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Impact of Air Pollution on Skin Health and Inflammatory Skin Diseases: A Narrative Review

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ABSTRACT

Background: Air pollution has become one of the most significant environmental threats to human health worldwide. Increasing evidence suggests that exposure to air pollutants may adversely affect skin homeostasis and contribute to the development or exacerbation of inflammatory skin diseases.

Aim: The aim of this narrative review was to summarize current evidence regarding the relationship between air pollution and selected inflammatory skin diseases, particularly acne vulgaris, atopic dermatitis, and psoriasis.

Material and methods: A narrative review of scientific literature published within the last ten years was conducted to evaluate the association between air pollution and inflammatory skin diseases.

Results: Available evidence indicates that exposure to pollutants such as particulate matter, nitrogen dioxide, ozone, and polycyclic aromatic hydrocarbons may damage the skin through mechanisms including oxidative stress, disruption of the epidermal barrier, and activation of inflammatory pathways. Epidemiological studies suggest that higher air pollution levels are associated with increased incidence or severity of acne vulgaris, atopic dermatitis, and psoriasis.

Conclusions: Air pollution appears to be an important environmental factor influencing skin health and inflammatory skin diseases. Further research is needed to clarify causal relationships and support development of preventive and therapeutic strategies.

Keywords: *air pollution; inflammatory skin diseases; acne vulgaris; atopic dermatitis; psoriasis; particulate matter; oxidative stress*

1. Introduction

Air pollution is widely recognized as an important environmental determinant of human health. Humans are continuously exposed to airborne pollutants through inhalation, making air quality a key component of environmental exposure 1,2. Air pollution is defined as the contamination of the indoor or outdoor environment by chemical, physical, or biological agents that alter the natural characteristics of the atmosphere 3. It originates from both natural and anthropogenic sources, including industrial emissions, fossil fuel combustion, vehicle exhaust, energy production, agricultural activities, waste incineration, and rapid urbanization, as well as natural events such as wildfires, dust storms, and volcanic eruptions 4,5. Despite global efforts to improve air quality, exposure to harmful pollutants remains widespread. According to the World Health Organization (WHO), nearly 99% of the global population lives in areas exceeding recommended limits, and air pollution remains the leading environmental risk factor, contributing to approximately 7 million premature deaths annually 3. The major air pollutants include particulate matter (PM), particularly PM_{10} and $PM_{2.5}$, as well as nitrogen oxides (NOx), sulfur dioxide (SO_2), ozone (O_3), carbon monoxide (CO), volatile organic compounds (VOCs), and polycyclic aromatic hydrocarbons (PAHs) 1,5. Particulate matter is a heterogeneous mixture of solid particles and liquid droplets suspended in the air and is commonly classified according to aerodynamic diameter into coarse particles (PM_{10}) and fine particles ($PM_{2.5}$). Fine particulate matter is of particular concern because its small size allows it to penetrate biological barriers and potentially enter systemic circulation 6.

The adverse health effects of air pollution have been extensively documented and involve nearly every organ system in the human body. The strongest associations have been demonstrated for respiratory and cardiovascular diseases, including asthma, chronic obstructive pulmonary disease, ischemic heart disease, hypertension, and stroke 7,8.

However, increasing evidence indicates that environmental pollution also has important implications for skin health. As the largest organ of the human body and the primary barrier separating the organism from the external environment, the skin is continuously exposed to environmental stressors such as airborne pollutants, ultraviolet radiation, and climatic factors 9.

Airborne contaminants may adhere to the skin surface or penetrate through the epidermal barrier, triggering a cascade of biological responses including oxidative stress, lipid

peroxidation, inflammation, disruption of skin barrier function, and alterations in the skin microbiome. These processes impair cutaneous homeostasis and may contribute both to premature skin aging and to the development or exacerbation of inflammatory skin diseases 10,11.

In recent years, growing attention has been directed toward the role of environmental pollution in dermatology. Epidemiological and experimental studies suggest that chronic exposure to air pollutants may aggravate or predispose individuals to several common inflammatory skin conditions, particularly acne vulgaris, atopic dermatitis, and psoriasis 12-14.

The purpose of this narrative review is to examine the current evidence linking air pollution with inflammatory skin diseases. Specifically, this review aims to summarize epidemiological data, discuss the molecular and cellular mechanisms underlying pollution-induced skin damage, and highlight potential preventive and therapeutic strategies that may help reduce the dermatological burden associated with environmental pollution.

2. Research materials and methods

This narrative review was conducted in accordance with established methodological principles for narrative literature reviews in biomedical research. A comprehensive literature search was performed using major scientific databases, including PubMed and Google Scholar to identify relevant publications on the impact of air pollution on skin health.

The search strategy combined the following keywords: "air pollution", "particulate matter", "PM2.5", "skin", "skin diseases", "dermatology", "acne vulgaris", "atopic dermatitis", "psoriasis", "oxidative stress", and "environmental exposure". Boolean operators ("AND", "OR") were used to refine the search results and ensure relevance.

The review focused on articles published in English between 2015 and 2026, including original research, epidemiological studies, experimental studies, and previous reviews that explored the association between environmental air pollutants and dermatological outcomes. Additional publications were identified through manual screening of the reference lists of selected articles.

Inclusion criteria were studies investigating the effects of environmental air pollutants on skin physiology, skin barrier function, inflammatory processes, or the incidence and exacerbation of inflammatory skin diseases. Exclusion criteria included articles unrelated to

dermatological outcomes, studies focusing exclusively on indoor air pollution without relevance to skin, and non-peer-reviewed publications.

The selected publications were analyzed qualitatively, and their findings were synthesized to provide a comprehensive overview of the mechanisms through which air pollution may contribute to the development and progression of inflammatory skin diseases.

3. Research results

1. Mechanisms of skin damage

3.1.1 Skin as a barrier and route of pollutant exposure

The skin is the largest organ of the human body and performs multiple essential functions, including protection against environmental stressors, prevention of excessive water loss, thermoregulation, immune defense, sensory perception, and metabolic processes such as vitamin D synthesis 10,15. Owing to its continuous exposure to the external environment, the skin serves as the primary interface between the body and environmental pollutants.

Structurally, the skin consists of three main layers: the epidermis, dermis, and hypodermis, each contributing to the maintenance of cutaneous homeostasis. The outermost layer of the epidermis, the stratum corneum, plays a central role in barrier function. It is composed of corneocytes embedded in a lipid-rich matrix, forming a highly organized structure that restricts the penetration of harmful substances and limits transepidermal water loss 16,17.

Maintenance of an intact epidermal barrier is crucial for protection against pathogens, irritants, and environmental toxins. However, disruption of this barrier increases skin permeability and facilitates the penetration of airborne pollutants, including particulate matter (PM), ozone (O_3), and volatile organic compounds (VOCs). These agents may accumulate on the skin surface or penetrate into deeper layers, triggering a cascade of biological responses that contribute to cutaneous damage 18,19.

Overall, impairment of the skin barrier represents a one of the key mechanism through which air pollutants exert their detrimental effects on skin health 20.

3.1.2 Skin barrier dysfunction and transepidermal water loss

The functional consequences of epidermal barrier impairment can be assessed using transepidermal water loss (TEWL), a widely applied non-invasive marker of skin barrier integrity. TEWL reflects passive water diffusion through the stratum corneum, and its elevation indicates compromised barrier function, which is associated with increased skin dryness, sensitivity, and susceptibility to irritation 21. In clinical settings, TEWL is commonly

used as an indicator of disease severity in inflammatory skin conditions such as atopic dermatitis and psoriasis 22.

Accumulating evidence suggests that exposure to air pollutants significantly affects TEWL levels. Experimental studies using in vitro models, animal systems, and human subjects have demonstrated that exposure to particulate matter (PM) and volatile organic compounds (VOCs) leads to a measurable increase in TEWL, indicating direct impairment of barrier function 20, 23-26.

In addition, epidemiological data indicate a positive association between exposure to fine particulate matter ($PM_{2.5}$) and elevated TEWL. Pollution exposure has also been associated with alterations in sebum production and composition, which may further contribute to barrier instability and increased susceptibility to environmental stressors 13,27.

3.1.3 Structural proteins and lipid damage

Beyond functional impairment, air pollution also affects key structural components of the epidermis that are essential for maintaining barrier integrity. One of the most important proteins involved in this process is filaggrin, which plays a crucial role in keratin filament aggregation and the formation of the stratum corneum. In addition, filaggrin degradation products contribute to the formation of the natural moisturizing factor (NMF), which is essential for maintaining adequate skin hydration 28,29.

Alterations in filaggrin expression have been associated with impaired barrier function and increased susceptibility to environmental stressors. Experimental studies indicate that exposure to particulate matter may modulate the expression of barrier-related proteins, including filaggrin, thereby exacerbating epidermal dysfunction 26,30.

Structural abnormalities within the stratum corneum, such as increased exposure of corneodesmosomes, may also occur under conditions of barrier disruption. These changes can facilitate adhesion and colonization by pathogenic microorganisms, including Staphylococcus aureus, thereby promoting cutaneous inflammation 31,32.

Air pollutants have also been implicated in lipid peroxidation, leading to qualitative and quantitative alterations in epidermal lipids. As intercellular lipids are essential for maintaining the cohesion and impermeability of the stratum corneum, their degradation further compromises barrier integrity and increases susceptibility to external irritants and allergens 33.

3.1.4 Skin microbiome alterations

An integral component of skin homeostasis is the cutaneous microbiome, a complex ecosystem of microorganisms that contributes to the maintenance of immune balance and epidermal integrity. The skin microbiota plays a crucial role in protecting against pathogenic colonization and modulating local immune responses 34.

The composition of the skin microbiome varies across anatomical sites and is influenced by multiple factors, including skin pH, sebum production, hydration, age, and environmental exposure 35. Disruption of this balanced ecosystem may promote inflammatory responses and increase susceptibility to skin disorders 36.

Exposure to air pollutants can alter both the composition and function of the skin microbiota. Pollutants such as ozone and particulate matter may influence the composition and diversity of the skin microbiome, potentially disrupting the balance of commensal organisms 37.

Experimental studies have demonstrated that exposure to ozone may lead to a substantial reduction---up to 50%---in the resident skin microflora, indicating its potential bactericidal effects. Conversely, pollutant-induced disruption of the skin barrier may facilitate colonization by opportunistic or pathogenic microorganisms, further contributing to inflammation and the development of skin diseases 38.

3.1.5 Oxidative stress and inflammation

Beyond microbial disruption, exposure to air pollution induces oxidative stress and inflammatory responses, which represent central mechanisms of pollution-related skin damage. A key pathway underlying these effects is the excessive generation of reactive oxygen species (ROS) following exposure to environmental pollutants such as particulate matter, ozone, and polycyclic aromatic hydrocarbons 12,39.

Particulate matter (PM) contributes to oxidative stress through multiple pathways, leading to both exogenous and endogenous ROS generation. Due to its highly reactive surface, PM can directly promote the formation of free radicals. In addition, its components, including transition metals, polycyclic aromatic hydrocarbons, and quinones, are capable of generating ROS through chemical reactions such as Fenton-like processes and redox cycling 40.

Beyond direct ROS production, PM exposure may also enhance intracellular oxidative stress by disrupting cellular homeostasis. It has been shown to induce dysfunction of key

organelles, particularly mitochondria and the endoplasmic reticulum. Mitochondrial impairment increases endogenous ROS production, while endoplasmic reticulum stress further contributes to oxidative imbalance through disturbances in calcium signaling and protein folding 41,42.

In addition to direct molecular damage, oxidative stress activates intracellular signaling pathways, including nuclear factor kappa B (NF-κB), which plays a central role in regulating inflammatory responses. This activation leads to increased production of pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), thereby amplifying local inflammation 43.

When ROS production exceeds the skin's antioxidant defense capacity, oxidative stress occurs, resulting in damage to lipids, proteins, and DNA, and ultimately impairing cellular and tissue function. Collectively, these processes disrupt skin homeostasis and may contribute to the development or exacerbation of inflammatory skin diseases 13.

3.2 Air pollution and acne vulgaris

Acne vulgaris is a common chronic inflammatory skin disease, particularly prevalent among adolescents and young adults. It is characterized by the presence of comedones, papules, and pustules, primarily affecting the face, as well as the neck, chest, and back. Its pathogenesis is multifactorial and involves increased sebum production, follicular hyperkeratinization, microbial colonization---mainly by Cutibacterium acnes---and inflammation. In addition to genetic and hormonal influences, environmental factors, including air pollution, are increasingly recognized as important contributors 44,45.

Epidemiological studies indicate that exposure to air pollutants, particularly particulate matter (PM) and nitrogen dioxide (NO_2), is associated with increased acne prevalence and a higher frequency of dermatological consultations. The underlying mechanisms appear to involve a combination of oxidative stress, alterations in sebum composition, and enhanced inflammatory responses 27,45,46.

Air pollutants such as particulate matter and ozone (O_3) can induce the generation of reactive oxygen species, leading to oxidative stress and lipid peroxidation. One of the key consequences is the oxidation of squalene, a major component of sebum, into comedogenic squalene peroxides, which contribute to follicular obstruction and lesion formation. At the same time, pollution exposure may alter sebum quantity and composition, further promoting an environment conducive to acne development 12,47.

Additionally, exposure to other environmental pollutants, such as cigarette smoke and PAHs, may further contribute to acne pathogenesis by promoting inflammation and acneiform eruptions 13.

Overall, current evidence suggests that air pollution may exacerbate acne through interconnected mechanisms involving oxidative stress, dysregulation of sebum, and inflammation

3.3 Air pollution and atopic dermatitis

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease characterized by pruritus, erythematous scaling plaques, crusting, fissuring, and lichenification, typically affecting the face, neck, and flexural areas such as the antecubital and popliteal fossae. It is one of the most common skin disorders in childhood, with onset occurring in up to 60% of cases within the first year of life and in the majority of patients before the age of five 48,49.

The pathophysiology of AD is complex and multifactorial, involving epidermal barrier dysfunction, immune dysregulation, and alterations in the skin microbiome. Increasing attention has been directed toward environmental factors, including air pollution, which may contribute to both the development and exacerbation of the disease 50. Epidemiological studies further suggest that exposure to ambient air pollution is associated with an increased incidence of atopic dermatitis, often demonstrating a dose--response relationship between pollutant levels and disease risk 51,52.

In patients with AD, who already present with impaired barrier function, exposure to air pollutants may further exacerbate oxidative stress and contribute to disease progression. Pollutants can induce the production of reactive oxygen species, leading to oxidative stress and subsequent damage to cellular components. This process promotes lipid peroxidation and depletion of cutaneous antioxidants, further weakening the skin barrier. In parallel, exposure to pollutants has been associated with increased TEWL and reduced expression of structural proteins such as filaggrin, thereby facilitating the penetration of irritants and allergens 53.

At the molecular level, PAHs can activate the aryl hydrocarbon receptor (AhR), a transcription factor involved in the regulation of inflammation and epidermal homeostasis. Activation of this pathway has been linked to increased expression of mediators associated with pruritus and inflammation, as well as to further impairment of the skin barrier, contributing to key clinical features of AD, including itch hypersensitivity and chronic inflammation 12,54.

Moreover, pollution-induced oxidative stress may activate inflammatory signaling pathways, including nuclear factor kappa B (NF-κB), leading to increased production of pro-inflammatory cytokines. Environmental exposure has also been associated with a shift toward a Th2-dominant immune response, characterized by increased levels of cytokines such as interleukin-4 (IL-4) and reduced interferon-gamma expression, which further contributes to disease progression 12,53,55.

Overall, current evidence suggests that air pollution may exacerbate atopic dermatitis through a combination of oxidative stress, barrier disruption, and immune dysregulation, acting in a highly interconnected manner.

3.4 Air pollution and psoriasis

Psoriasis is a chronic immune-mediated inflammatory skin disease characterized by abnormal keratinocyte proliferation and dysregulated epidermal turnover. The disease is clinically heterogeneous and may present in several phenotypes, including plaque, guttate, pustular, erythrodermic, and inverse psoriasis. Plaque psoriasis represents the most common form, accounting for approximately 80--90% of cases. It typically manifests as well-demarcated erythematous plaques covered with silvery-white scales, most frequently affecting the extensor surfaces of the elbows and knees, as well as the scalp and trunk 56.

The pathogenesis of psoriasis is multifactorial and involves genetic susceptibility, environmental triggers, and immune dysregulation. In contrast to atopic dermatitis, which is predominantly associated with a Th2-mediated immune response, psoriasis is primarily driven by the IL-23/Th17 inflammatory axis, which plays a central role in disease development and persistence 57.

Increasing evidence suggests that environmental factors, including air pollution, may contribute to both the onset and exacerbation of psoriasis. Epidemiological studies conducted in populations from China, Europe, and the United States have demonstrated that both short-term and long-term exposure to air pollutants such as particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), and ozone is associated with increased psoriasis incidence, greater disease severity, and more frequent disease flares 58-63.

Several mechanisms have been proposed to explain this association. Air pollutants may influence immune responses through pathways involved in inflammatory signaling and T-cell differentiation. In particular, particulate matter and PAHs can activate the AhR, a ligand-activated transcription factor involved in cellular responses to environmental toxins. Activation of AhR has been associated with modulation of T-cell activity and may promote

differentiation of Th17 cells, which are key mediators of psoriatic inflammation through the production of cytokines such as interleukin-17 (IL-17) 12,64.

In addition, pollution-induced oxidative stress and inflammatory signaling may further enhance immune activation in psoriatic skin, potentially contributing to the initiation or exacerbation of psoriatic lesions 65. Collectively, these findings suggest that environmental pollution may represent an important external factor capable of influencing psoriasis activity through mechanisms involving oxidative stress and immune dysregulation.

Disease	Dominant immune pathway	Major pollutants associated with disease activity	Proposed mechanisms
Acne vulgaris	Innate immune activation and sebaceous gland dysregulation	$PM_{2.5}$, NO_2, O_3	Sebum oxidation, lipid peroxidation, altered sebum composition, and inflammation within the pilosebaceous unit
Atopic dermatitis (AD)	Th2-mediated immune response	$PM_{2.5}$, VOCs, NO_2	Epidermal barrier disruption, increased TEWL, oxidative stress, and Th2-skewed cytokine responses
Psoriasis	IL-23/Th17-mediated inflammation	$PM_{2.5}$, NO_2, PAHs	AhR activation, Th17 polarization, oxidative stress, and immune activation

Table 1. Summary of proposed mechanisms linking air pollution with inflammatory skin diseases.

4. Discussion

Air pollution has emerged as an important environmental determinant of human health, and increasing evidence suggests that it also plays a significant role in inflammatory skin diseases. This narrative review summarizes current evidence linking exposure to pollutants with acne vulgaris, atopic dermatitis, and psoriasis.

Several biological mechanisms appear to underlie pollution-induced skin damage. One of the most consistently described processes is oxidative stress resulting from excessive production of ROS following exposure to pollutants such as particulate matter, ozone, and nitrogen oxides. Oxidative stress may lead to lipid peroxidation, depletion of cutaneous antioxidants, and cellular damage, ultimately disrupting skin homeostasis 12-14.

Another important mechanism involves impairment of epidermal barrier integrity. Air pollutants may alter the structure and function of the stratum corneum by affecting structural proteins and increasing TEWL, thereby facilitating the penetration of irritants and allergens and promoting cutaneous inflammation 21-22.

Although these mechanisms are shared across several dermatological conditions, the downstream immunological responses differ between diseases. Acne vulgaris is primarily associated with alterations in sebum composition and inflammation within the pilosebaceous unit 45. In AD, pollution appears to aggravate barrier dysfunction and Th2-mediated inflammation, whereas psoriasis is mainly linked to dysregulation of the IL-23/Th17 axis and pollutant-induced immune activation 50,57.

Epidemiological studies conducted in different populations have reported associations between exposure to $PM_{2.5}$, PM_{10}, nitrogen dioxide, ozone, and increased incidence or severity of inflammatory skin diseases 14, 51, 58, 62. However, many available studies remain observational and may be influenced by confounding factors such as climate, lifestyle, genetic susceptibility, and socioeconomic status. Therefore, further longitudinal and mechanistic studies are required to better clarify causal relationships and identify the most clinically relevant exposure pathways.

Improved understanding of pollution-induced skin damage may have important clinical and public health implications. Identification of environmental risk factors could support preventive strategies aimed at reducing exposure and may contribute to the development of

targeted therapeutic approaches focused on oxidative stress, barrier dysfunction, and immune dysregulation.

5. Conclusions

Current evidence suggests that air pollution is an important environmental factor that may contribute to both the development and exacerbation of inflammatory skin diseases. Experimental and epidemiological studies indicate that pollutants such as particulate matter, ozone, and nitrogen dioxide may impair skin homeostasis through oxidative stress, epidermal barrier disruption, and inflammatory activation.

Despite growing evidence supporting the relationship between air pollution and skin disease, further well-designed studies are needed to better define causal mechanisms and evaluate effective preventive and therapeutic strategies. A better understanding of the interaction between environmental exposure and skin health may contribute to improved management of common inflammatory dermatological conditions.

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During the preparation of this work, the authors used Chat GPT (OpenAI) to improve grammar and language corrections. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

1. Manisalidis I, Stavropoulou E, Stavropoulos A, Bezirtzoglou E. Environmental and health impacts of air pollution: a review. *Front Public Health.* 2020 Feb 20;8:14. https://doi.org/10.3389/fpubh.2020.00014. PMID: 32154200; PMCID: PMC7044178 https://doi.org/10.3389/fpubh.2020.00014.

2. Cohen AJ, Brauer M, Burnett R, et al. Estimates and 25-year trends of the global burden of disease attributable to air pollution. *Lancet.* 2017 Jun 17;389(10082):1907--1918. https://doi.org/10.1016/S0140-6736(17)30505-6 https://doi.org/10.1016/S0140-6736(17

3. World Health Organization. Air pollution. World Health Organization. Available from: https://www.who.int/health-topics/air-pollution#tab=tab_2. Accessed March 10, 2026 https://www.who.int/health-topics/air-pollution#tab=tab_2.

4. Chen F, Zhang W, Bani Mfarrej MF, Saleem MH, Khan KA, Ma J, Raposo A, Han H. Breathing in danger: understanding the multifaceted impact of air pollution on health impacts. *Ecotoxicol Environ Saf.* 2024;280:116532 https://doi.org/10.1016/j.ecoenv.2024.116532

5. Damiati S, AlMashrea BA, Rabiei N, Sujatha AP, Sabir DK, Alhosani M, Kodzius R. Aerosol pollutants and health: role of size and chemical composition. *Public Health Chall.* 2025 Sep 26;4(4):e70134. https://doi.org/10.1002/puh2.70134. PMID: 41020050; PMCID: PMC12466196 https://doi.org/10.1002/puh2.70134.

6. Hamanaka RB, Mutlu GM. Particulate matter air pollution: effects on the respiratory system. *J Clin Invest.* 2025 Sep 2;135(17):e194312. https://doi.org/10.1172/JCI194312. PMID: 40892514; PMCID: PMC12404767 https://doi.org/10.1172/JCI194312.

7. Forastiere F, Orru H, Krzyzanowski M, Spadaro JV. *The last decade of air pollution epidemiology and the challenges of quantitative risk assessment. Environ Health*. 2024 Nov 14;23(1):98. doi: 10.1186/s12940-024-01136-5. PMID: 39543692; PMCID: PMC11566658

8. Landrigan PJ, Fuller R, Acosta NJR, Adeyi O, Arnold R, Basu N, et al. The Lancet Commission on pollution and health. *Lancet.* 2018 Feb 3;391(10119):462--512. https://doi.org/10.1016/S0140-6736(17)32345-0 https://doi.org/10.1016/S0140-6736(17

9. Krutmann J, Bouloc A, Sore G, Bernard BA, Passeron T. The skin aging exposome. *J Dermatol Sci.* 2017 Mar;85(3):152--161 https://doi.org/10.1016/j.jdermsci.2016.09.015

10. Dąbrowska AK, Spano F, Derler S, Adlhart C, Spencer ND, Rossi RM. The relationship between skin function, barrier properties, and body-dependent factors. *Skin Res Technol*. 2018;24:165--174. https://doi.org/10.1111/srt.12424 https://doi.org/10.1111/srt.12424

11. Schikowski T, Hüls A. *Air Pollution and Skin Aging. Curr Environ Health Rep*. 2020 Mar;7(1):58-64. doi: 10.1007/s40572-020-00262-9. PMID: 31927691

12. Han HS, Seok J, Park KY. *Air Pollution and Skin Diseases. Ann Dermatol*. 2025 Apr;37(2):53-67. doi: 10.5021/ad.24.159. PMID: 40165563; PMCID: PMC11965873

13. Bocheva G, Slominski RM, Slominski AT. *Environmental Air Pollutants Affecting Skin Functions with Systemic Implications. Int J Mol Sci*. 2023 Jun 22;24(13):10502. doi: 10.3390/ijms241310502. PMID: 37445680; PMCID: PMC10341863

14. Singh N, Schikowski T, Krutmann J. Air Pollution and Skin Diseases: A Systematic Review of Epidemiological Evidence. *Am J Clin Dermatol*. 2026 Mar;27(2):261-276. doi: 10.1007/s40257-026-01006-5. Epub 2026 Feb 6. PMID: 41649748; PMCID: PMC13032956

15. Baker P, Huang C, Radi R, Moll SB, Jules E, Arbiser JL. Skin Barrier Function: The Interplay of Physical, Chemical, and Immunologic Properties. *Cells*. 2023; 12(23):2745 https://doi.org/10.3390/cells12232745

16. Lee T, Friedman A. Skin Barrier Health: Regulation and Repair of the Stratum Corneum and the Role of Over-the-Counter Skin Care. *J Drugs Dermatol*. 2016 Sep 1;15(9):1047-51. PMID: 27602965

17. Rajkumar J, Chandan N, Lio P, Shi V. The Skin Barrier and Moisturization: Function, Disruption, and Mechanisms of Repair. *Skin Pharmacol Physiol*. 2023;36(4):174-185. doi: 10.1159/000534136. Epub 2023 Sep 15. PMID: 37717558

18. Araviiskaia E, Berardesca E, Bieber T, Gontijo G, Sanchez Viera M, Marrot L, Chuberre B, Dreno B. *The impact of airborne pollution on skin. J Eur Acad Dermatol Venereol*. 2019 Aug;33(8):1496-1505. doi: 10.1111/jdv.15583. Epub 2019 Apr 26. PMID: 30897234; PMCID: PMC6766865

19. Zieneldien T, Ma S, Tan IJ, Kim J, Busot D, Cohen BA. The Environmental Determinants of Skin Health: Linking Climate Change, Air Pollution, and the Dermatologic Disease Burden. *Int J Environ Res Public Health*. 2025 Dec 4;22(12):1820. doi: 10.3390/ijerph22121820. PMID: 41464454; PMCID: PMC12732634

20. Hieda DS, Anastacio da Costa Carvalho L, Vaz de Mello B, Oliveira EA, Romano de Assis S, Wu J, Du-Thumm L, Viana da Silva CL, Roubicek DA, Maria-Engler SS, Berlanga de Moraes Barros S. *Air Particulate Matter Induces Skin Barrier Dysfunction and Water Transport Alteration on a*

Reconstructed Human Epidermis Model. J Invest Dermatol. 2020 Dec;140(12):2343-2352.e3. doi: 10.1016/j.jid.2020.03.971. Epub 2020 Apr 25. PMID: 32339540

21. Alexander H, Brown S, Danby S, Flohr C. Research Techniques Made Simple: Transepidermal Water Loss Measurement as a Research Tool. *J Invest Dermatol*. 2018 Nov;138(11):2295-2300.e1. doi: 10.1016/j.jid.2018.09.001. PMID: 30348333

22. Montero-Vilchez T, Segura-Fernández-Nogueras MV, Pérez-Rodríguez I, Soler-Gongora M, Martinez-Lopez A, Fernández-González A, Molina-Leyva A, Arias-Santiago S. Skin Barrier Function in Psoriasis and Atopic Dermatitis: Transepidermal Water Loss and Temperature as Useful Tools to Assess Disease Severity. *J Clin Med*. 2021 Jan 19;10(2):359. doi: 10.3390/jcm10020359. PMID: 33477944; PMCID: PMC7833436

23. Oh SJ, Yoon D, Park JH, Lee JH. Effects of Particulate Matter on Healthy Skin: A Comparative Study between High- and Low-Particulate Matter Periods. *Ann Dermatol*. 2021 Jun;33(3):263-270. doi: 10.5021/ad.2021.33.3.263. Epub 2021 May 4. PMID: 34079186; PMCID: PMC8137329

24. Kwon IJ, Lee EJ, Park JH, Kim JY, Park S, Bae YJ, Hwang S, Na HW, Cha N, Jang G, Kim HJ, Lee HK, Oh SH. *Independent and Combined Effects of Particulate Matter and Sleep Deprivation on Human Skin Barrier. Ann Dermatol*. 2025 Jun;37(3):131-139. doi: 10.5021/ad.25.003. PMID: 40432361; PMCID: PMC12117546

25. Dijkhoff IM, Drasler B, Karakocak BB, Petri-Fink A, Valacchi G, Eeman M, Rothen-Rutishauser B. Impact of airborne particulate matter on skin: a systematic review from epidemiology to in vitro studies. *Part Fibre Toxicol*. 2020 Jul 25;17(1):35. doi: 10.1186/s12989-020-00366-y. PMID: 32711561; PMCID: PMC7382801

26. Kim BE, Kim J, Goleva E, Berdyshev E, Lee J, Vang KA, Lee UH, Han S, Leung S, Hall CF, Kim NR, Bronova I, Lee EJ, Yang HR, Leung DY, Ahn K. *Particulate matter causes skin barrier dysfunction. JCI Insight*. 2021 Mar 8;6(5):e145185. doi: 10.1172/jci.insight.145185. PMID: 33497363; PMCID: PMC8021104

27. Okeah IR, Afzal UM, Ali FR. Impact of environmental pollution on acne: a systematic review. *Skin Health Dis*. 2025 Dec 30;6(1):12-19. doi: 10.1093/skinhd/vzaf090. PMID: 41646535; PMCID: PMC12867942

28. Hoober JK, Eggink LL. *The Discovery and Function of Filaggrin. Int J Mol Sci*. 2022 Jan 27;23(3):1455. doi: 10.3390/ijms23031455. PMID: 35163390; PMCID: PMC8835998

29. Kim Y, Lim KM. *Skin barrier dysfunction and filaggrin. Arch Pharm Res*. 2021 Jan;44(1):36-48. doi: 10.1007/s12272-021-01305-x. Epub 2021 Jan 18. PMID: 33462753

30. Martic I, Jansen-Dürr P, Cavinato M. *Effects of Air Pollution on Cellular Senescence and Skin Aging. Cells*. 2022 Jul 17;11(14):2220. doi: 10.3390/cells11142220. PMID: 35883663; PMCID: PMC9320051

31. Williams, M.R., Gallo, R.L. The Role of the Skin Microbiome in Atopic Dermatitis. *Curr Allergy Asthma Rep* 15, 65 (2015) https://doi.org/10.1007/s11882-015-0567-4

32. Feuillie C, Vitry P, McAleer MA, Kezic S, Irvine AD, Geoghegan JA, Dufrêne YF. *Adhesion of Staphylococcus aureus to Corneocytes from Atopic Dermatitis Patients Is Controlled by Natural*

Moisturizing Factor Levels. mBio. 2018 Aug 14;9(4):e01184-18. doi: 10.1128/mBio.01184-18. PMID: 30108169; PMCID: PMC6094479

33. Li Pomi F, Gammeri L, Borgia F, Di Gioacchino M, Gangemi S. Oxidative Stress and Skin Diseases: The Role of Lipid Peroxidation. *Antioxidants (Basel)*. 2025 May 7;14(5):555. doi: 10.3390/antiox14050555. PMID: 40427437; PMCID: PMC12108378

34. Li Z, Zhang J, Zhang Y, Chen H, Bao Y. Skin Microbiome in Health and Disease: Mechanisms and Emerging Therapeutic Strategies. *Clin Cosmet Investig Dermatol*. 2025 Dec 18;18:3443-3455. doi: 10.2147/CCID.S571984. PMID: 41438680; PMCID: PMC12720977

35. Smythe P, Wilkinson HN. The Skin Microbiome: Current Landscape and Future Opportunities. *Int J Mol Sci*. 2023 Feb 16;24(4):3950. doi: 10.3390/ijms24043950. PMID: 36835363; PMCID: PMC9963692

36. Egert, M., Simmering, R. and Riedel, C. (2017), The Association of the Skin Microbiota With Health, Immunity, and Disease. Clin. Pharmacol. Ther., 102: 62-69 https://doi.org/10.1002/cpt.698

37. Hong JY, Kwon D, Park KY. Microbiome-Based Interventions for Skin Aging and Barrier Function: A Comprehensive Review. *Ann Dermatol*. 2025 Oct;37(5):259-268. doi: 10.5021/ad.25.009. PMID: 41044805; PMCID: PMC12505367

38. Mancebo SE, Wang SQ. *Recognizing the impact of ambient air pollution on skin health. J Eur Acad Dermatol Venereol*. 2015 Dec;29(12):2326-32. doi: 10.1111/jdv.13250. Epub 2015 Aug 20. PMID: 26289769; PMCID: PMC5916788

39. Gangwar RS, Bevan GH, Palanivel R, Das L, Rajagopalan S. Oxidative stress pathways of air pollution mediated toxicity: Recent insights. *Redox Biol*. 2020 Jul;34:101545. doi: 10.1016/j.redox.2020.101545. Epub 2020 May 23. PMID: 32505541; PMCID: PMC7327965

40. Vilas-Boas V, Chatterjee N, Carvalho A, Alfaro-Moreno E. *Particulate matter-induced oxidative stress -- mechanistic insights and antioxidant approaches reported in in vitro studies. Environ Toxicol Pharmacol*. 2024;110:104529 https://doi.org/10.1016/j.etap.2024.104529

41. Wang Y, Zhang M, Li Z, Yue J, Xu M, Zhang Y, Yung KKL, Li R. *Fine particulate matter induces mitochondrial dysfunction and oxidative stress in human SH-SY5Y cells. Chemosphere*. 2019;218:577-588 https://doi.org/10.1016/j.chemosphere.2018.11.149

42. Qin Y, Qian C, Li W, Wang Q, Sheng Q, Chen Z, Zhang W, Li W, Ge G, Yan Z, Geng D. Oxidative Stress: Molecular Mechanisms, Diseases, and Therapeutic Targets. *MedComm (2020)*. 2026 Jan 29;7(2):e70600. doi: 10.1002/mco2.70600. PMID: 41624249; PMCID: PMC12856066

43. Liu S, Liu J, Wang Y, Deng F, Deng Z. Oxidative Stress: Signaling Pathways, Biological Functions, and Disease. *MedComm (2020)*. 2025 Jul 1;6(7):e70268. doi: 10.1002/mco2.70268. PMID: 40599237; PMCID: PMC12209598

44. Hazarika N. Acne vulgaris: new evidence in pathogenesis and future modalities of treatment. *J Dermatolog Treat*. 2021 May;32(3):277-285. doi: 10.1080/09546634.2019.1654075. Epub 2019 Aug 29. PMID: 31393195

45. Krutmann J, Moyal D, Liu W, Kandahari S, Lee GS, Noppakun N, Xiang LF, Seité S. Pollution and acne: is there a link?. *Clin Cosmet Investig Dermatol*. 2017;10:199-204 https://doi.org/10.2147/CCID.S131323

46. Li X, An SJ, Liu XL, Ji AL, Cao Y, Xiang Y, Ma XY, Hu Q, Yuan ZQ, Li YF, Lu YG, Cai TJ. The Association Between Short-Term Air Pollution Exposure and Post-Adolescent Acne: The Evidence from a Time Series Analysis in Xi'an, China. *Clin Cosmet Investig Dermatol*. 2021 Jun 25;14:723-731. doi: 10.2147/CCID.S320248. PMID: 34211290; PMCID: PMC8241005

47. Gu X, Li Z, Su J. Air pollution and skin diseases: A comprehensive evaluation of the associated mechanism. *Ecotoxicol Environ Saf*. 2024 Jun 15;278:116429. doi: 10.1016/j.ecoenv.2024.116429. Epub 2024 May 7. PMID: 38718731

48. Meledathu S, Naidu MP, Brunner PM. *Update on atopic dermatitis. J Allergy Clin Immunol*. 2025 Apr;155(4):1124-1132. doi: 10.1016/j.jaci.2025.01.013. Epub 2025 Jan 22. PMID: 39855361

49. Carr S, Pratt R, White F, Watson W. *Atopic dermatitis. Allergy Asthma Clin Immunol*. 2024 Dec 9;20(Suppl 3):63. doi: 10.1186/s13223-024-00927-2. PMID: 39654051; PMCID: PMC11629513

50. Çetinarslan T, Kümper L, Fölster-Holst R. *The immunological and structural epidermal barrier dysfunction and skin microbiome in atopic dermatitis-an update. Front Mol Biosci*. 2023 Aug 16;10:1159404. doi: 10.3389/fmolb.2023.1159404. PMID: 37654796; PMCID: PMC10467310

51. Luo P, Wang D, Luo J, Li S, Li MM, Chen H, Duan Y, Fan J, Cheng Z, Zhao MM, Liu X, Wang H, Luo XY, Zhou L. Relationship between air pollution and childhood atopic dermatitis in Chongqing, China: A time-series analysis. *Front Public Health*. 2022 Oct 6;10:990464. doi: 10.3389/fpubh.2022.990464. PMID: 36276372; PMCID: PMC9583006

52. Park SK, Kim JS, Seo HM. Exposure to air pollution and incidence of atopic dermatitis in the general population: A national population-based retrospective cohort study. *J Am Acad Dermatol*. 2022 Dec;87(6):1321-1327. doi: 10.1016/j.jaad.2021.05.061. Epub 2021 Jul 6. PMID: 34242692

53. Fadadu RP, Abuabara K, Balmes JR, Hanifin JM, Wei ML. Air Pollution and Atopic Dermatitis, from Molecular Mechanisms to Population-Level Evidence: A Review. *Int J Environ Res Public Health*. 2023 Jan 31;20(3):2526. doi: 10.3390/ijerph20032526. PMID: 36767891; PMCID: PMC9916398

54. Lai A, Owens K, Patel S, Nicholas M. *The Impact of Air Pollution on Atopic Dermatitis. Curr Allergy Asthma Rep*. 2023 Aug;23(8):435-442. doi: 10.1007/s11882-023-01095-w. Epub 2023 May 26. PMID: 37233850; PMCID: PMC10214316

55. Liu CX, Li L, Zeng YP. The Role of Air Pollution in the Pathogenesis of Atopic Dermatitis, With a Focus on Oxidative Stress. *Clin Transl Allergy*. 2025 Sep;15(9):e70104. doi: 10.1002/clt2.70104. PMID: 40955089; PMCID: PMC12437320

56. Raharja A, Mahil SK, Barker JN. Psoriasis: a brief overview. *Clin Med (Lond)*. 2021 May;21(3):170-173. doi: 10.7861/clinmed.2021-0257. PMID: 34001566; PMCID: PMC8140694

57. Rendon A, Schäkel K. *Psoriasis Pathogenesis and Treatment. Int J Mol Sci*. 2019 Mar 23;20(6):1475. doi: 10.3390/ijms20061475. PMID: 30909615; PMCID: PMC6471628

58. Wu J, Ma Y, Yang J, Tian Y. Exposure to Air Pollution, Genetic Susceptibility, and Psoriasis Risk in the UK. *JAMA Netw Open*. 2024 Jul 1;7(7):e2421665. doi: 10.1001/jamanetworkopen.2024.21665. PMID: 39012635; PMCID: PMC11252902

59. Bellinato F, Adami G, Vaienti S, Benini C, Gatti D, Idolazzi L, Fassio A, Rossini M, Girolomoni G, Gisondi P. *Association Between Short-term Exposure to Environmental Air Pollution and Psoriasis Flare. JAMA Dermatol*. 2022 Apr 1;158(4):375-381. doi: 10.1001/jamadermatol.2021.6019. PMID: 35171203; PMCID: PMC8851365

60. Hu H, Yang X, Chen Q, Huang X, Cao X, Zhang X, Xu Y. Causal association between air pollution and autoimmune diseases: a two-sample Mendelian randomization study. *Front Public Health*. 2024 Mar 28;12:1333811. doi: 10.3389/fpubh.2024.1333811. PMID: 38605869; PMCID: PMC11007215

61. Wang H, Liang J, Huang M, Li W, Sun J, Zhang S, Huang Z. Short-Term Exposure to Ambient Air Pollution and Psoriasis in Guangzhou, China: Estimating the Association and Population Attributable Fraction. *Atmosphere*. 2026; 17(2):198 https://doi.org/10.3390/atmos17020198

62. Yang, Y., Huang, B., Zhao, L., Liu, J., Han, Z., Xiao, M., Zhao, Y., Liu, C., Zhang, R., Li, P. (2024). A Systematic Review and Meta-analysis of Short-term Relationship between Ambient Air Pollution and Psoriasis. Aerosol Air Qual. Res. 24, 240062 https://doi.org/10.4209/aaqr.240062

63. Bi L, Wang Z, Yang J, Tian Z, Zhao H, Xu Z, Chen K, Zhuang Z, Huang X, Ouyang H, Sheng Y, Cui Y. Ambient air pollution and psoriasis: a nationwide cross-sectional study of 149 744 Chinese patients in 31 provinces. *J Glob Health*. 2026 Feb 27;16:04010. doi: 10.7189/jogh.16.04010. PMID: 41747084; PMCID: PMC12967242

64. Brembilla NC and Boehncke W-H (2023) Revisiting the interleukin 17 family of cytokines in psoriasis: pathogenesis and potential targets for innovative therapies. Front. Immunol. 14:1186455. doi: 10.3389/fimmu.2023.1186455

65. Pleńkowska J, Gabig-Cimińska M, Mozolewski P. *Oxidative Stress as an Important Contributor to the Pathogenesis of Psoriasis. Int J Mol Sci*. 2020 Aug 27;21(17):6206. doi: 10.3390/ijms21176206. PMID: 32867343; PMCID: PMC7503883