REVIEW ARTICLE

SKIN BARRIER DYSFUNCTION INDUCED BY ENVIRONMENTAL POLLUTANTS: OXIDATIVE STRESS, INFLAMMATION, AND IMMUNE RESPONSE

Dysfunkcja bariery skórnej indukowana przez zanieczyszczenia środowiskowe: stres oksydacyjny, zapalenie i odpowiedź immunologiczna

Oliwia Sędziak

Faculty of Medicine, University of Opole, Poland

Oliwia Sędziak – 0009-0003-2128-0662

Abstract

Environmental pollutants, including particulate matter, ozone, and polycyclic aromatic hydrocarbons, represent significant factors that impair skin function and integrity. Exposure to these substances has been shown to induce reactive oxygen species formation, activate inflammatory pathways, and impair the epidermal barrier. Oxidative stress and chronic inflammation result in lipid imbalance, collagen degradation, increased transepidermal water loss, and dysregulation of immune responses. Clinically, this manifests as accelerated skin aging, pigmentation disorders, and exacerbation of inflammatory dermatoses such as atopic dermatitis, psoriasis, and acne vulgaris. However, the skin has many complex defence mechanisms, including antioxidant systems, lipid barrier repair processes, and immune regulation. The skin microbiome also plays an important role in maintaining homeostasis. Under chronic exposure, these mechanisms gradually become impaired, leading to persistent skin barrier dysfunction. Understanding the molecular basis of these processes is crucial for developing effective preventive and therapeutic strategies, including the use of antioxidants, Nrf2 pathway activators, and interventions that support the skin microbiome. Environmental pollutants should be recognized as an important modifiable risk factor for skin diseases and considered in routine dermatological practice.

Streszczenie

Zanieczyszczenia środowiskowe, w tym pyły zawieszone, ozon i wielopierścieniowe węglowodory aromatyczne, stanowią istotny czynnik wpływający na funkcję i integralność skóry. Wykazano, że ekspozycja na te substancje prowadzi do powstawania reaktywnych form tlenu, aktywacji szlaków zapalnych oraz uszkodzenia bariery naskórkowej. Stres oksydacyjny i przewlekły stan zapalny skutkują zaburzeniem równowagi lipidowej, degradacją kolagenu, wzrostem przeznaskórkowej utraty wody oraz dysregulacją odpowiedzi immunologicznej. Klinicznie manifestuje się to przyspieszonym starzeniem skóry, zaburzeniami pigmentacji oraz nasileniem przebiegu dermatoz zapalnych, takich jak atopowe zapalenie skóry, łuszczyca czy trądzik pospolity. Skóra dysponuje jednak złożonymi mechanizmami obronnymi, obejmującymi system antyoksydacyjny, procesy naprawy bariery lipidowej, regulację immunologiczną oraz udział mikrobiomu w utrzymaniu homeostazy. W warunkach przewlekłej ekspozycji mechanizmy te ulegają stopniowemu osłabieniu, prowadząc do trwałej dysfunkcji bariery skórnej. Zrozumienie molekularnych podstaw tych procesów ma kluczowe znaczenie dla opracowania skutecznych strategii profilaktycznych i terapeutycznych, obejmujących stosowanie antyoksydantów, substancji aktywujących szlak Nrf2 oraz wspierających mikrobiom skóry. Zanieczyszczenia środowiskowe należy uznać za istotny, modyfikowalny czynnik ryzyka chorób skóry, który powinien być uwzględniany w codziennej praktyce dermatologicznej.

Keywords: oxidative stress; epidermal barrier; skin microbiome; environmental pollutants

Słowa kluczowe: stres oksydacyjny; bariera naskórkowa; mikrobiom skóry; zanieczyszczenia środowiskowe

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Corresponding author:
Oliwia Sędziak
Faculty of Medicine, University of Opole
48 Oleska St., 45-052 Opole
e-mail: oliwiaa000@gmail.com

Introduction

As the body's largest organ, the skin is a highly specialized structure that maintains the barrier between the external and internal environment. The epidermal barrier is essential for homeostasis, as it protects against excessive transepidermal water loss, as well as entry of microorganisms, allergens, and toxins [1, 2]. Recent years have seen a dynamic increase in population exposure to environmental pollutants, particularly in urban and industrialized regions. Air pollutants, including particulate matter ($PM_{2.5}$ and PM_{10}), ozone (O_3), nitrogen oxides (NO_x), sulphur dioxide (SO_2), and polycyclic aromatic hydrocarbons (PAHs), not only can damage skin structures, but they can also induce cellular and molecular changes [1–4].

Epidemiological studies indicate that exposure to environmental pollutants is associated with a higher incidence of dermatological conditions, including atopic dermatitis (AD), psoriasis, acne vulgaris, and pigmentary disorders [5]. Furthermore, chronic exposure to PM and ozone accelerates extrinsic skin aging, contributing to increased wrinkle and lentigo formation [4, 5]. Pollutants may also act synergistically with ultraviolet (UV) radiation, exacerbating oxidative stress and DNA damage [6, 7].

Oxidative stress is a key mechanism underlying pollution-related skin damage. Pollutant-induced reactive oxygen species (ROS) and reactive nitrogen species (RNS) oxidize lipids, proteins, and nucleic acids, thereby disrupting the integrity of epidermal barrier [5, 8]. Excessive ROS production activates the nuclear factor kappa B (NF-κB) and mitogen-activated protein kinase (MAPK) signalling pathways, promoting the expression of proinflammatory cytokines and matrix metalloproteinases (MMPs), thereby accelerating collagen degradation and skin aging [9].

The aryl hydrocarbon receptor (AhR) is a key player in the skin's response to environmental pollutants. When activated by ligands such as polycyclic aromatic hydrocarbons (PAHs), it modulates the transcription of proinflammatory and detoxification-related genes. AhR overactivation may contribute to skin barrier dysfunction, impaired keratinocyte differentiation, and hyperpigmentation [10, 11].

Environmental pollutants can also affect cutaneous immune function. Activation of Langerhans cells may enhance antigen presentation and shift immune responses toward Th2- or Th17-dominant profiles, accompanied by increased cytokine production [11, 12]. These changes may promote chronic inflammation, as well as increase epidermal permeability and susceptibility to infections and allergens, thereby triggering or exacerbating inflammatory skin conditions such as AD and psoriasis [5, 13].

A growing body of research indicates that air pollution not only compromises the skin barrier but also alters dermal microbiome by reducing its diversity and promoting the growth of potentially pathogenic species. These changes in microflora may further exacerbate inflammation and impair epidermal regeneration [14].

Understanding the molecular mechanisms underlying pollutant-induced skin barrier damage is crucial for developing effective preventive and therapeutic strategies.

Aim

The aim of this paper was to discuss the effects of environmental pollutants on skin barrier function, with particular emphasis on oxidative stress, inflammatory pathways, and immune response modulation.

Materials and methods

As part of this review, a comprehensive search of the scientific literature was conducted using PubMed, ScienceDirect, and Google Scholar databases. Publications in both Polish and English were considered, using keywords related to environmental pollutants, the epidermal barrier, oxidative stress, and the skin microbiome.

The analysis included peer-reviewed original and review papers looking into the effects of pollutants on epidermal barrier function and underlying molecular mechanisms, including oxidative stress, activation of inflammatory pathways, and immune response modulation. Studies addressing clinical implications, such as skin aging, hyperpigmentation, and exacerbation of dermatoses, were also included, along with *in vitro* and *in vivo* research involving both human skin and skin models. Meta-analyses and systematic reviews providing synthesized evidence were likewise considered.

Non-peer-reviewed publications, popular science or marketing reports, duplicate papers, and experimental studies conducted exclusively in animals without explicit relevance to mechanisms in human skin were excluded.

Selection of publications proceeded in three stages: identification and screening of titles and abstracts, followed by full-text assessment for consistency with the review objectives. Of the 68 publications initially identified, 25 papers met the inclusion criteria after duplicate removal and eligibility screening, and were included as the evidence base for this review.

Data from the selected publications were used to provide a synthesized overview of molecular mechanisms, clinical implications, and potential protective and therapeutic strategies.

Discussion

The physiology of skin barrier

The skin barrier is a complex structure encompassing the stratum corneum, intercellular junctions, lipids, and immune components. Its primary role is to protect the body from water loss as well as physical, chemical, and biological stressors while also modulating immune responses [1]. The stratum corneum consists of corneocytes embedded in a lipid matrix rich in ceramides, cholesterol, and free fatty acids; disruption of the balance among these components may increase transepidermal water loss (TEWL) and skin permeability [1, 2]. Structural

proteins, including filaggrin, loricrin, and involucrin, are essential players in keratinocyte differentiation. Filaggrin is involved in the aggregation of keratin filaments and contributes to the generation of natural moisturizing factors; its deficiency is therefore associated with skin barrier dysfunction and AD. Intercellular junction proteins, such as occludin, claudins, and desmogleins, are likewise critical for maintaining epidermal integrity and limiting pathogen penetration [2].

The skin also serves important immunological functions. The epidermis contains Langerhans cells, which initiate immune responses to pathogens and barrier disruption [1, 2]. Keratinocytes actively produce cytokines, chemokines, and antimicrobial peptides, including defensins and cathelicidin (LL-37), thereby modulating inflammation, whereas the papillary dermis contains dendritic cells, tissue-resident memory T cells, and mast cells, which enhance local immune defences [15]. Skin barrier integrity is closely linked to its microbiome. Commensal species, including *Staphylococcus epidermidis* and *Cutibacterium acnes*, compete with pathogens, support immune system maturation, and help regulate inflammatory responses, whereas microbiome dysbiosis may promote overcolonization by pathogens such as *Staphylococcus aureus*, thereby exacerbating inflammation and skin barrier damage [14, 16].

Skin barrier function is also influenced by physiological factors, including dermal pH, hydration, enzymatic activity, and hormones. A slightly acidic skin surface pH of approximately 4.5–5.5 promotes the activity of enzymes involved in epidermal maturation and inhibits pathogen growth [16], while proteolytic enzymes regulate corneocyte desquamation and skin barrier remodelling, with their dysregulation potentially leading to hyperkeratosis or impaired epidermal structure [2, 6].

In summary, the skin barrier is a dynamic, multicomponent system whose proper function depends on the physical integrity of the stratum corneum, stability of the lipid matrix, structural proteins, intact intercellular junctions, balanced microbiome, and appropriately modulated immune responses. Disruption of any of these components may increase dermal susceptibility to environmental stressors, including air pollution, and may contribute to inflammation and the development of dermatological conditions.

Types of environmental pollutants and their interactions with the skin

Environmental pollutants represent a heterogeneous group of natural and anthropogenic substances that can affect the skin through direct contact or systemic exposure. The most important ones include particulate matter (PM), ozone (O_3), nitrogen oxides (NO_x), sulphur dioxide (SO_2), polycyclic aromatic hydrocarbons (PAHs), heavy metals, and tobacco smoke [2]. $PM_{2.5}$ and PM_{10} are complex mixtures of solid and liquid particles classified based on their aerodynamic diameter. PM_{10} particles predominantly deposit on the skin surface, whereas the smaller $PM_{2.5}$ particles can penetrate through hair follicles and sebaceous glands, reaching the viable layers of the epidermis [16]. These particles may contain heavy metals, sulphates, nitrates, oxidized lipids, and toxic organic compounds able to induce oxidative stress and inflammatory responses [16, 17]. Ozone, a potent oxidant formed through photochemical reactions between nitrogen oxides and volatile organic compounds, primarily affects the lipid layer of the stratum corneum, where it induces lipid peroxidation and generates secondary inflammatory mediators. Ozone does not penetrate deeply into the skin; however, its breakdown products can activate keratinocytes and trigger inflammatory responses [5]. Nitrogen oxides, particularly NO_2, react with epidermal lipids and promote RNS formation, thereby increasing oxidative stress and protein nitrosylation. Additionally, NO_2 may activate proinflammatory signalling pathways, including NF-κB, promoting cytokine expression and extracellular matrix degradation [5, 16].

Exposure of the skin to air pollutants, including $PM_{2.5}$, PM_{10}, O_3, NO_2, and PAHs, promotes ROS and RNS generation. The resulting oxidative stress may lead to the oxidization of lipids, proteins, and DNA, thereby contributing to epidermal damage. Activation of NF-κB and MAPK signalling pathways initiates an inflammatory cascade, increasing the expression of proinflammatory cytokines and matrix metalloproteinases, which may in turn promote the degradation of collagen and other extracellular matrix (ECM) components. Prolonged AhR activation may disrupt keratinocyte differentiation and contribute to hyperpigmentation, while alterations in the skin microbiome can further exacerbate inflammation and accelerate environmentally induced skin aging (Fig. 1).

Sulphur dioxide (SO_2) can irritate the skin, disrupt its acid-base balance, and damage the lipid barrier, with chronic exposure associated with increased skin dryness and pruritus [16]. Polycyclic aromatic hydrocarbons are lipophilic compounds that readily penetrate the skin and bind to the aryl hydrocarbon receptor (AhR), the activation of which induces the expression of detoxification-related genes, including *CYP1A1*, as well as proinflammatory and pro-oxidant genes. Prolonged AhR activation may contribute to hyperpigmentation, impaired keratinocyte differentiation, and carcinogenesis [16, 18]. Heavy metals, including arsenic, cadmium, and lead, can bind to structural proteins in the skin and inhibit antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase, thereby increasing oxidative stress. Arsenic is a known carcinogen and may contribute to skin cancer by causing DNA damage and disrupting genomic repair mechanisms [18].

Tobacco smoke contains more than 4,000 chemical compounds, including PAHs, heavy metals, and aldehydes, all of which cause damage to the epidermal barrier, accelerate skin aging, and promote pigmentary changes; tobacco smoke components may also activate macrophages and mast cells, thereby increasing inflammation [19, 20]. Some pollutants may act synergistically, thereby amplifying their harmful effects. For example, combined exposure to PAHs and UV has been shown to induce greater ROS generation and DNA damage than either factor alone [20]. A similar synergistic effect has been observed between ozone and PM: ozone causes lipid damage, while PM promotes the production of proinflammatory cytokines [5].

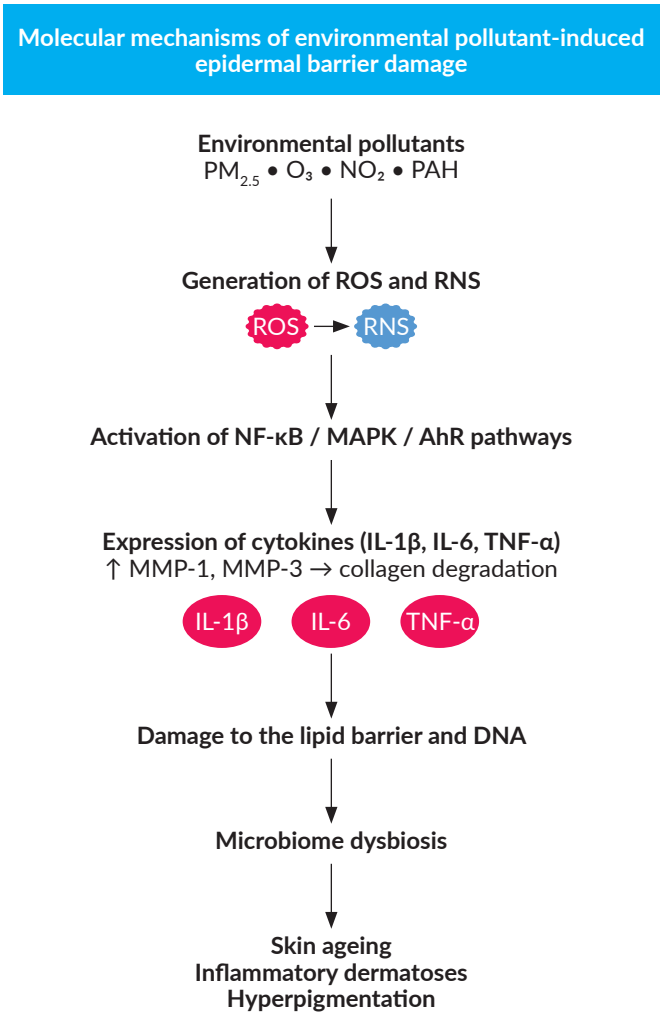

Figure 1. Molecular mechanisms of environmental pollutant-induced epidermal barrier damage

Environmental pollutants may affect the skin both through direct contact, and indirectly, through systemic circulation. PM and PAHs can interact with Toll-like receptors (TLRs) and the aryl hydrocarbon receptor (AhR) on keratinocytes, Langerhans cells, and fibroblasts, activating inflammatory and immune signalling pathways [9, 11]. Additionally, pollutants can alter the skin microbiome by promoting pathogenic colonization, thereby contributing to dysbiosis and chronic inflammation [14].

These findings indicate that, despite their differing chemical properties, environmental pollutants share common effects: they compromise skin barrier function, intensify oxidative stress, promote inflammation, and modulate immune responses, thereby contributing to clinically evident skin changes.

Inflammatory mechanisms and activation of the immune response

Dermal exposure to environmental pollutants triggers a cascade of inflammatory reactions, representing a second major mechanism underlying epidermal barrier dysfunction alongside oxidative stress. This inflammatory process may be initiated through direct activation of pattern-recognition receptors and through secondary oxidative mediators produced during lipid peroxidation [2, 3]. Pollutants such as PM and PAHs can activate Toll-like receptors (TLR2 and TLR4) and AhR, which are expressed on keratinocytes, fibroblasts, and Langerhans cells. TLR activation triggers NF-κB and MAPK signalling pathways, increasing the transcription of genes encoding proinflammatory cytokines. This in turn promotes the local recruitment of neutrophils and monocytes and increases dermal microvascular permeability [11].

AhR activation plays an important role in maintaining cutaneous immune homeostasis; although short-term receptor activation may have protective effects, chronic stimulation by PAHs can promote the overproduction of cytokines and chemokines and disrupt the balance between Th17 and Treg cells, contributing to persistent inflammatory responses observed in conditions such as AD and psoriasis [13]. Ozone and nitrogen oxides can increase local expression of adhesion molecules (ICAM-1 and VCAM-1), on endothelial cells and keratinocytes, thereby promoting leukocyte migration into the skin. Simultaneously, ozone may reduce antioxidant enzyme activity, further amplifying inflammatory responses, while lipid oxidation products (e.g. 4-HNE) can act as ligands for TRPA1 and TLR4, activating neurogenic and immune components of inflammation [5, 11].

Under chronic exposure, inflammatory responses may be sustained by epigenetic mechanisms, including methylation of genes encoding anti-inflammatory mediators and histone acetylation that regulates the expression of proinflammatory cytokines. This may lead to persistent, low-grade subclinical inflammation, causing extracellular matrix damage and accelerated skin aging [9, 21]. Chronic pollutant-induced inflammation can increase the activity of matrix metalloproteinases, which degrade type I and type III collagen in the dermis, contributing to reduced skin elasticity, laxity, and wrinkle formation. Additionally, cytokines such as IL-6 and TNF-α can inhibit collagen synthesis by fibroblasts, further intensifying degenerative changes [9].

Pollution can also affect the skin microbiome – an integral component of innate immunity. PM and ozone may alter commensal flora composition and increase the relative proportion of opportunistic bacteria, such as *Staphylococcus aureus*, thereby impairing immune tolerance and increasing inflammation. Skin dysbiosis may therefore act as a mediator between environmental exposure and the host inflammatory response [2, 6].

In summary, environmental pollutants activate multiple interconnected inflammatory and immunological mechanisms, including stimulation of TLRs and AhR, activation of NF-κB and MAPK signalling pathways, increased expression of proinflammatory cytokines and chemokines, dysregulation of Th17/Treg responses, extracellular-matrix degradation, and disruption of the skin microbiome. These processes contribute to chronic, low-grade inflammation, a key driver of skin barrier dysfunction, loss of epidermal integrity, and the development or exacerbation of environmentally induced dermatoses.

Clinical implications of skin exposure to environmental pollutants

The multifaceted effects of environmental pollutants on the skin are reflected in clinical manifestations such as accelerated skin aging, exacerbation of inflammatory dermatoses, and pigmentary disorders. These symptoms result from chronic oxidative stress, inflammation, and immune dysregulation, with their severity depending on the type and duration of exposure as well as individual susceptibility [2, 11]. Environmental skin aging, which arises from the combined effects of UV radiation, ozone, and PM represents the most extensively documented consequence of cutaneous exposure to air pollution. Epidemiological studies have reported links between higher $PM_{2.5}$ levels and increased wrinkle formation, reduced skin elasticity, and facial hyperpigmentation [4, 16]. Long-term exposure to air pollution can promote collagen fibre degradation by increasing the activity of MMP-1 and MMP-3 while reducing collagen synthesis by fibroblasts. This contributes to skin thinning, reduced firmness, and increased susceptibility to wrinkle formation. The damage may be further exacerbated by the synergistic effects of UV and PAHs [9, 15].

Environmental pollutants may also contribute to the pathogenesis and exacerbation of AD, psoriasis, and acne vulgaris. Upon exposure to $PM_{2.5}$ and NO_2, patients with AD may experience increased epidermal barrier dysfunction due to reduced filaggrin expression and increasing TEWL; these pollutants can also activate Th2 signalling pathways and increase the production of IL-4, IL-13, and IL-31, thereby contributing to pruritus and chronic inflammation [9]. In psoriasis, environmental pollutants can induce oxidative stress and activate the IL-23/Th17 pathway, thereby promoting keratinocyte proliferation and sustained inflammation. In patients with acne vulgaris, pollutants may stimulate sebum production and increase its viscosity, as well as induce inflammatory responses within hair follicles. Ozone and PM may also promote colonization by *Cutibacterium acnes*, contributing to inflammatory lesions [9, 14].

Pollutants, PAHs and ozone in particular, can affect melanocyte metabolism by activating AhR and oxidative pathways. Overproduction of ROSs and increased levels of α-melanocyte-stimulating hormone (α-MSH) may enhance melanogenesis, clinically manifesting as lentigines and uneven facial pigmentation. Population-based studies have reported a relationship between $PM_{2.5}$ exposure and hyperpigmentation, particularly among middle-aged women; additionally, chronic inflammation and epidermal barrier damage may contribute to post-inflammatory hyperpigmentation [19, 22].

Certain pollutant components, including arsenic, cadmium, and PAHs, have carcinogenic potential. Their metabolites can bind to epidermal DNA, causing mutations and impairing genomic repair mechanisms. AhR overactivation and chronic oxidative stress may promote neoplastic transformation and increase the risk of skin cancer, squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) in particular. The combined effects of PAHs and UV radiation can upregulate the formation of pyrimidine dimers, thereby intensifying photocarcinogenic processes [1, 3].

Environmental pollutants can also disrupt the balance of the skin microbiome by reducing the diversity of commensal species and promoting pathogenic colonization. These changes may lower the threshold for cutaneous immune reactivity and contribute to the development of allergic contact dermatitis and environmental urticaria. Furthermore, increased epidermal permeability facilitates the penetration of allergens and irritants, promoting contact hypersensitivity [6, 14].

Epidemiological studies in urban populations have demonstrated clear associations between air pollution levels and the incidence and severity of cutaneous conditions, with the strongest correlations reported between exposure to $PM_{2.5}$, NO_2, and ozone and the severity of AD, psoriasis, acne, and hyperpigmentation. Even short-term exposure to elevated $PM_{2.5}$ levels may cause a transient yet measurable increase in TEWL and a reduction in epidermal antioxidant levels [10, 23].

These findings indicate that environmental pollutants constitute an important risk factor for dermatological disorders. Their effects may be both direct and indirect, mediated through oxidative stress, inflammation, immune modulation, and skin microbiome dysbiosis.

Skin adaptive response and defence mechanisms against environmental pollution

Despite harmful effects of environmental pollutants, the skin has a range of adaptive defence mechanisms that help maintain homeostasis and limit exposure-related damage. These mechanisms involve biochemical and molecular responses, as well as structural and microbiological adaptations.

The skin's extensive antioxidant system constitutes its first line of defence against oxidative stress. It comprises both enzymatic and low molecular weight antioxidants. Key antioxidant enzymes include superoxide dismutase (SOD), catalase, and glutathione peroxidase, which neutralize superoxide anions and hydrogen peroxide, thereby limiting the formation of highly reactive hydroxyl radicals. Non-enzymatic antioxidants, including vitamins E and C, glutathione, uric acid, carotenoids, and coenzyme Q10, are also present in the stratum corneum [11, 24]. The skin's response to chronic exposure to environmental pollutants initially involves compensatory increase in the expression of antioxidant enzymes. This mechanism is associated with activation of the transcription factor Nrf2 (nuclear factor erythroid 2–related factor 2), which interacts with antioxidant response elements (AREs) in the promoter regions of genes encoding cytoprotective proteins, including heme oxygenase-1 (HO-1), NAD(P)H:quinone oxidoreductase 1 (NQO1), and γ-glutamylcysteine synthetase (γ-GCS). The Nrf2/ARE pathway is a central component of the cellular adaptive response to oxidative stress, enabling detoxification of reactive metabolites and the regeneration of glutathione. However, chronic exposure to PM or ozone may deplete these protective mecha-

nisms, resulting in impaired antioxidant defences and persistent epidermal damage [7, 16, 19].

The integrity of the skin barrier also relies on the proper lipid arrangement within the stratum corneum.

In response to irritants and oxidants, keratinocytes initiate repair processes that renew the lipid matrix, with increased expression of ceramide-synthesizing enzymes and lipid transport proteins (ABCA12), further enhancing barrier reconstruction following oxidative stress-induced damage.

This process is modulated by IL-1α and TGF-β, and its effectiveness depends on the metabolic status of epidermal cells and the availability of lipid precursors. It represents an adaptive dermal response that allows for partial restoration of skin barrier function and reduced TEWL once exposure to toxic agents has ceased [15, 20].

The immune system plays a crucial role in the skin's adaptation to environmental exposures. Langerhans cells and keratinocytes exert immunoregulatory effects, among other things, by secreting anti-inflammatory cytokines (IL-10 and TGF-β), as well as by modulating Treg activity, thereby limiting excessive inflammation and maintaining local immune tolerance. In addition to its pro-inflammatory effects, AhR also has some immunomodulatory functions: when activated in a controlled manner, it promotes Treg differentiation and IL-22 production, thereby supporting epidermal barrier regeneration. This phenomenon, which depends on ligand levels and the length of exposure, is often referred to as biphasic AhR response [4, 18].

The skin microbiome functions as a biological barrier by competing with pathogenic microorganisms and modulating local immune responses. In response to environmental pollutants, the commensal skin microbiota may undergo transient adaptive changes, including an increased relative proportion of microorganisms with antioxidant properties (e.g. *Staphylococcus epidermidis* and *Cutibacterium acnes*). These microbes produce metabolites with antioxidant and anti-inflammatory activity, including lactic acid, short-chain fatty acids, and antimicrobial peptides. Maintaining microbiome eubiosis is therefore an important component of the skin's adaptive response to environmental stress [6, 14].

Keratinocytes and fibroblasts can also activate repair mechanisms once pollutant exposure has ceased. These mechanisms include the induction of growth factors (VEGF, EGF, and FGF-2), which promote cell proliferation and differentiation, as well as increase the activity of DNA-repair enzymes (PARP-1, XRCC1), and thus help eliminate oxidative damage and restore normal epidermal cell function. Although these processes are adaptive, their effectiveness declines with age and prolonged exposure to pollution [12, 25].

Together, all the above mentioned mechanisms constitute a complex defence system that protects the skin against environmental pollutants. Their effectiveness determines the ability to maintain cutaneous homeostasis and shapes individual susceptibility to clinically apparent changes. However, chronic exposure can progressively exhaust these protective reserves, ultimately contributing to persistent barrier dysfunction and accelerated skin aging.

Conclusions

Based on the reviewed scientific literature, environmental pollutants are recognized as important risk factors for dermatological disorders. They can induce oxidative stress, inflammatory responses, and epidermal barrier dysfunction. Prolonged exposure may contribute to premature skin aging, pigmentary abnormalities, and exacerbation of inflammatory dermatoses, including atopic dermatitis and psoriasis. The efficacy of skin's antioxidant and immune mechanisms that can partially compensate for pollutant-induced damage may decline as a result of chronic exposure. Therefore, protection against environmental pollutants should be an integral component of dermatological prevention and treatment, particularly in urban settings.

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