

Review Article

When skincare backfires: The microbiome-stress-allergy axis in cosmetic hypersensitivity

Divina Mariya Puthooran¹, Ayesha Fatima Syeda², Sashri Rangika Kunnatha Cyrilge¹,
Darshita Manjunatha Nanjundappa³, Malaz Hamed Nasr Mohamed⁴, Tanmay Shivanand Masti³

¹Department of Medicine, International Faculty of Medicine, Tbilisi State Medical University, Tbilisi, Georgia, ²Department of Medicine, Shadan Institute of Medical Sciences, Hyderabad, Telangana, ³Department of Medicine, Georgian National University (SEU), ⁴Department of Medicine, Faculty of Medicine, University of Georgia, Tbilisi, Georgia.



***Corresponding author:**

Divina Mariya Puthooran,
Department Of Medicine,
Faculty Of Medicine, Tbilisi
State Medical University, Tbilisi,
Georgia.

divinamariyap@gmail.com

Received: 18 July 2025

Accepted: 04 September 2025

Published: 03 November 2025

DOI

10.25259/CSDM_126_2025

Quick Response Code:



ABSTRACT

Globally, the number of hypersensitivity reactions has increased with the usage of cosmetics, which mostly manifest as allergic or irritant contact dermatitis. This pathologic triad, now referred to as the microbiome-stress-allergy axis, is caused by the complex interactions of the skin microbiota, psychological stress, and immunological systems. Cosmetic contact allergy is prevalent globally, with lower positive patch test rates in European populations than those from the US, Asia, and the Middle East. Women are more susceptible due to exposure to allergens in cosmetics and household products. Incidence rates are higher in urban populations due to occupational and environmental factors. Certain occupations, such as healthcare professionals, hairdressers, and food handlers, are at particular risk of cosmetic-related hypersensitivity due to frequent exposure to soaps, disinfectants, hair dyes, and cosmetic chemicals, which compromise the skin barrier and increase allergen sensitization. This narrative review examines how the presence of allergens and preservatives in cosmetics can alter the skin barrier integrity and microbiome, making people more prone to inflammation and sensitization. In addition, it has been shown that psychological stress weakens skin immunity through cortisol and neuropeptide-based hormonal cascades, exacerbating skin disorders such as psoriasis, atopic dermatitis (AD), and acne. With an emphasis on mast cell function, T-cell imbalances, and compromised barriers, the review highlights recent research on immunological dysregulation caused by alteration of the microbiota and neuroendocrine variables. Clinical signs are discussed, such as urticaria, typical eczematous responses, and pigmentation diseases like Riehl melanosis. Populations at increased risk include women, healthcare professionals, those with an atopic background, and those who regularly use intricate skincare routines. Management strategies include using targeted patch testing to prevent allergies, probiotic or ceramide-based skincare products to rebuild the barrier, and psychodermatologic treatments to reduce stress. Adjunct pharmacologic therapy, including topical corticosteroids, calcineurin inhibitors, Janus kinase inhibitors, and phosphodiesterase-4 inhibitors, has shown efficacy in treating AD. Advances in microbiome profiling methods, such as metagenomic shotgun sequencing and amplicon-based sequencing, which are useful in profiling the skin microbiota, are spurring demand for personalized skincare. Artificial intelligence algorithms can predict component sensitivity to enhance tailored skincare by analyzing chemical compositions and comparing them with individual skin and allergy profiles. By integrating dermatological, immunological, and psychological knowledge, this review provides a thorough framework for understanding and managing cosmetic hypersensitivity in the modern era.

Keywords: Contact dermatitis, Cosmetic hypersensitivity, Cutaneous immune response, Skin barrier dysfunction, Skin microbiome dysbiosis, Skincare-induced dermatoses

INTRODUCTION

Natural plant-based skincare and perfumes that have been used since ancient times have now advanced to be commercial cosmetic products that are carefully formulated from both

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, transform, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

©2025 Published by Scientific Scholar on behalf of CosmoDerma

natural and synthetic chemicals, to maximize benefits for humankind. Cosmetic products, mainly classified into two categories, personal care products and make-up products, are used topically to serve three main purposes: to modify physical appearance, to protect from physiological aging and environmental pollutants, and to maintain skin health.^[1]

Paralleling the growth of the global cosmetic market is the surge of adverse skin events that are collectively known as cosmetic hypersensitivity. It refers to a range of skin conditions triggered by exposure to cosmetic ingredients. A pivotal point to be considered in the world of cosmetics is the sensitive skin syndrome (SSS), which is defined as the occurrence of uncomfortable skin sensations, with a varying presentation, including but not limited to redness, itching, burning, or stinging sensations, triggered by stimuli that typically provoke no sensation in the general healthy skin population and affects 71% of adult population worldwide.^[2,3] Owing to the high prevalence of SSS and increasing cosmetic use, studies show a rising prevalence of cosmetic hypersensitivity, frequently presenting as contact dermatitis (CD). CD is characterized by redness, itching, or blistering and includes two subtypes: Irritant CD (ICD), a non-immunologic irritant reaction, and allergic CD (ACD), an immune-mediated inflammatory response.^[4-7]

ICD is caused by direct damage to the epidermal layer through frequent use of soaps, detergents, solvents, and oils, which disrupts the normal barrier function.^[8] Coupled with immune responses is another crucial aspect that leads to damaged skin barrier: Physiological stress through neuropeptides and cortisol, the primary stress hormone.^[9] In addition, cosmetics designed to maintain the skin flora homeostasis are also known to disrupt the microbiome.^[10] This review aims to explore the integrated axis – skin barrier dysfunction, microbial imbalance, psychological stress, and neuroimmune dysregulation that often co-exist in cosmetic hypersensitivity, while highlighting clinical patterns, diagnostic challenges, and emerging management strategies.

MATERIAL AND METHODS

A comprehensive literature review was conducted using PubMed, Scopus, and Google Scholar. English language articles published between 2018 and 2025 were included. Keywords included “cosmetic allergy,” “skin microbiome,” “psychodermatology,” and “contact dermatitis.” Articles were selected based on clinical relevance, originality, and focus on the microbiome-stress-allergy axis in dermatological hypersensitivity.

Epidemiology

A 2025 meta-analysis comprising >4,000 children across 17 studies demonstrated that cosmetic contact allergy is

prevalent in children globally, though positive patch test rates were comparatively lower in European populations than in those from the US, Asia, and the Middle East.^[11] Women are more susceptible to ACD, as they are more likely to be exposed to allergens in cosmetics and household products. Incidence rates are higher in urban populations than in rural ones, as a result of occupational and environmental factors. Cosmetic-related ACD accounts for a significant proportion of dermatology consultations, with fragrances, preservatives, and hair dye ingredients being among the most frequently implicated allergens. Studies report that women and professionals with repeated exposure to cosmetic agents, such as healthcare workers and hairdressers, form a substantial subset of these consultations, underscoring cosmetics as an important driver of contact allergy in clinical practice.^[12,13] Sensitization thresholds vary among individuals in the general population, and this suggests that while some people can withstand much higher doses of an allergen, others may develop contact allergies after being exposed to extremely low quantities.^[14] Epidemiological studies reported that 15% of individuals get allergic contact eczema annually, suggesting it has a high frequency and prevalence in populations of all ages. Teenagers had a point prevalence of 15.2% while adults showed 18.6%, which is significantly higher.^[15]

Skin microbiome and barrier function

The skin barrier is a complex system comprising physical, chemical, immune, and microbial barriers. The microbial barrier includes a diverse range of bacterial, fungal, and viral species that colonize the skin from birth and exhibit significant antimicrobial actions.^[16-19] Detergents can affect the skin's epithelial barrier, leading to allergic diseases. They impair tight junction molecules of keratinocytes, increasing water loss and altering skin pH. This can lead to microbial dysbiosis, defined as an imbalance in microbiota, reducing microbial diversity and allowing allergens to penetrate, triggering inflammation leading to allergic diseases. Common handwashing, alcohol-based sanitizers, and antimicrobial soaps also compromise the microbial barrier. Studies found that topical commensal microbiota can help restore the skin's microbial barrier.^[20,21] A study found that using acidic, 100% natural skincare formulations can significantly increase skin microbial diversity and improve barrier integrity, with the fastest improvement observed in the natural formulation group compared to synthetic product users, and this study reported no industry sponsorship.^[22] A study found that cosmetics can significantly alter the skin microbiota, leading to changes in skin hydration, reduced water loss, and increased bacterial diversity. Some positive effects include reducing acne risk, but the disruption of commensal skin microbiota, such as *Staphylococcus* and *Corynebacterium* raises concerns about unintended microbial balance

disruption. In addition, prolonged mask usage has increased facial dermatoses (FD) such as CD and acne mechanica, particularly among healthcare personnel. Importantly, these conditions often overlap with cosmetic hypersensitivity, as compromised barrier function and occlusion from mask use may enhance penetration of cosmetic allergens and irritants, thereby exacerbating reactions to skincare and personal care products.^[23,24]

Active compounds and preservatives in cosmetics can alter skin flora by either disrupting beneficial commensals or exhibiting antimicrobial activity against harmful microbiomes. Preservatives such as parabens reduce microbial diversity and are linked with increased *Staphylococcus aureus* colonization, while methylisothiazolinone exerts strong antimicrobial effects that disrupt both harmful and commensal bacteria. Similarly, alcohols and surfactants decrease hydration and damage barrier lipids, impairing tight junctions and altering bacterial diversity. In contrast, barrier-enhancing compounds like ceramides support microbial diversity and improve barrier integrity.^[21] These ingredient-specific effects are summarized in Table 1. This highlights the connection between skin barrier function, microbial diversity, and cosmetic formulations, indicating that product composition is crucial in preventing or aggravating hypersensitive skin reactions.^[21]

Psychological stress and skin immunity

Stress affects the skin differently, with short-term stress enhancing immune protection and long-term stress linked to skin inflammatory conditions. Adrenocorticotrophic hormone, corticotropin-releasing hormone, cortisol, and neuropeptides are among the hormones released in response to stress that impair skin barrier integrity, inhibit immune cell activity, and reduce the synthesis of antimicrobial peptides. These mediators increase the risk of infections and inflammatory conditions such as acne, psoriasis, and atopic dermatitis (AD). The local hypothalamus-pituitary-adrenal-like system, in which skin cells express these hormones, enables them to react to stress by sending immunomodulatory

signals.^[25,26] As a result of adrenergic signaling, transforming growth factor β synthesis, fibroblast adipogenesis inhibition, and a decrease in cyclic adenosine monophosphate production, psychological stress increases skin susceptibility to *S. aureus* infection in murine studies.^[27] In a study comparing stressed and unstressed individuals, stress was associated with skin microbiome imbalance, reduced microbial diversity, and increased acidophilic and anaerobic bacteria contributing to skin disturbances characterized by lower skin pH, erythema, and blemishes.^[28] Stress is a major aggravating factor for acne, particularly in adult women with hormonal imbalances, and high stress levels have been positively correlated with acne severity through cortisol and androgen-mediated sebum overproduction, cytokine-driven inflammation, and barrier impairment.^[29-31] These same mechanisms, barrier breakdown, chronic inflammation, and immune dysregulation, also increase skin vulnerability to cosmetic allergens and irritants, thereby linking stress not only to acne but also to cosmetic hypersensitivity. In addition, stress-driven lifestyle changes such as poor sleep, altered diet, and intensified skincare routines may further amplify hypersensitivity reactions.^[32] Studies found that both acute and chronic stress were strongly linked with worsening of eczema symptoms. Stressful life factors such as family issues, financial issues, work overload, and examinations trigger psychological issues in students, leading to skin and hair-related problems like acne and sensitivity to cosmetics. Eczema also causes psychological distress, so addressing this aspect is crucial. Psychological interventions such as meditation, mindfulness, music therapy, massage therapy, and cognitive behavioral therapy are recommended.^[33-35]

Allergic responses to cosmetic ingredients

The increasing prevalence of allergic reactions to cosmetics in the past two decades highlights a global dermatological concern, demanding investigation into the immunological mechanisms and specific haptens, which are low molecular weight monomers responsible for these dermatoses.^[36] ACD is a type IV delayed hypersensitivity response, with

Table 1: Effects of cosmetic compounds on the skin microbiome.

| Compound/class | Function in cosmetics | Reported effect on microbiome | References |
|-------------------------------------|-------------------------|--|------------|
| Parabens | Preservatives | Reduce microbial diversity; associated with increased <i>Staphylococcus aureus</i> colonization | 21 |
| Methylisothiazolinone | Preservative | Strong antimicrobial effect; disrupts both harmful and commensal bacteria | 21 |
| Alcohols (ethanol, isopropyl) | Solvents, sanitizers | Decrease skin hydration and reduce commensal <i>Corynebacterium</i> and <i>Staphylococcus</i> spp. | 21 |
| Surfactants | Cleansing agents | Damage barrier lipids, impair tight junctions, and alter bacterial diversity | 21 |
| Essential oils (tea tree, lavender) | Fragrance/antimicrobial | Exhibits broad-spectrum antimicrobial activity but may reduce beneficial commensals | 21 |
| Ceramides | Skin barrier enhancers | Supports microbial diversity and barrier integrity | 21 |

a sensitization phase where an exogenous allergen, such as parabens, formaldehyde, and methylisothiazolinone, often found in cosmetics, penetrates the skin, forming an antigen complex with skin proteins and eliciting cytokine release, including interleukin-1 (IL-1) and tumor necrosis factor- α , followed by uptake by antigen-presenting cells (APC).^[12,37,38] APCs activate naive T-cells that ultimately differentiate into memory T-cells, which in the elicitation phase cause an inflammatory response approximately 72 h after the allergen reexposure through cytokines such as interferon-gamma, IL-2, and IL-17, resulting in the immune cell influx, which brings about the visible symptoms of ACD.^[6] In contrast to ACD, ICD lacks the sensitization phase and thereby has an immediate onset of dermatitis ranging from minutes to a few hours after exposure and is mainly an outcome of amplified innate immunity, rather than adaptive immunity. ICD is prompted by direct damage to the epidermal layer through the use of substances (such as cinnamon and cayenne pepper essential oils in lip plumper products), which disrupt the normal skin barrier function, producing an inflammatory response regulated by keratinocytes, fibroblasts, and other epidermal cells.^[39] Patch test studies have shown that cosmetic allergens are strongly associated with location-specific clinical manifestations. For instance, fragrance allergens such as linalool and limonene are linked to dermatitis (erythema, vesicles, and pruritus)

on the face, neck, and flexural areas, while hair dyes (p-phenylenediamine [PPD]) predominantly affect the scalp, hairline, and eyelids. Nail resins often cause periungual eczema but can also spread to the eyelids through transfer. Likewise, benzophenones in sunscreens are associated with photoallergic dermatitis on photo-exposed areas, whereas nickel and cobalt from cosmetic tools or applicators commonly cause periocular or periorbital dermatitis.^[40] A summary of common cosmetic allergens, their predominant clinical sites, and presentation patterns based on patch testing is provided in Table 2.

Prominent pruritus and vesicles with ill-defined borders are the main symptoms in ACD, whereas burning and stinging sensations in a well-demarcated area are the chief complaint in ICD. Both conditions share overlapping features, including erythema, edema, and dryness.^[12] Table 3 summarizes the key clinical features and onset of ACD, ICD, and contact urticaria. Urticaria, a wheal and flare reaction – raised erythematous, itchy skin, usually observed immediately after exposure to chemicals such as thiomersal in eye make-up products, PPD in hair care products.^[41] Unlike ACD and ICD, contact urticaria can occur without previous sensitization or through an immunological type 1 hypersensitivity reaction leading to the degranulation of histamines from immunoglobulin E-mediated mast cells.^[42]

Table 2: Cosmetic allergens and location-based clinical manifestations from patch test studies.

| Cosmetic allergen | Common exposure source | Predominant clinical sites affected | Clinical presentation (ACD) | References |
|--|--|---|--|------------|
| Fragrances (e.g., linalool, limonene) | Perfumes, creams, lotions | Face, neck, flexural areas | Erythema, vesicles, pruritus | 40 |
| Preservatives (parabens, MI/MCI) | Lotions, shampoos, moist wipes | Hands, face | Scaling, eczematous lesions | 40 |
| Hair dye (p-phenylenediamine, PPD) | Hair colorants | Scalp, hairline, eyelids | Pruritic, erythematous dermatitis | 40 |
| Nail resins (toluene-sulfonamide formaldehyde resin) | Nail polish, adhesives | Periungual skin, eyelids (via transfer) | Chronic periungual eczema, eyelid dermatitis | 40 |
| Sunscreen agents (benzophenones) | Sunscreens, lip balms | Face, lips, photo-exposed sites | Photoallergic dermatitis | 40 |
| Metals (nickel, cobalt in applicators/ jewelry) | Cosmetic applicators, eyelash curlers, jewelry | Eyelids, ears, periorbital skin | Erythema, papules, pruritus | 40 |

ACD: Allergic contact dermatitis, MI: Methylisothiazolinone, MCI: Methylchloroisothiazolinone

Table 3: Clinical presentations of cosmetic hypersensitivity.

| Condition | Onset | Clinical features | Common triggers | References |
|-----------------------------|---------------------------------------|---------------------------------------|---|------------|
| Allergic contact dermatitis | Delayed (24–72 h) | Erythema, pruritus, vesicles, scaling | Fragrances, preservatives, dyes | 3,19,28,40 |
| Irritant contact dermatitis | Immediate to early (minutes to hours) | Burning, stinging, erythema, dryness | Surfactants, alcohols, acids | 16,26,29 |
| Contact urticaria | Immediate (within minutes) | Wheals, itching, swelling, erythema | Preservatives (e.g., parabens), hair dyes | 22,36,42 |

The allergens in cosmetics are generally classified based on their functions and are as follows:^[40]

- Fragrances
- Preservatives
- Hair dyes
- Sunscreens
- Conditioning agents
- Surfactants
- Excipients
- Nail product resins.

Fragrance allergens such as linalool and benzyl alcohol, in cosmetics for sensitive skin, have been found to be the leading cause of allergic reactions.^[3] Despite their growing use in cosmetic formulations, natural ingredients such as fruit extracts, like mango, have strong sensitizing effects.^[43] Methylisothiazolinone, found in skin care, hair care, and baby products, is the foremost preservative allergen in the non-formaldehyde-releasing preservative category, followed by parabens, which are identified in a multitude of products.^[40] Retrospective studies conducted through standardized patch tests reveal toluene-sulfonamide formaldehyde resin and PPD as one of the more common allergens, especially among women.^[44]

Microbiome-stress-allergy axis: Synergy and interplay

This triad, which is depicted in Figure 1, is relevant, especially in cosmetic hypersensitivity and AD, where dysregulated immune responses can worsen symptoms.^[45]

Stress-induced microbiome changes

Dysbiosis, defined as alterations in the microbiome induced by psychological stress, begins in the oral cavity before progressing to the gut and skin. Stress may elevate levels of hormones like cortisol and glucose, and these levels depend

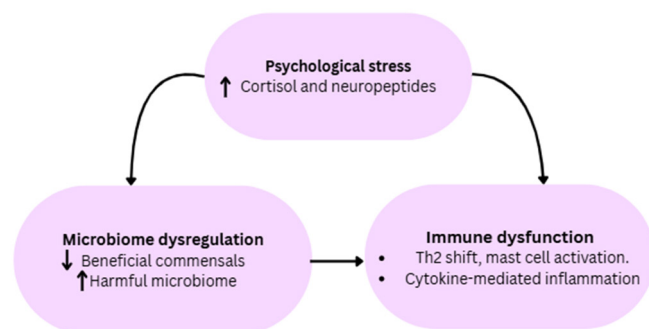


Figure 1: The microbiome-stress-allergy axis: Psychological stress (causes an increase in cortisol and neuropeptides) contributes to microbiome dysregulation marked by a decrease in beneficial commensals and an increase in harmful microbes, which in turn leads to immune dysfunction which includes T-helper cell 2 polarization, mast cell activation, and cytokine-mediated inflammation.

on the diversity and forms of bacteria that are present. Higher diversity is associated with longer cortisol clearance and a slower, healthier glycemic response. Conversely, less diversity results in impaired stress regulation and metabolic dysfunction.^[46,47] Stress also affects the microbiota in the skin and the gut. Immune cells in the blood, especially innate immune cells such as monocytes, neutrophils, T-helper cells (Th1 and Th17), and macrophages, are activated, producing immune responses, and disruption of these microbiomes results in increased gut permeability, which allows bacterial components to leak into the bloodstream. Furthermore, stress hormones, which lead to dysbiosis and interfere with the organ's natural barriers, may alter microbial diversity. It increases the population of pathogenic microbes while decreasing that of beneficial bacteria.^[48-50]

Immune dysregulation and allergic responses

Immune homeostasis is disturbed by dysbiosis, which is seen in allergic diseases such as asthma and AD. This dysregulation affects the gut and skin barriers, which are composed of stratified squamous epithelium and epithelial cells. Allergy diseases arise as a result of this damage, which causes inflammation and barrier breakdown.^[48,49,51] Hormones such as catecholamines and cortisol are released by the hypothalamic-pituitary-adrenal (HPA) axis. This axis can be activated by psychological stress, which causes the immune system to change to a Th2-dominant profile which is especially evident in allergic reactions.^[51-54]

Clinical manifestations and trends

Cosmetic hypersensitivity can often manifest as ACD.^[55] ACD can be acute and become chronic, especially if the allergen is not identified. It presents with erythema, blisters, pustules, crusts, scales, erosions, hemorrhage, and pain. Skin lesions in the areas of contact are characterized by asymmetric yet sharp-edged with symmetrical spread, developing in 24–48 h, compared to ICD, which has a rapid onset with no spreading.^[56,57] In addition, eczematous lesions with poorly defined erythema that affect the face, torso, and occasionally even on the exterior surface of the limbs are also characteristics of ACD.^[58] Whereas Riehl melanosis, another kind of ACD, manifests as either only pigmentation or pigmentation following erythema, edema, and pruritus, mostly presenting on the face, forehead, or zygomatic regions.^[59] Apart from this, ACD can also present itself as urticaria that mostly lasts as long as 24 h or less.^[60] Based on a retrospective study, terpene hydroperoxides like limonene and linalool hydroperoxides found in fragrances are major contributors to fragrance contact allergy, with 30.6% of patients developing cosmetic-induced FD as a result.^[56,61] Table 4 outlines agent-wise allergy patterns of cosmetic ingredients.

Table 4: Agent-wise allergy patterns in cosmetic hypersensitivity.

| Agent/ingredient | Function | Type of reaction | Common products | Mechanism | Reference |
|--|-----------------------------|-------------------|-------------------------------------|---|-----------|
| Parabens | Preservative | ACD | Lotions, creams, moisturizers | Type IV delayed hypersensitivity reaction. | 40,42 |
| Cayenne pepper, Cinnamon oil | | ICD | Lip plumper | Skin barrier disruption producing inflammation | 39 |
| Methylisothiazolinone | Preservative | ACD | Skin care, hair care, baby products | Cytokine-mediated T-cell activation | 40,43 |
| Formaldehyde | Preservative | ACD, ICD | Nail polish, shampoos | Barrier damage and activation of APCs. | 40,44 |
| Linalool | Fragrance (natural terpene) | ACD | Essential oils, perfumes | Sensitization and differentiation of naive T- cells into memory T-cells | 42,56,61 |
| Thiomersal | Preservative | Contact Urticaria | Eye makeup | Type I (IgE-mediated mast cell degranulation) reaction | 41,42 |
| PPD (p-phenylenediamine) | Hair dyes | Urticaria, ACD | Hair care products | Type I and Type IV hypersensitivity reactions | 41,44 |
| R-TSF (Toluene-sulfonamide formaldehyde resin) | Nail resin | ACD | Nail polish | Type IV delayed hypersensitivity reaction. | 44 |
| Mango extract | Natural ingredient | ACD | Fruit-based cosmetics | Type IV delayed hypersensitivity reaction. | 33 |

ACD: Allergic contact dermatitis, ICD: Irritant contact dermatitis, APC: Antigen-presenting cells

A community-based cross-sectional study from India involving 1,000 participants reported that 12.5% experienced cosmetic-related adverse events, most commonly acneiform eruptions (4.3%), CD (3.5%), and pigmentation changes (2.1%). Less frequent but notable reactions included conjunctivitis, photo-allergic/toxic responses, itching, and scalp damage. The authors emphasized that adverse effects often manifested after repeated product use and highlighted the importance of consumer awareness and dermatological consultation in early diagnosis and prevention.^[62]

Diagnosing ACD might be challenging due to its diverse clinical presentations. Therefore, prompt diagnosis and updated treatment are crucial to preventing adverse effects on a patient's quality of life (QoL).^[60]

Risk factors

An experimental study using patch testing in 77 patients with facial dermatitis demonstrated that 27.4% had cosmetic-related facial ACD, with a female predominance of 9.1% and a mean age of 37.3 ± 14.8 years. The study identified positive patch test reactions to common cosmetic allergens, particularly fragrances and preservatives. Overall, 45.5% of patients were diagnosed with ACD, with key risk factors including female sex, prior history of cosmetic allergy, and occupational exposure such as the use of hairdressing products.^[63] According to studies, repeated exposure to allergens may also raise the risk of developing ACD in those with AD.^[64]

Prevention and management strategies

Cosmetic allergen avoidance

Avoiding exposure to allergens is crucial in prevention. Among the most common allergens known is *Myroxylon pereirae*, a naturally derived material used in fragrances.^[65] Multiple patch tests are required for allergen identification due to the wide variety of chemicals used in products. A low threshold concentration is proof of sensitization and a strong indication of allergic response to that particular substance.^[65,66] After identifying the irritant, the patient must be counseled to avoid that substance and educated on the possibility of cross-reactivity. An adjunct pharmacotherapeutic treatment should be prescribed, including a list of substances safe for the patient to increase compliance.^[65]

Barrier repair and restoration of skin microbiome

Probiotics can modulate local immune response and compete against pathogens, offering protection by skin barrier enhancement, inflammation reduction, and sebum regulation.^[67] Nigerian and Canadian researchers developed a formulation containing *Lactiplantibacillus pentosus*, aiming to reduce odor-generating bacteria. Reduction in *Corynebacterium*, Actinobacteria, and Firmicutes and an increase in *Lactobacillus* were seen in 25 participants in the clinical trial, highlighting the efficacy of topical probiotics in the modulation of skin microbiome and QoL

improvement.^[67] In a study, LactoSporin (*Bacillus coagulans*) was found to reduce skin barrier markers, inflammation, and oxidative stress, thereby protecting against ultraviolet- and ozone-induced skin damage.^[67] Salt particles or ground almond shells are used to exfoliate and remove the stratum corneum, while carboxyl groups in soaps damage the skin barrier, causing protein damage, enzyme denaturation, and altered corneocyte water-holding.^[68,69]

Stress management in dermatology

Studies found that psychological stress is linked to chronic inflammation in certain brain regions. Nuclear factor erythroid 2-related factor 2 (NRF2) is a transcription factor that plays a crucial role in modulating inflammation by downregulating nuclear factor-B pathways and inhibiting IL-6 and IL-1 expression. Knockout of NRF2 in mice reduces dopamine and serotonin levels. High optimism is linked to low inflammation, IL-6, and endothelial dysfunction markers. Stress-induced HPA axis activation leads to higher cortisol levels and increased 11β-hydroxysteroid dehydrogenase (11β-HSD) expression, inhibiting keratinocyte differentiation, causing deterioration.^[70,71] Reduction in cortisol and 11β-HSD1 expression was noted with selective serotonin reuptake inhibitor (SSRI) therapy. In a study on patients with

depression, a 6-week treatment with the SSRI escitalopram led to a measurable improvement in skin barrier function. Specifically, reductions were observed in transepidermal water loss (TEWL) and stratum corneum integrity (i.e., improved delta TEWL), alongside increased hydration of the stratum corneum.^[71] In addition, a decrease in 11β-HSD1 expression – a key enzyme driving local cortisol activation was reported, suggesting normalization of the HPA axis and local glucocorticoid levels contributed to barrier recovery. These findings suggest that modifying underlying psychological stresses can be beneficial in skin barrier restoration.^[71]

Adjunct pharmacologic therapy

Topically prescribed agents such as corticosteroids (TCS), calcineurin inhibitors, Janus kinase inhibitors, phosphodiesterase-4 inhibitors, and moisturizers are used in AD management. It was found that TCS group 1 was most efficacious in improving AD severity, TCS groups 2–5, and high-dose 0.1% topical tacrolimus, which were most efficient in improving itch severity, and delgocitinib was best suited for improving QoL in eczema. Least effective were topical antibiotics alone or combined with other topical treatments. Long-term maintenance was better seen with TCS group 5.^[72] Figure 2 summarizes the main strategies discussed.

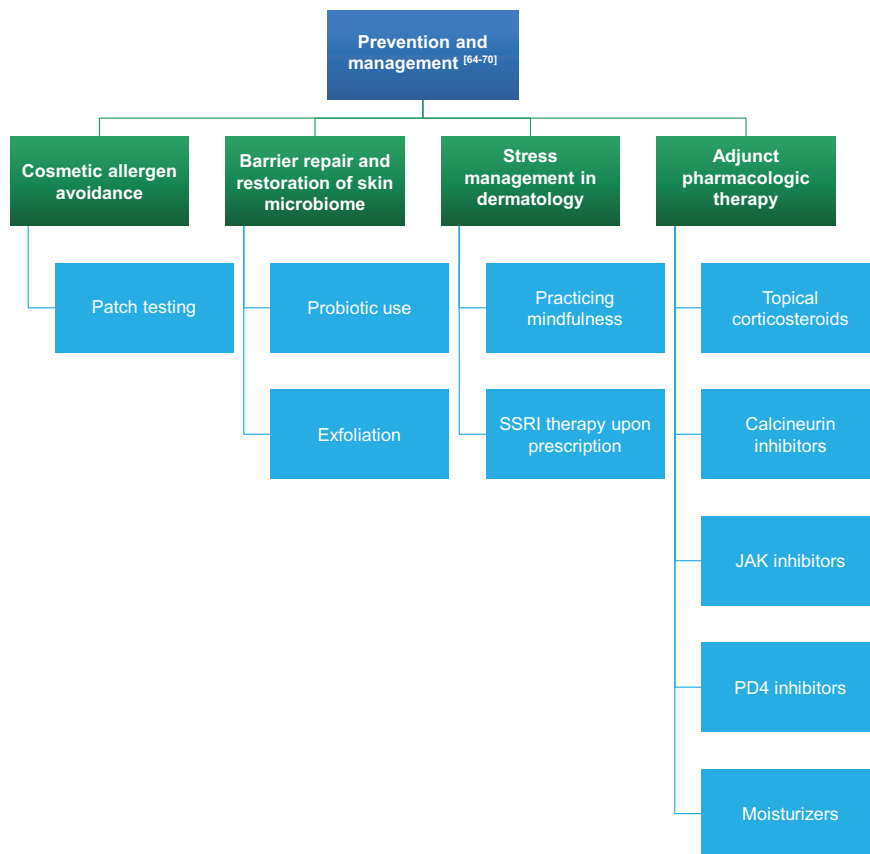


Figure 2: Summary of prevention and management strategies for cosmetic hypersensitivity.

Future directions

Advances in microbiome profiling methods, such as metagenomic shotgun sequencing and amplicon-based sequencing, are spurring demand for personalized skincare. The epidermal permeability barrier can be disrupted by stress, leading to dermatological conditions such as AD. Collaboration with mental health specialists may mitigate the detrimental effects of these conditions.^[73,74] Future diagnostic approaches for cosmetic hypersensitivity should explore reactive and personalized tools, potentially including mobile-based patch testing, wearable irritant sensors, or predictive algorithms to enable early detection and individualized risk assessment, aligning with broader trends in healthcare innovation.^[75] AI algorithms can predict ingredient sensitivity by analyzing chemical compositions for retinoids, parabens, and sulfates. Further cross-referencing with skin profiles, including allergy history, would enhance skincare personalization. Multiplex target detection using biosensors can detect and analyze microbiomes. AI-based applications like dermacompass can detect conditions like hand eczema and grade them based on severity. An interdisciplinary framework is needed to understand multifactorial causations and advance holistic diagnostic strategies.^[76-79]

CONCLUSION

Cosmetic hypersensitivity is a challenging condition mainly triggered by immunological dysregulation, microbial imbalance, and stress. Understanding the microbiome-stress-allergy axis provides a clinical basis for personalized interventions. An awareness of the microbiome-stress-allergy axis offers a therapeutic foundation for personalized interventions. Future dermatological care must include psychological support, barrier restoration, allergen avoidance, and AI-driven individualization to prevent better and treat skin conditions caused by cosmetics.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: Patient's consent is not required as there are no patients in this study.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that they have used artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript or image creation.

REFERENCES

- Liu JK. Natural products in cosmetics. *Nat Prod Bioprospect* 2022;12:40.
- Hassan I, Krishan K. Sensitive skin and its management. *Cosmoderma* 2021;1:53.
- Martins MS, Ferreira MS, Almeida IF, Sousa E. Occurrence of allergens in cosmetics for sensitive skin. *Cosmetics* 2022;9:32.
- Farage MA. The prevalence of sensitive skin. *Front Med (Lausanne)* 2019;6:98.
- Kumari S, Atem TD, Chaudhary V, Sahu SK, Pal B. Prevalence and risk factors of cosmetic-induced adverse events: A systematic review and meta-analysis. *J Appl Pharm Sci* 2024;14:1-9.
- Brar KK. A review of contact dermatitis. *Ann Allergy Asthma Immunol* 2021;126:32-9.
- Kuric I, Hadzavdic SL. Clinical presentation, diagnosis, differential diagnosis and management of contact allergy. *CosmoDerma* 2022;2:26.
- Patel K, Nixon R. Irritant contact dermatitis-a review. *Curr Dermatol Rep* 2022;11:41-51.
- Lee SH, Son ED, Choi EJ, Park WS, Kim HJ. Stress hormone cortisol damages the skin barrier by regulating tight junctions. *J Soc Cosmet Sci Korea* 2020;46:73-80.
- Carvalho MJ, Oliveira AL, Santos Pedrosa S, Pinto-Ribeiro I, Madureira AR. Skin microbiota and the cosmetic industry. *Microb Ecol* 2023;86:86-96.
- Isufi D, Jensen MB, Kursawe Larsen C, Alinaghi F, Baselius Schwensen JF, Johansen JD. Allergens responsible for contact allergy in children from 2010 to 2024: A systematic review and meta-analysis. *Contact Dermatitis* 2025;92:327-43.
- Tramontana M, Hansel K, Bianchi L, Sensini C, Malatesta N, Stingeni L. Advancing the understanding of allergic contact dermatitis: From pathophysiology to novel therapeutic approaches. *Front Med (Lausanne)* 2023;10:1184289.
- Boonchai W, Likittanasombat S, Viriyaskultorn N, Kanokrunsee S. Gender differences in allergic contact dermatitis to common allergens. *Contact Dermatitis* 2024;90:458-65.
- Basketter D, Safford B. Skin sensitization quantitative risk assessment: A review of underlying assumptions. *Regul Toxicol Pharmacol* 2016;74:105-16.
- Kostner L, Anzengruber F, Guillod C, Recher M, Schmid-Grendelmeier P, Navarini AA. Allergic contact dermatitis. *Immunol Allergy Clin North Am* 2017;37:141-52.
- Chen Y, Fischbach M, Belkaid Y. Skin microbiota-host interactions. *Nature* 2018;553:427-36.
- Lunjani N, Hlela C, O'Mahony L. Microbiome and skin biology. *Curr Opin Allergy Clin Immunol* 2019;19:328-33.
- Byrd A, Belkaid Y, Segre J. The human skin microbiome. *Nat Rev Microbiol* 2018;16:143-55.
- Eyerich S, Eyerich K, Traidl-Hoffmann C, Biedermann T. Cutaneous barriers and skin immunity: Differentiating a connected network. *Trends Immunol* 2018;39:315-27.
- Lu H, Zhou Y, Yang L, Zhou Q, Wang X, Qiu S, *et al.* Involvement and repair of epithelial barrier dysfunction in allergic diseases. *Front Immunol* 2024;15:1348272.
- Mim MF, Sikder MH, Chowdhury MZ, Bhuiyan AU, Zinan N, Islam SM. The dynamic relationship between skin microbiomes and personal care products: A comprehensive review. *Heliyon* 2024;10:e34549.
- Wallen-Russell C. The role of every-day cosmetics in altering the skin Microbiome: A study using biodiversity. *Cosmetics* 2019;6:2.

23. Lee HJ, Jeong SE, Lee S, Kim S, Han H, Jeon CO. Effects of cosmetics on the skin microbiome of facial cheeks with different hydration levels. *Microbiologyopen* 2017;7:e00557.
24. Perera MH, Joshi M, Govindan AK, Edpuganti S, Korrapati NH, Kiladze N. Impact of mask wear on the skin of clinical year medical students during the COVID-19 pandemic: A cross-sectional study. *CosmoDerma* 2022;2:96.
25. Zhang H, Wang M, Zhao X, Wang Y, Chen X, Su J. Role of stress in skin diseases: A neuroendocrine-immune interaction view. *Brain Behav Immun* 2023;116:286-302.
26. Pondeljnak N, Lugović-Mihić L. Stress-induced interaction of skin immune cells, hormones, and neurotransmitters. *Clin Ther* 2020;42:757-70.
27. Chan H, Li F, Dokoshi T, Cavagnero KJ, Li Q, Chen Y, *et al.* Psychological stress increases skin infection through the action of TGFβ to suppress immune-acting fibroblasts. *Sci Immunol* 2025;10:eads0519.
28. Morvan PY, Vallee R. Evaluation of the effects of stressful life on human skin microbiota. *Appl Microbiol Open Access* 2018;4.
29. Sutrisno AR, Jusuf NK, Putra IB. Correlation between stress scale and severity of acne vulgaris. *Bali Med J* 2020;9:376-9.
30. Bhaijamaal RA, Nayak SP. The impact of stress on acne. *Asian J Pharm Res Dev* 2023;11:143-50.
31. Sardana K, Bansal P, Vats G, Sharma L, Garga U, Khurana A. A prospective study examining trigger factors and hormonal abnormalities in adult female acne. *Indian Dermatol Online J* 2020;11:544.
32. Siddiqui I, Rais U, Tahir M. Exploring stress-induced mechanisms in acne pathogenesis. *Res Square* 2024.
33. Lönndahl L, Abdelhadi S, Holst M, Lonne-Rahm SB, Nordlind K, Johansson B. Psychological stress and atopic dermatitis: A focus group study. *Ann Dermatol* 2023;35:342-7.
34. Perera MH, Ahmed NK, Korrapati NH, Edpuganti S, Bhowmik P, Govindan AK, *et al.* Hair loss in medical students: A global approach. *Int J Prog Sci Technol* 2021;28:608-18.
35. Oska C, Nakamura M. Alternative psychotherapeutic approaches to the treatment of eczema. *Clin Cosmet Investig Dermatol* 2022;15:2721-35.
36. Tang X, Lin L, Yu F, Ma Y, Liu Z, Xu X. Allergic-related skin diseases: Global disease burden from 1990 to 2021 and future trends. *World Allergy Org J* 2025;18:101072.
37. Bialas I, Zelent-Kraciuk S, Jurowski K. The skin sensitisation of cosmetic ingredients: Review of actual regulatory status. *Toxics* 2023;11:392.
38. Scarberry KB, Mahlberg SJ, Nedorost S. Trends in positive patch tests for formaldehyde-containing allergens found in personal care products. *J Am Acad Dermatol* 2023;89:808-10.
39. Scheinman PL, Vocanson M, Thyssen JP, Johansen JD, Nixon RL, Dear K, *et al.* Contact dermatitis. *Nat Rev Dis Primers* 2021;7:38.
40. Fonacier L, Frankel D, Mawhirt S. Contact allergens for the allergist. *Ann Allergy Asthma Immunol* 2022;128:629-44.
41. Shaji M, Merin KA, Kameswaran R. A review on cosmetics causing contact urticaria. *Indian J Dermatol* 2022;67:392-8.
42. Verhulst L, Goossens A. Cosmetic components causing contact urticaria: A review and update. *Contact Dermatitis* 2016;75:333-44.
43. Edpuganti S, Gaikwad JR, Maliyil BT, Koshy RR, Potdar R, Latheef S, *et al.* The not-so-sweet side of mango: Mango allergy. *CosmoDerma* 2025;5:18.
44. Hafner MD, Rodrigues AC, Lazzarini R. Allergic contact dermatitis to cosmetics: Retrospective analysis of a population subjected to patch tests between 2004 and 2017. *An Bras Dermatol* 2020;95:696-701.
45. Bistoletti M, Bosi A, Banfi D, Giaroni C, Baj A. The microbiota-gut-brain axis: Focus on the fundamental communication pathways. *Prog Mol Biol Transl Sci* 2020;176:43-110.
46. Paudel D, Uehara O, Giri S, Yoshida K, Morikawa T, Kitagawa T, *et al.* Effect of psychological stress on the oral-gut microbiota and the potential oral-gut-brain axis. *Jpn Dent Sci Rev* 2022;58:365-75.
47. Charalambous EG, Mériaux SB, Guebels P, Muller CP, Leenen FA, Elwenspoek MM, *et al.* The oral microbiome is associated with HPA axis response to a psychosocial stressor. *Sci Rep* 2024;14:15841.
48. Sameer AS, Nissar S. Toll-like receptors (TLRs): Structure, functions, signaling, and role of their polymorphisms in colorectal cancer susceptibility. *Biomed Res Int* 2021;1157023.
49. De Pessemier B, Grine L, Debaere M, Maes A, Paetzold B, Callewaert C. Gut-skin axis: Current knowledge of the interrelationship between microbial dysbiosis and skin conditions. *Microorganisms* 2021;9:353.
50. Duran-Pinedo AE, Solbiati J, Frias-Lopez J. The effect of the stress hormone cortisol on the metatranscriptome of the oral microbiome. *NPJ Biofilms Microbiomes* 2018;4:25.
51. Kim HB, Alexander H, Um JY, Chung BY, Park CW, Flohr C, *et al.* Skin microbiome dynamics in atopic dermatitis: Understanding host-microbiome interactions. *Allergy Asthma Immunol Res* 2025;17:165.
52. Balakin E, Yurku K, Ivanov M, Izotov A, Nakhod V, Pustovoyt V. Regulation of stress-induced immunosuppression in the context of neuroendocrine, cytokine, and cellular processes. *Biology* 2025;14:76.
53. Xiang Y, Türk M, Ojeda IC, Elieh-Ali-Komi D, Salman A, Kocatürk E. Psychological stress and urticaria: Pathophysiologic and therapeutic updates. *Curr Treat Options Allergy* 2024;11:194-210.
54. Theoharides TC. The impact of psychological stress on mast cells. *Ann Allergy Asthma Immunol* 2020;125:388-92.
55. Bennike NH, Palangi L, Christensson JB, Nilsson U, Zachariae C, Johansen JD, *et al.* Allergic contact dermatitis caused by hydroperoxides of limonene and dose-response relationship—a repeated open application test (ROAT) study. *Contact Dermatitis* 2019;80:208-16.
56. Peng F, Mu Z, He C, Xue C, Li W, Wang Q, *et al.* Patch testing in facial dermatitis using Chinese baseline series (60 allergens) and cosmetic series (58 allergens). *J Eur Acad Dermatol Venereol* 2018;32:E288-9.
57. Novak-Bilić G, Vučić M, Japundžić I, Meštrović-Štefekov J, Stanić-Duktaj S, Lugović-Mihić L. Irritant and allergic contact dermatitis - skin lesion characteristics. *Acta Clin Croat* 2018;57:713-20.
58. Napolitano M, Fabbrocini G, Martora F, Genco L, Noto M, Patruno C. Children atopic dermatitis: Diagnosis, mimics, overlaps, and therapeutic implication. *Dermatol Ther* 2022;35:e15901.

59. Daadaa N, Sathe NC, Ben Tanfous A. Riehl melanosis. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2025. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557437> [Last accessed on 2024 Apr 08].
60. Yang TB, Kim BS. Pruritus in allergy and immunology. *J Allergy Clin Immunol* 2019;144:353-60.
61. Bruusgaard-Mouritsen MA, Garvey LH, Johansen JD. Facial contact dermatitis caused by cosmetic-relevant allergens. *Contact Dermatitis* 2021;85:650-9.
62. Kumari S, Pal B, Tewari D, Sahu SK. Cosmetics utilization and associated adverse events: A community based cross-sectional study. *Clin Epidemiol Glob Health* 2023;23:101382.
63. Kasemsarn P, Iamphonrat T, Boonchai W. Risk factors and common contact allergens in facial allergic contact dermatitis patients. *Int J Dermatol* 2016;55:417-24.
64. Lee J, Guo S, Dinalo J, DeLeo VA, Adler BL. Consort allergic contact dermatitis: A systematic review. *Dermatitis* 2022;33:181-6.
65. Fonacier L, Noor I. Contact dermatitis and patch testing for the allergist. *Ann Allergy Asthma Immunol* 2018;120:592-98.
66. Aalto-Korte K, Suuronen K, Frosch PJ. Patch testing with the patients' own products. In: *Contact Dermatitis*. New York: Springer; 2019. p. 1-19.
67. Alves AC, Da Silva Braga Martins SM, Belo JV, Lemos MV, De Matos Chaves Lima CE, Da Silva CD, *et al.* Global trends and scientific impact of topical probiotics in dermatological treatment and skincare. *Microorganisms* 2024;12:2010.
68. Wernham A, Cain O, Thomas A. Effect of an exfoliating skincare regimen on the numbers of epithelial squames on the skin of operating theatre staff, studied by surface microscopy. *J Hosp Infect* 2018;100:190-4.
69. Draelos ZD. The science behind skin care: Cleansers. *J Cosmet Dermatol* 2018;17:8-14.
70. Ghezzi P, Floridi L, Boraschi D, Cuadrado A, Manda G, Levic S, *et al.* Oxidative stress and inflammation induced by environmental and psychological stressors: A biomarker perspective. *Antioxid Redox Signal* 2018;28:852-72.
71. Choe SJ, Kim D, Kim EJ, Ahn J, Choi E, Son ED, *et al.* Psychological stress deteriorates skin barrier function by activating 11 β -hydroxysteroid dehydrogenase 1 and the HPA axis. *Sci Rep* 2018;8:6334.
72. Chu DK, Chu AW, Rayner DG, Guyatt GH, Yepes-Nuñez JJ, Gomez-Escobar L, *et al.* Topical treatments for atopic dermatitis (eczema): Systematic review and network meta-analysis of randomized trials. *J Allergy Clin Immunol* 2023;152:1493-519.
73. Grogan MD, Bartow-McKenney C, Flowers L, Knight SA, Uberoi A, Grice EA. Research techniques made simple: Profiling the skin microbiota. *J Invest Dermatol* 2019;139:747-52.
74. Kaye AD, Islam RK, Castine AM, Wester GC, Barham WT, Nguyen CG, *et al.* Exploring the relationship between psychiatric illness and dermatological disorders: A narrative review. *Cureus* 2025;17:e78731.
75. Edpuganti S, Shamim A, Gangolli VH, Weerasekara RA, Yellamilli A. Artificial intelligence in cardiovascular imaging: Current landscape, clinical impact, and future directions. *Diagnostics (Craiova)* 2025;13:e211.
76. Gupta M, Khan H, Nijhawan VS, Gaba S, Gupta M. Revisiting a case of parathyroid adenoma with bilateral staghorn calculus. *Cureus* 2020;12:e8251.
77. Sosnowski K, Akarapipad P, Yoon J. The future of microbiome analysis: Biosensor methods for big data collection and clinical diagnostics. *Med Devices Sens* 2020;3:e10085.
78. Li Z, Koban KC, Schenck TL, Giunta RE, Li Q, Sun Y. Artificial intelligence in dermatology image analysis: Current developments and future trends. *J Clin Med* 2022;11:6826.
79. Azambuja RD. The need of dermatologists, psychiatrists and psychologists joint care in psychodermatology. *An Bras Dermatol* 2017;92:63-71.

How to cite this article: Puthooran DM, Syeda A, Kunnatha Cyrilge SR, Nanjundappa DM, Mohamed MH, Masti TS. When skincare backfires: The microbiome-stress-allergy axis in cosmetic hypersensitivity. *CosmoDerma*. 2025;5:119. doi: 10.25259/CSDM_126_2025