Indonesian Journal of Pharmaceutics
IdJP
ISSN : 2597-8748

Vol. 7, Issue 2, 2025 (64-76)
http://journal.unpad.ac.id/IdJP

Unlocking the Secrets to Truly Healthy Facial Skin: A Comprehensive Review of Its Influences

Stella Aurelia Huang[1], Rasya Salma Fauziyyah[1] , Khanna Ragita Aurelia[1], Eri Amalia[2*]

[1]Undergraduate Student, Faculty of Pharmacy, Universitas Padjadjaran, Jl. Raya Bandung-Sumedang Km. 21, Bandung 45363, Indonesia
[2]Department of Pharmaceutics and Pharmaceutical Technology, Faculty of Pharmacy, Universitas Padjadjaran, Jl. Raya Bandung-Sumedang Km. 21, Bandung 45363, Indonesia

Submitted: 21/04/2025, Revised: 30/4/2025, Accepted: 10/05/2025, Published: 30/05/2025

ABSTRACT

Facial skin health results from a complex interaction between intrinsic biological processes and extrinsic environmental exposures, extending beyond the effects of topical skincare alone. This narrative review synthesizes evidence from original studies published between 2015 and 2025, retrieved from PubMed and Google Scholar using targeted search terms related to internal factors such as hydration status, hormonal regulation, and genetic variation, as well as external influences including ultraviolet radiation, air pollution, and light absorption. Eligible studies were full-text original articles in English or Indonesian that examined skin-active ingredients or physiological mechanisms affecting facial skin. The literature consistently identifies hydration as a fundamental determinant of skin elasticity, smoothness, and luminosity, mediated through corneocyte organization, lipid barrier integrity, and aquaporin-3–regulated water transport. Hormonal factors, particularly estrogen, play a critical role in maintaining dermal quality by stimulating collagen synthesis, enhancing skin thickness, and reducing oxidative damage. Genetic variability further contributes to individual differences in skin characteristics by influencing collagen density, antioxidant defense systems, pigmentation pathways, and inflammatory responses. External stressors significantly modulate these intrinsic processes, with ultraviolet radiation and air pollution accelerating skin aging through oxidative stress, collagen degradation, barrier disruption, and pigmentary alterations. In addition, light absorption and reflection at the skin surface, affected by sebum composition and optical properties of the stratum corneum, influence perceived skin tone and radiance. Collectively, the evidence underscores the importance of understanding how intrinsic and extrinsic factors interact to shape facial skin health. A holistic, evidence-based approach that integrates topical treatments with internal health optimization, hormonal balance, lifestyle modification, and personalized care strategies is essential for achieving and maintaining optimal facial skin condition.

Keywords: Healthy facial skin, intrinsic factors, extrinsic factors, skin hydration, holistic skincare.

*Corresponding author,
e-mail: amalia@unpad.ac.id E. Amalia)

https://doi.org/10.24198/idjp.v7i2.69834

1. Introduction

The skin, as the largest organ of the human body, constitutes a dynamic and multifunctional barrier that envelops the entire external surface (1). The skin does more than just protect us from physical, chemical, microbial, and ultraviolet (UV) damage. It also helps regulate body temperature, keep an eye on the immune system, sense things, and control the flow of water through the skin, all of which help keep the body in balance (2). The skin is made up of three layers: the epidermis, the dermis, and the hypodermis. Each layer has its own unique anatomical and physiological features that are important for skin function(1).

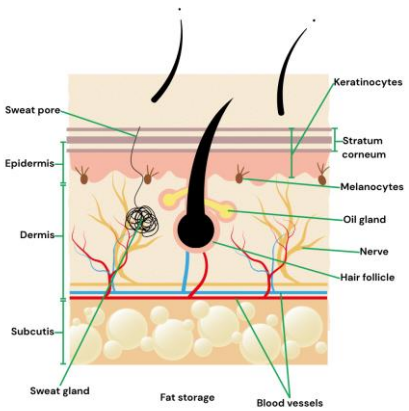

Figure 1. Anatomy of Skin (1)

These layers collectively sustain mechanical resilience, biochemical defense, and epidermal renewal. As shown in figure 1. The epidermis, which is mostly made up of keratinocytes, is the body's first line of defense against germs and other things. The outermost stratum corneum is made up of corneocytes that have fully developed and are embedded in a lipid-rich matrix. This forms a barrier that lets some things through but not others and is constantly being replaced through desquamation (1). The dermis is a network of connective tissue that is divided into papillary and reticular layers. It contains blood vessels, nerves, sebaceous glands, and other structures that serve sensory, thermoregulatory, and reparative processes (3). The hypodermis, or subcutaneous tissue, predominantly composed of adipocytes and loose connective tissue, provides mechanical cushioning, energy storage, and thermoregulation, while integrating vascular and neural pathways essential for deeper tissue support (1).

To better understand what makes skin healthy, experts have identified three main areas of focus which are visual, mechanical, and topographical qualities. Healthy skin has even pigmentation, which means there are no dark spots or red spots. The skin is good at reflecting light, which makes it look bright or glowing. Healthy skin looks fresh and moist, not dry or too shiny, and it displays evidence of being well-hydrated (4). Healthy skin is strong and elastic, and it quickly stretches and goes back to its natural shape. It doesn't feel loose or saggy, which means that the skin's structure is still solid. Healthy skin has a smooth surface with no rough spots. It has fewer wrinkles, smaller pores, and no fine or crepey textures. These traits make the skin look young and well-maintained (4).

The benefits of healthy skin go far beyond looks. Skin serves as the body's first line of defense, protecting it from bacteria, viruses, UV rays, pollutants, and physical injuries. It also plays a key role in body temperature control and helps prevent excessive water loss. Furthermore, healthy skin is essential for the body's immune response and vitamin D production, both of which support overall health (5). From a psychological point of view, skin that appears smooth, radiant, and youthful can improve a person's confidence and mental

well-being. It also helps with sensation such as touch, temperature, and pain and supports faster wound healing.

Despite its regenerative capabilities, the skin remains susceptible to a myriad of pathophysiological conditions across the lifespan. Common dermatological disorders—including acne, atopic dermatitis, vitiligo, melasma, and rosacea—exemplify disruptions in barrier integrity, inflammatory regulation, pigmentation, and microbial equilibrium. Acne, for instance, arises from hyperseborrhea, follicular occlusion, and microbial colonization by Propionibacterium acnes (6). Eczema, or atopic dermatitis, reflects a genetic-immune-environmental interplay that compromises barrier proteins and elicits chronic inflammation (7). Disorders such as vitiligo and melasma result from melanocyte dysfunction or dysregulated pigment metabolism, often exacerbated by hormonal or phototoxic stimuli (8), while rosacea is underpinned by neurovascular dysregulation and persistent inflammation (9).

To maintain optimal skin health, it is essential to consider both internal and external influences. Skin health depends on a balance between damaging exposures and the body's intrinsic defenses. Key intrinsic factors that affect skin health are epidermal barrier, immune system, metabolic processes, genetic, and hormonal. Besides, skin hydration has been related to healthier looking skin. Extrinsic factors from the environmental stressors such as Sunlight (UV), air pollutants (ozone, particulate matter), and light absorption generate reactive oxygen species (ROS) that damage DNA, proteins, lipids and trigger inflammation, accelerating skin aging and diseases like psoriasis or atopic dermatitis (10–12).

This review prioritized examining skin hydration and its associated parameters over immune-related factors because hydration research offers a substantially larger and more established body of literature. Studies in dermatological and cosmetic contexts have extensively measured outcomes such as moisture levels, pigmentation changes, and the efficacy of topical treatments. This wealth of peer-reviewed evidence supports a rigorous, consistent analytical framework, whereas immune-related investigations are often more complex, variable, and less standardized. Focusing on hydration aligns the review with a well-supported scientific foundation and ensures methodological robustness. Additionally, by synthesizing intrinsic and extrinsic influences on facial skin health, the article provides a comprehensive literature review to help individuals understand and optimize their skincare strategies for healthier, balanced skin.

2. Method

Methods The article search was conducted online using the Google Scholar and PubMed databases with keywords related to active ingredients that affect facial skin. These factors were divided into internal factors (hydration, hormones, and genetics) and external factors (sun radiation, air pollution, and light absorption). The selected articles met specific inclusion and exclusion criteria. The inclusion criteria were articles published within the last 10 years (2015–2025), original research studies, focused on the effects of active ingredients on skin health, written in English or Indonesian, and available in full text. The exclusion criteria were articles published before 2015, focused on other organ systems, and review articles.

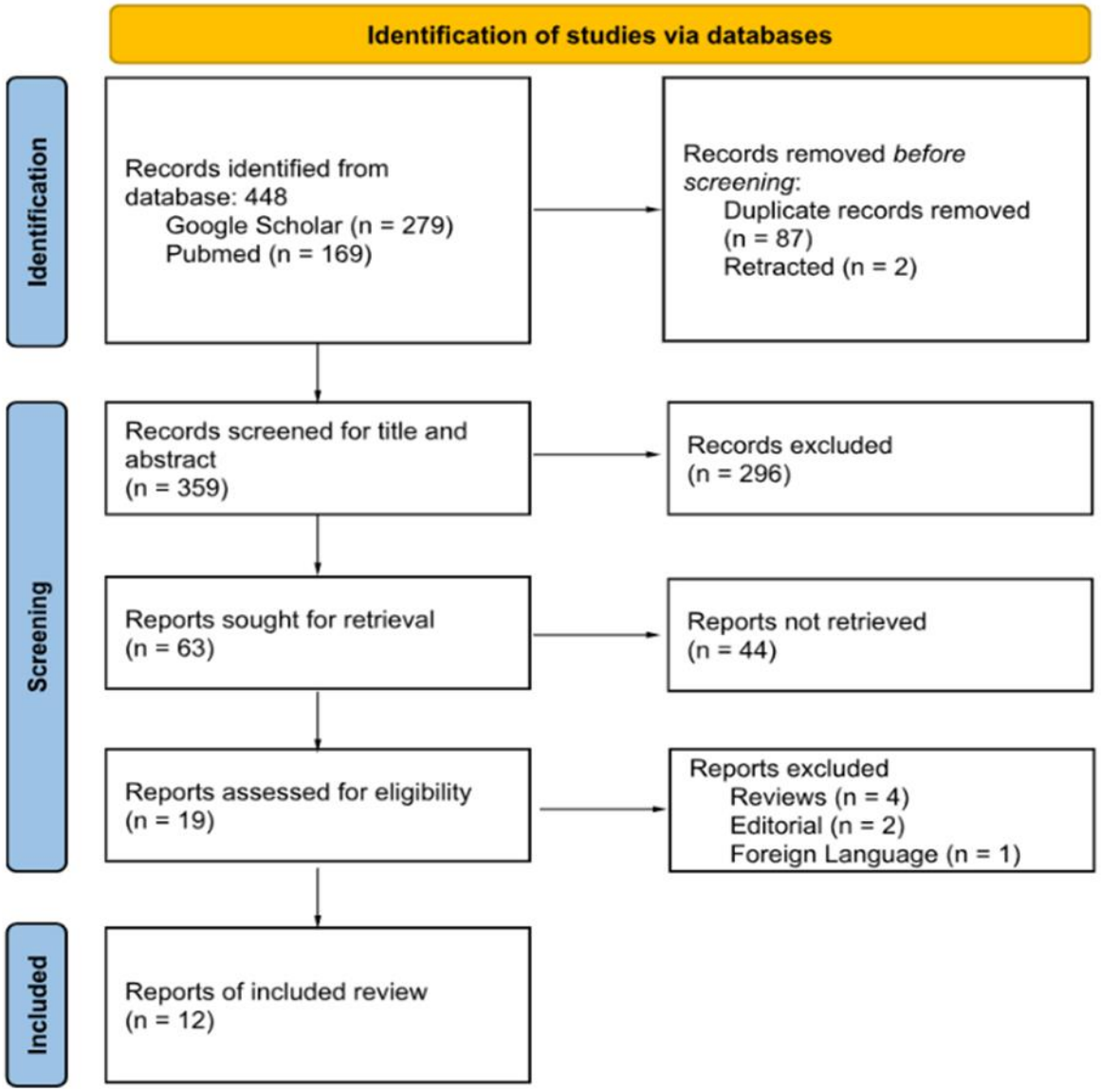

Figure 2. PRISMA Diagram

3. Result

Results A comprehensive understanding of internal physiological factors is essential for advancing skin health strategies beyond topical applications. Key intrinsic factors such as hydration mechanisms, hormonal regulation, and genetic predispositions significantly influence skin function, resilience, and aging. Rather than relying solely on topical moisturizers or synthetic formulations, recent studies have increasingly focused on intrinsic biological

mechanisms that regulate water retention in the stratum corneum, estrogen levels, and genetic markers modulate hydration, collagen synthesis, and oxidative defense. The following table outlines the current knowledge regarding major internal factors affecting skin condition, supported by comparative findings and recent literature, thereby providing a framework for integrative and personalized.

Table 1. Internal Factors Affecting Skin Conditions

Factors	Comparative Findings	References
Hydration	Skin hydration depends on molecular changes in keratin filaments and corneocyte swelling at RH ≥ 85%, the optimized hydration condition led to increased keratin interchain spacing (from ~9.5 Å to ~10.7 Å), enhanced molecular mobility in serine and glycine residues located in the protruding terminals of keratin filaments (as indicated by INEPT NMR signals), and a shift in secondary protein structure from α-helix to β-sheet.	(13)
	Hydration is related to the level of pigmentation but not to skin pH or other barrier functions such as TEWL or stratum corneum cohesion. This means that skin hydration is more influenced by pigmentation level than by surface pH or skin barrier strength. In healthy skin, moisture levels typically range from 40–65% (or approximately 30–60 arbitrary units on a corneometer), with values below 35–40% indicating dehydration and above 65–70% suggesting excess hydration, often from external factors.	(14)
Estrogen hormones	Estrogen is believed to influence skin aging by improving wrinkle appearance and skin rigidity; however, observational studies may be confounded. Estrogen's potential skin benefits include structural support and improved dermal quality through hormonal pathways.	(15)
	Estrogens improve skin by enhancing collagen, elastin, hydration, and wound healing while inhibiting MMPs and oxidative stress. Decline in estrogen postmenopausal impairs dermal health, with HT or SERMs offering protective and restorative benefits to facial skin.	(16)
Genetic	Genetic variants in genes like COL1A1, ESR1, VDR, and IL-6 influence aging traits such as bone density and skin structure. Estrogen receptor genes interact with environmental factors like calcium and estrogen, highlighting the pleiotropic and population-specific nature of aging-related gene expression.	(17)
	DNA-based skin analysis reveals individual genetic predispositions affecting antioxidant capacity, glycation, inflammation, acne risk, and pollution sensitivity. These findings support personalized skin care based on genetic traits and emphasize the genetic contribution to skin aging and resilience.	(18)

Table 2. External Factors Affecting Skin Conditions

Factors	Comparative Findings	References
Light Absorption	Super Anomalous Skin Effect (SASE) increases light absorption through surface plasmon excitation and longitudinal electric fields, which depend on the light's angle and polarization.	(19)
	Facial skin reflectance is influenced by sebum levels, with a negative correlation in the 400–600 nm range and a positive one in the infrared range. This suggests that sebum affects both light absorption and the visual appearance of skin tone, such as redness.	(20)
Air Pollutants	Particulate matter and ozone penetrate the skin barrier and generate reactive oxygen species (ROS), which oxidize lipids and degrade collagen, accelerating signs of aging like wrinkling, dullness, and pigmentation.	(21)
	PAHs from urban air activate AhR in keratinocytes and fibroblasts, increasing matrix metalloproteinases (MMPs), stimulating melanogenesis, and causing hyperpigmentation and textural deterioration. Prolonged exposure rapidly induces visible skin aging.	(22)
UV Radiation	Chronic UVA/UVB exposure overwhelms endogenous defenses, leading to leathery texture, erythema, edema, wrinkles, roughness, dryness, sagging, pigmentation irregularities and impaired wound healing. UVR-induced damage accelerates molecular aging pathways, compromising the skin's protective barrier and increasing cancer risk.	(23)
	Highly energetic UVB, which is largely absorbed in the epidermis and papillary dermis, and the more abundant but lower-energy UVA, particularly long-wave UVA1, which penetrates deeply into the dermal layer. These solar rays generate reactive oxygen species (ROS) within skin cells, deplete endogenous antioxidants, and activate key signaling pathways (ERK1/2, p38 MAPK, JNK, PI3K/AKT, STAT3), thereby promoting molecular changes associated with photoaging	(24)

4. Discussion

Discuss how internal factors such as hydration, genetics, and estrogen levels significantly influence facial skin condition and appearance. Proper hydration maintains skin smoothness and elasticity by strengthening the barrier function and reducing transepidermal water loss. This process involves the coordinated action of Natural Moisturizing Factors (NMF), intercellular lipid layers, and aquaporin-3 channels, which facilitate water and glycerol transport into the epidermis. Interestingly, hydration is more strongly influenced by skin pigmentation level than by surface pH or barrier integrity (13,14).

Skin hydration relies on the coordinated function of humectants, lipid barriers, and water channels, each playing a distinct yet interconnected role in maintaining moisture balance and skin barrier integrity. Humectants, such as glycerol, urea, and amino acids from the Natural Moisturizing Factor (NMF), actively attract and bind water molecules from the environment or deeper skin layers to the outermost layer of the epidermis, the stratum corneum. This helps keep corneocytes hydrated and supple. Meanwhile, lipid barriers composed mainly of ceramides, cholesterol, and free fatty acids form a structured lamellar matrix between corneocytes, which acts as a waterproof seal to prevent transepidermal water loss (TEWL). These lipids are critical for maintaining the skin's barrier function and structural cohesion. Lastly, water channels like aquaporin-3 facilitate the intracellular transport of water and glycerol from the dermis into the viable layers of the epidermis, ensuring continuous internal hydration. The efficiency of skin hydration depends on the proper functioning of all three components; disruption in any part whether due to environmental stress, aging, or pathology can lead to dryness, irritation, and impaired barrier function.

a. Hydration Studies

Mojumdar et al. (13) reported that hydration of the stratum corneum (SC) induced significant molecular and structural changes at a threshold of 85% relative humidity (RH). The SC is composed of flattened, dead skin cells known as corneocytes, which are embedded in a lipid matrix. Inside these corneocytes are densely packed keratin filaments—intermediate filaments with a rigid core and flexible terminal regions that play a key role in maintaining the skin's mechanical integrity. At RH ≥ 85%, the optimized hydration condition led to increased keratin interchain spacing (from ~9.5 Å to ~10.7 Å), enhanced molecular mobility in serine and glycine residues located in the protruding terminals of keratin filaments (as indicated by INEPT NMR signals), and a shift in secondary protein structure from α-helix to β-sheet (confirmed by FTIR and WAXD). These changes corresponded with a sharp rise in water uptake and improved SC flexibility. In contrast, under dry conditions (RH < 85%), the keratin structure remained compact and rigid, with limited water uptake, absence of molecular mobility, and predominant α-helical configuration, resulting in brittle mechanical behavior. Furthermore, treatment with 20–30 wt% urea at 80% RH produced similar outcomes to high RH hydration, including expanded keratin spacing (~10.1 Å) and increased amino acid mobility, attributed to urea's ability to substitute water and retain molecular fluidity under reduced humidity. These findings were ascribed to the hydration-induced relaxation of keratin terminals, enabling long-range repulsive forces and greater corneocyte swelling, comparable to flexible polymer brush behavior.

While Voegeli et al. (14) stated that skin hydration correlates more strongly with pigmentation level than with surface pH, TEWL, or SC cohesion. In healthy skin, moisture levels typically range from 40–65% (or approximately 30–60 arbitrary units on a corneometer), with values below 35–40% indicating dehydration and above 65–70% suggesting excess hydration, often from external factors. When moisture is too low, skin may appear dull, rough, tight, flaky, and show fine lines, particularly around the eyes. Conversely, overly

hydrated skin—such as from overuse of occlusive products or exposure to humid conditions—can appear shiny, soft, or even macerated (pale and soggy), and may be prone to breakouts or microbial imbalance. While pigmentation plays a key role in hydration regulation, maintaining optimal moisture balance is still critical to preserve the skin's visual appearance

b. Hormones

In addition to hydration, estrogen, particularly 17β-estradiol, plays a critical role in skin regeneration and homeostasis. Lephart (16) reported that this reproductive hormone prevents the loss of collagen in postmenopausal women, thereby helping to maintain skin thickness, elasticity, and structural integrity. Estrogen acts through ERα, ERβ, and GPER1 receptors to stimulate fibroblasts and keratinocytes, promoting the synthesis of collagen, elastin, hyaluronic acid, and acid mucopolysaccharides—key components for maintaining moisture and reinforcing the stratum corneum barrier. This hormonal activity not only improves dermal density and hydration but also supports the skin's mechanical strength and resilience. Lephart also shows that topical and systemic estrogen therapies can significantly increase skin collagen content and epidermal thickness, particularly in estrogen-deficient conditions. Furthermore, estrogen deprivation has been associated with visible aging signs such as wrinkling, dryness, atrophy, laxity, impaired wound healing, hot flashes, and vulvar atrophy (16). Maintaining estrogen levels within an optimal physiological or therapeutic range (approximately 65–125 pg/ml) helps mitigate these symptoms and supports continuous cell regeneration. However, supraphysiological estrogen levels may pose systemic risks, which underscores the need for tightly regulated hormone therapy strategies to preserve dermal health without inducing adverse effects.

c. Genetics

Genetic factors exert a broad influence on skin physiology, but they act through distinct mechanisms depending on the biological pathway involved. Ortuño et al. (17) reported that genes such as COL1A1 play a structural role, directly influencing the physical architecture of the skin by encoding type I collagen, a key component of the dermal extracellular matrix. Variations in COL1A1 affect collagen density, fiber organization, and turnover, which directly impacts skin thickness, elasticity, and the formation of wrinkles. These collagen-related genes are unique in that they determine the mechanical strength and durability of skin tissue.

In contrast, other genetic variants such as those in IL-6, IL18, CAT, NQO1, AGER, and TYR function in regulatory or protective roles, modulating processes like inflammation, oxidative stress response, pigmentation, and immune sensitivity. These genes influence how the skin reacts to environmental stressors, rather than forming the skin's core structure. Akbar et al. (18) further demonstrated that genetic screening can identify predispositions to glycation, acne, or pollution sensitivity, which primarily affect surface-level skin appearance and barrier function, rather than dermal scaffolding.

This distinction highlights the unique importance of collagen-related genes like COL1A1 in age-associated structural decline, while regulatory genes contribute more to dynamic

responsiveness and short-term skin health maintenance. A comprehensive approach to personalized skincare and anti-aging strategies must therefore consider both structural and regulatory genetic profiles to effectively address the multifactorial nature of skin aging.

d. Light Absorption

Light interacts with human skin through absorption, reflection, and scattering—processes governed by both the structural composition and biochemical state of the skin. Chromophores such as melanin, hemoglobin, and carotenoids absorb specific wavelengths of light, thereby influencing skin tone, brightness, and redness. Additionally, the skin's surface characteristics—particularly refractive index variations and sebum content—modulate how light is reflected or diffused, affecting whether the skin appears matte or glossy. Recent studies provide deeper insight into these phenomena. Vagov et al. (19) highlighted the role of the Super Anomalous Skin Effect (SASE), a physical principle mainly observed in conductive materials but now applied to biological tissue. Their findings demonstrate that light absorption can be significantly enhanced through surface plasmon excitation and longitudinal electric fields, depending on the light's angle and polarization. This mechanism contributes to the understanding of skin brightness and translucency beyond pigmentation alone. Complementarily, Banyś et al. (20)emphasized the role of biochemical surface conditions by showing that higher sebum levels reduce light reflectance in the visible range (400–600 nm), leading to duller skin appearance, while increasing reflectance in the infrared spectrum, which may contribute to a shiny look. Together, these studies illustrate how both physical optics and skin surface composition interact to shape the visual perception of facial skin.

e. Air Pollutants

Air pollution has emerged as a critical extrinsic factor affecting skin appearance, especially in urban populations. Roberts (21) highlights that airborne pollutants such as particulate matter (PM2.5), nitrogen dioxide (NO_2), and polycyclic aromatic hydrocarbons (PAHs) can penetrate the skin barrier and disrupt homeostasis through oxidative stress. These pollutants increase reactive oxygen species (ROS) production, which in turn accelerates the expression of matrix metalloproteinases, damages the extracellular matrix, and results in visible skin aging—manifesting as hyperpigmentation, lentigines (age spots), melasma, and wrinkle.

Specifically, Roberts notes epidemiological evidence linking increased levels of airborne PM to a higher incidence of lentigines, especially on sun-exposed areas like the forehead and cheeks. One study found that residents living within 100 meters of a busy road had 35% more senile lentigines on the forehead. Additionally, melasma is reportedly more prevalent in heavily polluted regions among individuals with Fitzpatrick skin types III to VI, potentially due to synergistic effects of UV and pollution-induced activation of the aryl hydrocarbon receptor (AHR), which influences melanocyte activity.

Complementing these findings, the report by Bocheva et al. (22) airborne pollutants such as particulate matter (PM) and ozone can penetrate the skin barrier and induce oxidative stress by generating reactive oxygen species (ROS). These ROS oxidize lipids, impair mitochondrial

function, and degrade extracellular matrix components such as collagen and elastin, ultimately leading to visible signs of aging including wrinkles, pigmentation disorders, and loss of skin elasticity. PAHs, another class of environmental toxins present in urban air, activate the aryl hydrocarbon receptor (AhR) in keratinocytes and fibroblasts. This receptor activation stimulates the expression of matrix metalloproteinases (MMPs), enhances melanogenesis.

f. UV Radiation

Stoykova et al. (23) emphasize that ultraviolet radiation (UVR) represents the most significant environmental driver of premature skin aging, profoundly altering both appearance and function. At the molecular level, UV radiation induces excessive generation of reactive oxygen and nitrogen species (ROS/RNS), which activate signaling cascades such as MAPK/AP-1, PI3K/Akt, NF-κB, JAK/STAT and KEAP1/NRF2 in keratinocytes and fibroblasts. This results in overexpression of matrix metalloproteinases (MMP-1, -3, -9), breakdown of the extracellular matrix and reduced collagen synthesis, fostering chronic inflammation and dermal degradation. Simultaneously, UVB photons directly form cyclobutane pyrimidine dimers and 6–4 photoproducts in DNA, while UVA-induced ROS oxidize nucleobases (e.g., 8-oxoG), triggering p53-mediated cell-cycle arrest, nucleotide excision repair and, under severe damage, apoptosis. Furthermore, ROS and Ca^{2+} dysregulation compromise mitochondrial membrane integrity, activating caspase-3/9 and PARP-mediated parthanatos, as well as inflammasome (NLRP3) and cGAS-STING pathways, which amplify inflammation and cell death. Persistent UV exposure also drives telomere attrition and up-regulation of p16^INK4a/p21, inducing a senescence-associated secretory phenotype (SASP) rich in pro-inflammatory cytokines (IL-1β, IL-6, IL-8) and MMPs, while hyperactivation of PI3K/Akt/mTOR and suppression of AMPK/SIRT1 impair autophagy/mitophagy, further undermining proteostasis and cellular resilience

Another report by Grenier et al (24) also mentioned at the molecular level, UV-induced ROS activate matrix metalloproteinase-1 (MMP-1), initiating the proteolytic cleavage of collagen fibers and other extracellular matrix components. Concurrently, chronic UV exposure disrupts the transforming growth factor-β (TGF-β)/Smad signaling cascade by reducing Smad2 phosphorylation, which leads to a profound decrease in procollagen I synthesis. UV radiation also stimulates ERK1/2 and JNK pathways, resulting in formation of AP-1 transcription factor complexes that bind to MMP promoters and further elevate collagenase expression. The combined effect is an accelerated breakdown of collagen types III and IV and inhibition of new collagen production, manifesting clinically as wrinkles and loss of skin elasticity

g. Challenges and Limitations

While the interaction between internal and external factors in determining facial skin health is widely acknowledged, several limitations persist within the current understanding of this topic. First, the complexity of skin physiology and aging involves highly individualized responses influenced by genetics, environment, lifestyle, and hormonal status, making it difficult to isolate the effects of single variables. For

example, hydration, hormone levels, and gene expression are deeply interrelated, and the extent to which each factor independently contributes to visible skin outcomes remains incompletely understood (25). Additionally, most studies on topical active ingredients are conducted under controlled conditions that may not reflect real-world usage, including variability in product formulations, application consistency, and patient adherence (26). There is also a lack of long-term clinical data evaluating the sustained effectiveness and safety of many ingredients, especially when combined. In the case of internal factors like estrogen or genetic profiling, ethical concerns, population-specific variability, and access to personalized testing limit the feasibility of translating such insights into universally applicable skincare recommendations. Finally, although external agents like niacinamide and vitamin C show promise, their efficacy can be influenced by skin barrier status, environmental exposure, and interactions with other topical or systemic treatments (27), factors that are not always accounted for in current research.

H. Recommendations

To optimize facial skin health, both external skincare and internal habits play essential roles. Polyphenols such as those in rice water, green tea, and tocopherol are recommended for use in topical products like sunscreens and serums due to their strong antioxidant, anti-inflammatory, and skin-brightening effects(28). Niacinamide supports the skin barrier, reduces inflammation and pigmentation, and boosts collagen, making it ideal for daily moisturizers and anti-aging treatments (29). Vitamin C, with its collagen-stabilizing and antioxidant properties, is best applied in the morning for UV protection(30,31).

AHAs and BHAs are effective exfoliants for improving skin texture and acne but should be used cautiously with sun protection (32). Retinoids are best applied at night to stimulate collagen and repair signs of aging (33). Internally, a diet rich in antioxidants and polyphenols, along with adequate sleep and stress management, supports skin regeneration, hormonal balance, and barrier function. Together, these approaches offer a holistic strategy for healthier, youthful-looking skin.

5. Conclusion

Facial skin health is the result of a complex interplay between internal physiological mechanisms such as hydration, hormonal regulation, and genetic predispositions—and external environmental exposures including UV radiation, air pollution, and light absorption. Hydration plays a foundational role in maintaining skin barrier integrity, elasticity, and appearance, and is regulated by natural moisturizing factors, intercellular lipids, and aquaporin channels. Estrogen contributes to skin firmness and repair by stimulating collagen production and reducing oxidative stress, while genetic factors influence both structural components (e.g., collagen density) and regulatory pathways (e.g., inflammation, antioxidant defense). Externally, UV radiation and pollutants accelerate visible aging and compromise skin health through oxidative damage, DNA injury, and extracellular matrix degradation. Light interaction with the skin further modifies its visual quality, influenced by both physical and biochemical surface properties. While numerous interventions focus on topical treatments, this review emphasizes the necessity of addressing intrinsic factors and their interactions

efocus on topical treatments, this review emphasizes the necessity of addressing intrinsic factors and their interactions with environmental stressors. A holistic and personalized approach incorporating skincare products with proven active ingredients, hormone regulation where appropriate, genetic understanding, and lifestyle factors such as nutrition and sun protection is essential to achieving and maintaining healthy, resilient skin. Future research and clinical strategies should further explore the synergistic effects of these factors and develop tailored interventions that account for individual variability in skin biology and exposure history.

6. References

1. H. Yousef, M. Alhajj, A.O. Fakoya, S. Sharma, Anatomy, Skin (Integument), Epidermis, 2025.
2. G. McKnight, J. Shah, R. Hargest, Surgery (Oxford) 40 (2022) 8–12.
3. Z. Lotfollahi, Wound Practice and Research 32 (2024).
4. S. Humphrey, S. Manson Brown, S.J. Cross, R. Mehta, Dermatologic Surgery 47 (2021) 974–981.
5. I.A. Ahmed, M.A. Mikail, Nutrition 119 (2024) 112350.
6. P. Ravisankar, O.S. Koushik, V. Himaja, J. Ramesh, P. Pragna, J Pharm Res 5 (2015) 209–301.
7. Y. Tokura, M. Yunoki, S. Kondo, M. Otsuka, J Dermatol 52 (2025) 192–203.
8. J. Chen, S. Li, C. Li, Med Res Rev 41 (2021) 1138–1166.
9. Y. Nobeyama, J Dermatol 51 (2024) 1143–1156.
10. R.S. Hussein, S. Bin Dayel, O. Abahussein, A.A. El-Sherbiny, J Cosmet Dermatol 24 (2025).
11. L. Rodrigues, L. Palma, L. Tavares Marques, J. Bujan Varela, Clin Cosmet Investig Dermatol (2015) 413.
12. B. Woodby, K. Penta, A. Pecorelli, M.A. Lila, G. Valacchi, Annu Rev Food Sci Technol 11 (2020) 235–254.
13. E.H. Mojumdar, Q.D. Pham, D. Topgaard, E. Sparr, Sci Rep 7 (2017) 15712.
14. R. Voegeli, A. V. Rawlings, B. Summers, Int J Cosmet Sci 37 (2015) 241–252.
15. C.M. Owen, L. Pal, S.L. Mumford, R. Freeman, B. Isaac, L. McDonald, N. Santoro, H.S. Taylor, E.F. Wolff, Fertil Steril 106 (2016) 1170-1175.e3.
16. E.D. Lephart, J Cosmet Dermatol 17 (2018) 282–288.
17. R. Romero-Ortuno, R.A. Kenny, R. McManus, Exp Gerontol 129 (2020) 110781.
18. S.A. Akbar, S.M.J. Hassan, Z.M. Raoof, M.M.M. Saeed, UHD Journal of Science and Technology 8 (2024) 151–163.
19. A. Vagov, I.A. Larkin, M.D. Croitoru, V.M. Axt, Sci Rep 13 (2023) 5103.
20. A. Banyś, M. Hartman-Petrycka, K. Kras, M. Kamińska, B. Krusiec-Świdergoł, P. Popielski, A. Lebiedowska, S. Wilczyński, Applied Sciences 13 (2023) 2838.
21. W. Roberts, Int J Womens Dermatol 7 (2021) 91–97.
22. G. Bocheva, R.M. Slominski, A.T. Slominski, Int J Mol Sci 24 (2023).
23. I.D. Stoykova, I.K. Koycheva, B.K. Binev, L. V. Mihaylova, M.I. Georgiev, Phytochemistry Reviews 24 (2025) 119–150.
24. A. Grenier, M.C. Morissette, P.J. Rochette, R. Pouliot, Sci Rep 13 (2023) 17969.
25. H. Knaggs, E.D. Lephart, Cosmetics 10 (2023) 142.

26. R.S. Hussein, S. Bin Dayel, O. Abahussein, A.A. El-Sherbiny, J Cosmet Dermatol 24 (2025).
27. J. Pullar, A. Carr, M. Vissers, Nutrients 9 (2017) 866.
28. M. Farhan, Molecules 29 (2024) 865.
29. C. Marques, F. Hadjab, A. Porcello, K. Lourenço, C. Scaletta, P. Abdel-Sayed, N. Hirt-Burri, L.A. Applegate, A. Laurent, Antioxidants 13 (2024) 425.
30. F. Al-Niaimi, N.Y.Z. Chiang, J Clin Aesthet Dermatol 10 (2017) 14–17.
31. P. Telang, Indian Dermatol Online J 4 (2013) 143.
32. K. Karwal, I. Mukovozov, Cosmetics 10 (2023) 131.
33. M. Zasada, E. Budzisz, Advances in Dermatology and Allergology 36 (2019) 392–397.