
Model 2 HDEI adjusted	HDEI	0.012	-0.023 to 0.047	0.504	1.20	0.083	0.051
Model 2 HDEI adjusted	Age (years)	0.010	-0.016 to 0.036	0.445	1.01	0.083	0.051
Model 2 HDEI adjusted	BMI (kg/m ²)	0.055	0.007 to 0.104	0.025	5.70	0.083	0.051
Model 2 HDEI adjusted	Menstrual phase: ovulatory	-0.249	-0.614 to 0.116	0.179	-22.03	0.083	0.051
Model 3 SPEI adjusted	SPEI	0.045	-0.001 to 0.092	0.057	4.64	0.108	0.077
Model 3 SPEI adjusted	Age (years)	0.009	-0.013 to 0.030	0.427	0.86	0.108	0.077
Model 3 SPEI adjusted	BMI (kg/m ²)	0.062	0.014 to 0.110	0.011	6.43	0.108	0.077
Model 3 SPEI adjusted	Menstrual phase: ovulatory	-0.244	-0.604 to 0.115	0.181	-21.69	0.108	0.077
Model 4 PPEI adjusted	PPEI	0.039	0.011 to 0.067	0.007	3.94	0.136	0.106
Model 4 PPEI adjusted	Age (years)	-0.010	-0.037 to 0.017	0.463	-1.00	0.136	0.106
Model 4 PPEI adjusted	BMI (kg/m ²)	0.054	0.007 to 0.101	0.025	5.51	0.136	0.106
Model 4 PPEI adjusted	Menstrual phase: ovulatory	-0.262	-0.616 to 0.092	0.145	-23.06	0.136	0.106
Model 5 combined indices adjusted	CEI	0.088	0.022 to 0.154	0.009	9.17	0.166	0.122
Model 5 combined indices adjusted	HDEI	0.021	-0.014 to 0.055	0.237	2.08	0.166	0.122
Model 5 combined indices adjusted	SPEI	0.053	0.007 to 0.099	0.025	5.42	0.166	0.122
Model 5 combined indices adjusted	Age (years)	-0.006	-0.033 to 0.021	0.642	-0.63	0.166	0.122
Model 5 combined indices adjusted	BMI (kg/m ²)	0.054	0.007 to 0.101	0.025	5.54	0.166	0.122

Model	Predictor	β	95% CI	p	Approximate % Change in CRP + 0.1	R ²	Adjusted R ²
Model 5 combined indices adjusted	Menstrual phase: ovulatory	-0.258	-0.609 to 0.093	0.149	-22.71	0.166	0.122
Model 6 combined + whole-blood Pb/Cd	CEI	0.088	0.022 to 0.153	0.009	9.16	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	HDEI	0.023	-0.011 to 0.057	0.188	2.30	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	SPEI	0.062	0.009 to 0.115	0.023	6.39	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	Pb (per 10 µg/L)	1.87	-0.92 to 4.66	205.8	20.58	1.97	1.39
Model 6 combined + whole-blood Pb/Cd	Cd (per 1 µg/L)	0.2505	0.0005 to 0.5005	0.0495	28.47	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	Age (years)	-0.007	-0.034 to 0.019	0.580	-0.75	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	BMI (kg/m ²)	0.056	0.010 to 0.103	0.019	5.80	0.197	0.139
Model 6 combined + whole-blood Pb/Cd	Menstrual phase: ovulatory	-0.249	-0.597 to 0.098	0.158	-22.07	0.197	0.139

Appendix G. Heavy-metal calibration and instrument documentation.

This appendix documents the element-specific seven-point calibration curves used for whole-blood Pb and Cd analysis by graphite-furnace atomic absorption spectrometry (GFAAS). The calibration characteristics reproduce the final instrument file used to establish linearity and the method-specific LOD/LOQ values reported in Section 3.12.

Table G.1 Analytical calibration characteristics for Pb and Cd.

Element	Calibration range (µg/L)	Regression equation	R ²	Syx	LOD / LOQ (µg/L)
Pb	0–40	$y = 0.01799x + 0.00016$	0.99999	0.000506	0.093 / 0.281
Cd	0–8	$y = 0.08494x + 0.00028$	0.99999	0.000533	0.021 / 0.063

GFAAS calibration curves for whole-blood Pb and Cd

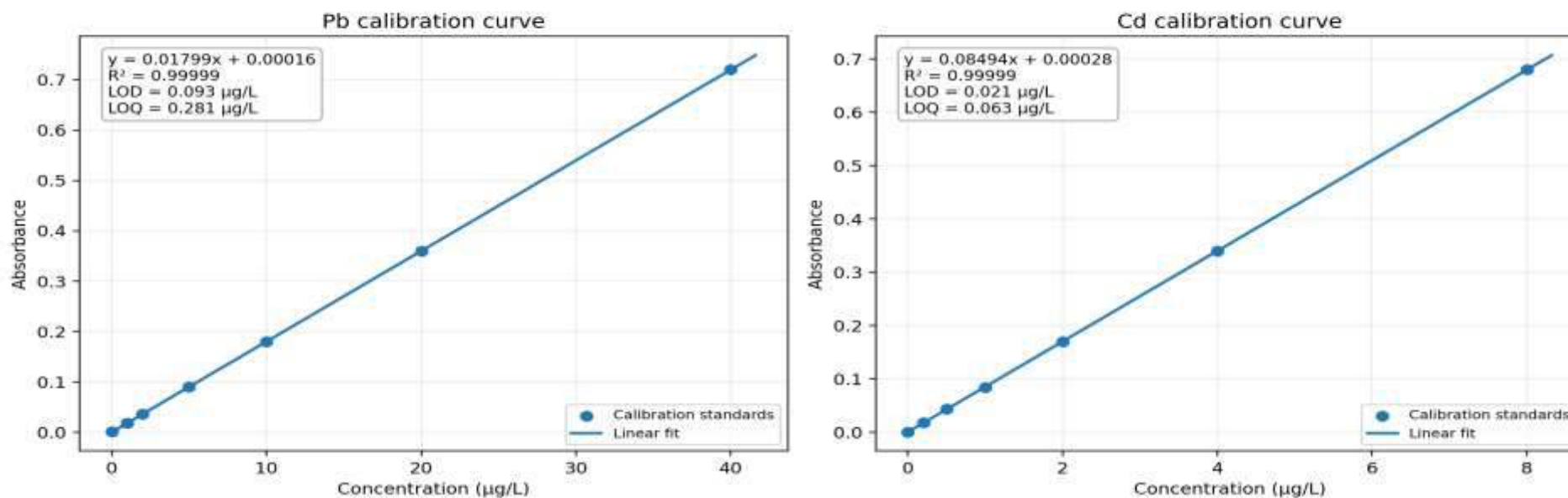


Figure G.1 Seven-point GFAAS calibration curves for Pb and Cd, including the fitted equations, R², LOD, and LOQ.

Appendix H. Additional Materials Related to the Study



Figure H.1. Collected Blood Samples (Complete Set)

Note: Participants' names and personal identifiers have been redacted to protect privacy and maintain confidentiality.

Appendix H (Continued)



Figure H.2. Blood Samples in Rack

Note: Participants' names and personal identifiers have been redacted to protect privacy and maintain confidentiality.

Appendix H (Continued)



Figure H.3. Official Approval Letter for Conducting the Study (Arabic Original)

الخلاصة

الخلاصة

هدفت هذه الدراسة إلى تقييم العلاقة بين أنماط التعرض لمنتجات العناية الشخصية، بما في ذلك مستحضرات التجميل، وصبغات الشعر، ومنتجات التنحيف، وبين التغيرات في المؤشرات الالتهابية والبيوكيميائية والهرمونية والغذائية، إضافة إلى تراكيز الرصاص والكاديوم في الدم لدى النساء البالغات في محافظة بابل، العراق. أجريت الدراسة وفق تصميم مقطعي تحليلي ضمن إطار المراقبة الحيوية البيئية، وشملت 120 مشاركة بمتوسط عمر بلغ (34.38 ± 7.90) سنة ومتوسط مؤشر كتلة جسم (26.56 ± 3.33) كغم/م². تم تصنيف المشاركات وفق أنماط التعرض إلى مستخدمات صبغات الشعر فقط ($n=34$)، ومستخدمات منتجات التنحيف فقط ($n=30$)، ومستخدمات صبغات الشعر ومنتجات التنحيف معاً ($n=56$). كما تم تقييم التعرض لمستحضرات التجميل كمجال تعرض مستقل ومتداخل باستخدام مؤشرات كمية شملت مؤشر التعرض لمستحضرات التجميل (CEI)، ومؤشر التعرض لصبغات الشعر (HDEI)، ومؤشر التعرض لمنتجات التنحيف (SPEI)، والمؤشر المركب للتعرض للمنتجات الشخصية (PPEI). تم قياس مستوى بروتين سي التفاعلي (CRP) إلى جانب مجموعة واسعة من المؤشرات الكبدية والكلى والهرمونية والدموية والغذائية، إضافة إلى قياس الرصاص (Pb) والكاديوم (Cd) في الدم الكامل. بلغ متوسط تركيز CRP (3.53 ± 6.02) ملغم/لتر، في حين بلغ متوسط تركيز الرصاص (6.8 ± 14.3) ميكروغرام/لتر، ومتوسط الكاديوم (0.608 ± 0.763) ميكروغرام/لتر. تضمنت التحليلات الإحصائية اختبارات المقارنة غير المعلمية، وتحليل الارتباط سبيرمان، وتصحيح معدل الاكتشاف الكاذب (Benjamini-Hochberg FDR)، إضافة إلى نماذج الانحدار الخطي متعدد المتغيرات بعد ضبط العمر، ومؤشر كتلة الجسم، ومرحلة الدورة الشهرية.

أظهرت النتائج أن أقوى ارتباط التهابي كان بين التعرض لمستحضرات التجميل ومستوى CRP؛ إذ أظهر مؤشر التعرض لمستحضرات التجميل (CEI) ارتباطاً إيجابياً مع CRP ($p=0.278$)، $p=0.002$ ، $q=0.013$ ، كما أظهر المؤشر المركب للتعرض للمنتجات الشخصية (PPEI) ارتباطاً إيجابياً أقوى ($p=0.327$)، $p<0.001$ ، $q=0.002$ وفي نماذج الانحدار المعدلة، ارتبط كل من CEI و PPEI بزيادة قيمة $\log(CRP+0.1)$ ، حيث بلغ معامل الانحدار لـ CEI ($\beta=0.079$)، $CI: 0.012-0.145$ ، $p=0.021$ ، ولـ PPEI ($\beta=0.039$)، $CI: 0.011-0.067$ ، $p=0.007$. عند إدخال مؤشرات التعرض بشكل مشترك في النموذج، احتفظ كل من CEI و SPEI بالارتباط الإحصائي، بينما لم يظهر HDEI ارتباطاً مستقلاً.

بالنسبة للمعادن الثقيلة، كانت جميع قياسات الرصاص أعلى من حد القياس الكمي، بينما تم تسجيل حالتين للكاديوم بين حد الكشف وحد القياس الكمي. وعلى الرغم من ظهور بعض الارتباطات الأولية بين الرصاص والكاديوم وأنماط التعرض، فإن هذه النتائج لم تبقَ مستقرة بعد تحليلات الحساسية، وخاصة بعد استبعاد الدفعة المختبرية المنفصلة المكونة من 20 عينة؛ إذ أصبحت الفروق بين مجموعات التعرض غير معنوية للرصاص ($p=0.953$).

والكادميوم ($p=0.347$) لذلك تم تفسير النتائج المتعلقة بالمعادن على أنها ملاحظات استكشافية غير مستقرة، وليست دليلاً على وجود تعرض معدني مرتبط بشكل مباشر بمنتجات معينة. كما لم تظهر فروق معنوية بين المنتجات الطبيعية والتجارية بعد تطبيق تصحيح FDR.

تستنتج هذه الدراسة أن أنماط استخدام منتجات العناية الشخصية، وبالأخص التعرض لمستحضرات التجميل المقاس بواسطة CEI، ترتبط بالتباين في المؤشرات الالتهابية الجهازية لدى النساء ضمن مجتمع الدراسة. ومع ذلك، فإن الطبيعة المقطعية للدراسة وعدم وجود مجموعة غير متعرضة بشكل كامل تحد من إمكانية إثبات العلاقة السببية أو تحديد مصدر التعرض بصورة قاطعة. وتوصي الدراسة بإجراء أبحاث مستقبلية تتضمن التحليل الكيميائي المباشر للمنتجات المستخدمة من قبل المشاركات، وأخذ عينات بيئية مرافقة، واستخدام أدوات تعرض موثقة، وقياسات حيوية متكررة، ودراسات طولية لتحديد مسارات التعرض والتأثيرات البيولوجية بصورة أكثر دقة.



جمهورية العراق

وزارة التعليم العالي والبحث العلمي جامعة

القادسية كلية العلوم / قسم البيئية

تقييم التعرض لمنتجات العناية الشخصية واستخدام صبغات الشعر
ومنتجات التحفيف وعلاقتها بمستويات الرصاص والكاديوم في الدم
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العراق.

رسالة

مقدمة إلى مجلس كلية العلوم جامعة القادسية كجزء من متطلبات درجة الماجستير
في علوم البيئية

من قبل

زينب قصي طاحب الحداد

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