

Review Article



Air Pollution and Skin Diseases

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ABSTRACT

Air pollution is a widespread environmental issue, with substantial global implications for human health. Recent epidemiological studies have shown that exposure to air pollution exacerbates various inflammatory skin conditions, including atopic dermatitis, psoriasis, or acne. Furthermore, air pollutants are associated with accelerated skin aging, hair loss, and skin cancer. The aim of this review is to elucidate the current understanding of the impact of air pollution on skin health, emphasizing the underlying mechanisms involved and existing therapeutic and cosmetic interventions available to prevent or mitigate these effects. A pivotal factor in the harmful effects of air pollution is the formation of reactive oxygen species and the resulting oxidative stress. The aryl hydrocarbon receptor signaling pathway also substantially contributes to mediating the effects of air pollutants on various skin conditions. Moreover, air pollutants can disrupt the skin barrier function and trigger inflammation. Consequently, antioxidant and anti-inflammatory therapies, along with treatments designed to restore the skin barrier function, have the potential to mitigate the adverse effects of air pollutants on skin health.

Keywords: Air pollutants; Aryl hydrocarbon receptor; Skin diseases; Oxidative stress

INTRODUCTION

Most of the world's population resides in areas where air quality fails to meet the guidelines for pollutant levels set by the World Health Organization. Air pollution consists of various nano- to micro-sized particles and gaseous components. It consists of particulate matter (PM) of various sizes, polycyclic aromatic hydrocarbons (PAHs), and gaseous components, including ozone (O₃), carbon monoxide (CO), sulfur dioxide (SO₂), nitrogen oxide (NO₂), and volatile organic compounds (VOCs).

PM is a mixture of minute particles or liquid droplets

composed of metals, organic compounds, and dust particles. PM is classified by particle size as PM_{0.1}, PM_{2.5}, and PM₁₀¹. PM_{0.1} and PM_{2.5} typically comprises hydrocarbons, metals, and secondary particles formed by atmospheric gases². In addition, PM can transport considerable amounts of absorbed contaminants, including organic compounds, metals, or PAHs (**Fig. 1**)³⁻⁵.

Studies have indicated a clear correlation between various levels of air pollution and increased risks of mortality, as well as various respiratory or cardiovascular diseases^{6,7}. Although majority of studies on air pollution focus on its influence on the respiratory and cardiovascular systems, prolonged exposure to PM can also

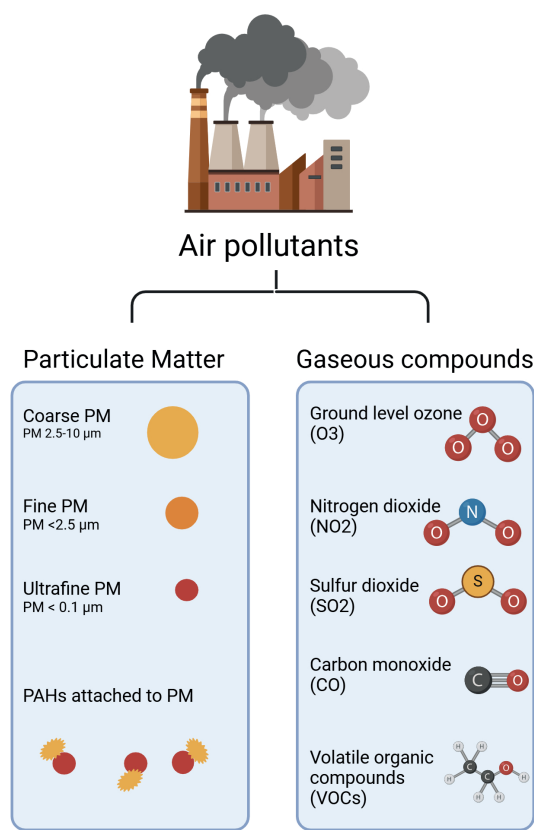


Fig. 1. Classification of air pollutant. Created with BioRender.com.
PM: particulate matter, PAH: polycyclic aromatic hydrocarbon.

substantially affect the skin^{8,13}. Different mechanisms are responsible for skin damage, influenced by the specific pollutants or co-pollutants involved and the duration of exposure¹. Through this review, we aim to consolidate the existing knowledge on the effect of air pollution on skin and the associated physiological mechanisms.

AIR POLLUTANT-INDUCED OXIDATIVE STRESS

The negative effects of air pollution are primarily mediated by the production of reactive oxygen species (ROS)¹⁴. When ROS generation surpasses the skin's antioxidant defenses, it results in oxidative stress, a condition that can impair lipids, proteins, and DNA, thereby, disrupting various cellular organelles and tissue functions (Fig. 2)¹⁴.

Previous studies have demonstrated that PM induces exogenous and endogenous ROS formation through various mechanisms. First, PM contributes to exogenous ROS production owing to its highly reactive surface, which facilitates free radical synthesis^{15,16}. Second, transition metals within PM can generate highly reactive hydroxyl radicals ($\cdot\text{OH}$) by Fenton reactions¹⁷.

Third, soluble redox-active substances (PAHs, quinones) can form ROS and active electrophilic metabolites through bioactivation and redox cycling^{3,18}. Lastly, superoxide ($\text{O}_2^{\cdot-}$) and hydrogen peroxide (H_2O_2) can be produced via intracellular signaling pathways through activation of membrane bound oxides^{3,19}.

In addition, PM and its adsorbed constituents (PAHs and metals) can enhance endogenous ROS generation through endoplasmic reticulum (ER) and mitochondrial stress and enhancing the activity of enzymes involved in ROS production. Topical exposure to PM has can induce mitochondrial and ER stress, indicated by the swelling of these organelles²⁰. Mitochondria, the main source of endogenous ROS in cells, exhibit dysfunction upon PM exposure, characterized by notable structural impairment^{20,21}. This results in mitochondrial ROS generation, increased radical production, and elevated levels of mitochondrial Ca^{2+} levels²⁰. PM exposure can also result in ER stress, as indicated by increased intracellular Ca^{2+} levels²⁰.

EFFECTS OF PM-INDUCED ROS ON SKIN

The detrimental effects of ROS on the skin are well documented, including their capacity to directly damage skin lipids, proteins, and DNA. ROS generated by major air pollutants can facilitate the oxidation of squalene in the skin, resulting in the formation of comedogenic molecules^{22,23}. In addition to squalene, other sebum lipids, including wax esters, cholesterol, triglycerides, or free fatty acids, are similarly vulnerable to ROS peroxidation. Oxidative stress can induce biochemical changes in the sebum and corneal layers. Such reactions are characterized by reduced levels of squalene and vitamin E²⁴. Furthermore, endogenous ROS can react with polyunsaturated fatty acids in cell membranes, which results in the formation of reactive aldehyde byproducts such as malondialdehyde and 4-hydroxy-2-nonenal²⁵. This results in carbonylated proteins formation, and this can induce conformational changes and permanent damage to the polypeptide chain²⁶. Finally, ROS can directly damage DNA, forming DNA adducts²⁷, which leads to apoptosis and cellular senescence²⁸. A previous study has also shown that $\text{PM}_{2.5}$ induces cellular senescence in a ROS-dependent manner²⁹.

ROLES OF THE ARYL HYDROCARBON RECEPTOR (AhR) IN RESPONSE TO AIR POLLUTANTS

The AhR is widely expressed in the skin, and is important in maintaining skin integrity and immune functions³⁰. This chemical

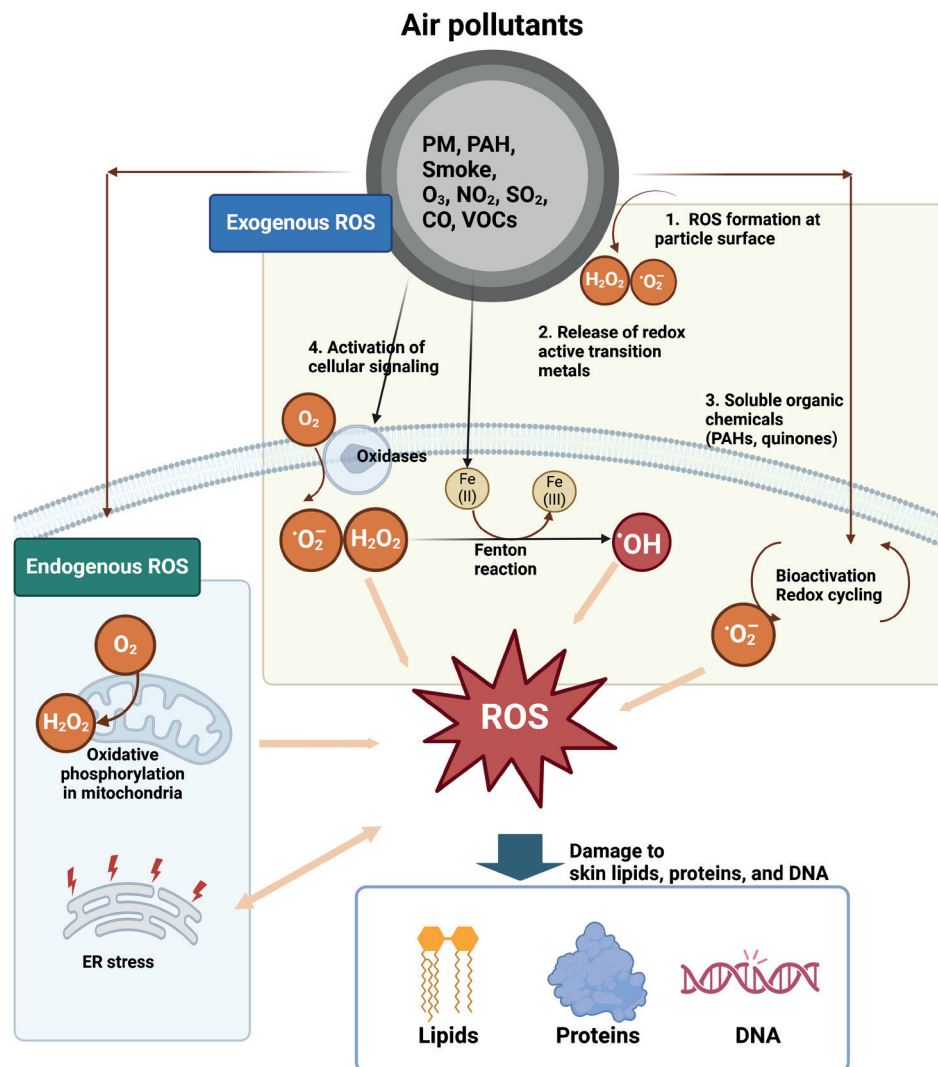


Fig. 2. Air pollutant-induced oxidative stress and its effects on skin. Created with BioRender.com.
PM: particulate matter, PAH: polycyclic aromatic hydrocarbon, VOC: volatile organic compound, ROS: reactive oxygen species.

sensor interacts with various ligands present in the skin is crucial in the skin's physiological responses to pollution. Air pollutants contain various exogenous ligands with high affinities for AhR, including PAHs and halogenated aromatic hydrocarbons such as 2,3,7,8-tetrachlorodibenzodioxin (TCDD), dimethylbenz[a]anthracene, methylcholanthrene, and benzo[a]pyrene (BaP)³⁰. After ligand binding, AhR translocates from the cytoplasm to the nucleus and forms a dimer with the AhR nuclear translocator (ARNT). AhR activation modifies proteins and DNA and is associated with various skin issues, such as immune-mediated skin diseases, cosmetic concerns, and skin cancer (**Fig. 3**)³¹.

Previous studies have demonstrated that AhR activation disrupts skin barrier function, decreases filaggrin levels, and induces symptoms similar to atopic dermatitis (AD)³²⁻³⁵. Pollutant-induced

AhR signaling also triggers inflammatory cascades, amplifying cyclooxygenase-2 (COX-2), tumor necrosis factor- α (TNF- α), interleukin (IL)-1 α , IL-8, and prostaglandin E2³⁶. The AhR activation promotes IL-8 production in human epidermal keratinocytes exposed to cigarette smoke³⁷, correlating with findings that smoking adversely affects acne and palmoplantar pustulosis^{38,39}. Moreover, AhR influences growth factor modulation, such as epidermal growth factor, and PAH-induced AhR activation reduced cell proliferation and induced apoptosis in keratinocytes and fibroblasts⁴⁰. Finally, AhR regulate the differentiation of T helper 17 (Th17) and regulatory T cell (Treg) cells. PM_{2.5} exposure has been shown to facilitate Th17 polarization and activation through an AhR-dependent pathway, thereby modulating T-cell activity and exacerbating Th17-related diseases⁴¹.

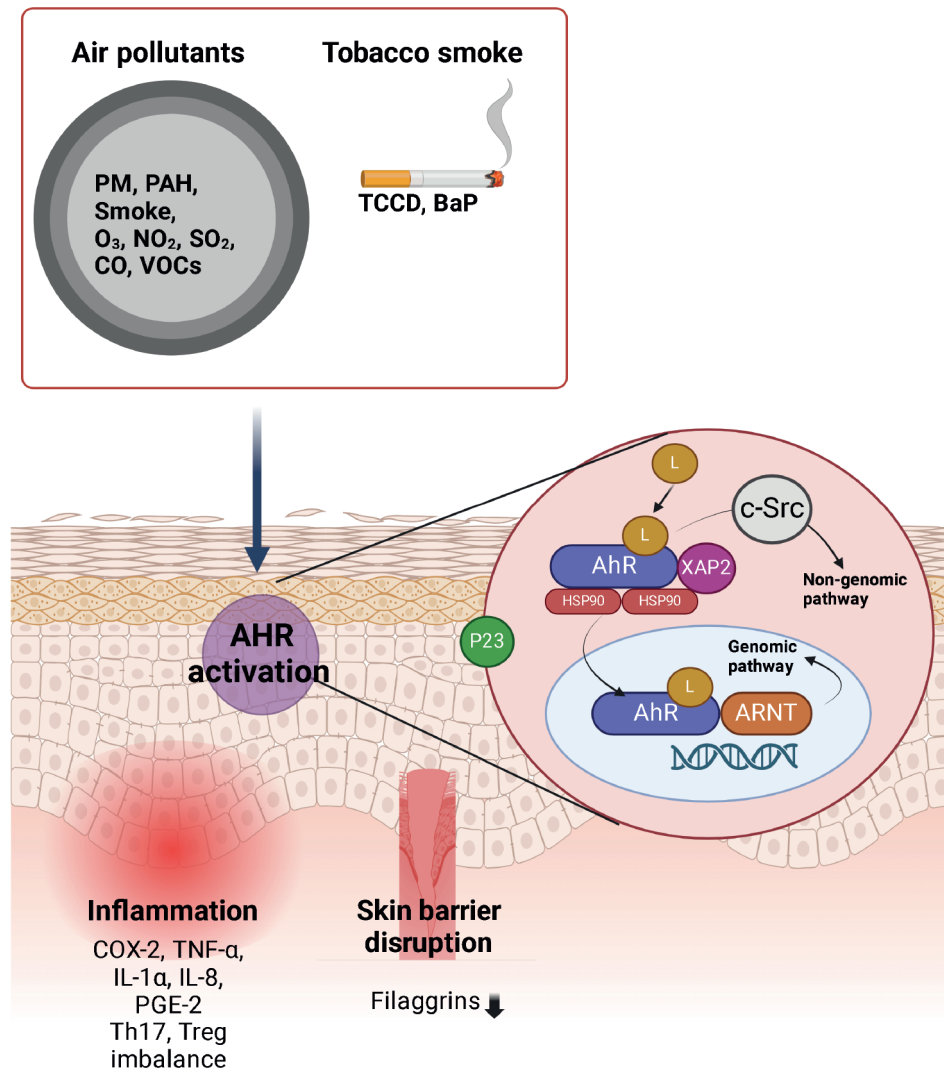


Fig. 3. Roles of the aryl hydrocarbon receptor in response to air pollutants. Created with BioRender.com.

PM: particulate matter, PAH: polycyclic aromatic hydrocarbon, VOC: volatile organic compound, TCCD: 2,3,7,8-tetrachlorodibenzodioxin, BaP: benzo[a]pyrene, AhR: aryl hydrocarbon receptor, ARNT: aryl hydrocarbon receptor nuclear translocator, COX-2: cyclooxygenase-2, TNF-α: tumor necrosis factor-α, IL: interleukin, PGE-2: prostaglandin E2, Th17: T helper 17, Treg: regulatory T cell.

IMPACTS OF AIR POLLUTANTS ON SKIN BARRIER AND IMMUNITY

Air pollutants can interfere with the integrity of the skin barrier and initiate inflammation in human skin⁴². These pollutants disrupt both the physical and chemical barriers of the skin. Various studies have revealed that PM exposure decreases the expression of key proteins, such as filaggrin, cytokeratin 10, involucrin, and loricrin, which are crucial in maintaining skin structure⁴³⁻⁴⁵. Additionally, PM modulates tight junction proteins. PM induces the degradation of Zonula Occludens-1, leading to increased endothelial barrier permeability⁴⁶⁻⁴⁸. This disruption of physical skin

barrier leads to enhanced skin permeability and altered skin morphology⁴⁹⁻⁵¹. PM also affects skin chemical barriers, including antimicrobial peptides (Fig. 4).

Air pollutants can also stimulate inflammatory reaction in human skin⁴². For instance, PAHs can activate Toll-like receptors (TLRs), activating intracellular pro-inflammatory pathways (nuclear factor-kappa B [NF-κB] and mitogen-activated protein kinase [MAPK])⁵². PM_{2.5} can bind to TLR5, activating NF-κB, which upregulates the expression of IL-1α, IL-1β, IL-6, IL-8, intercellular adhesion molecule 1, COX-2, and TNF-α^{44,53-64}. NF-κB activation also promotes the transcription of the nod-like receptor family pyrin domain, initiating inflammasome activation⁵³.

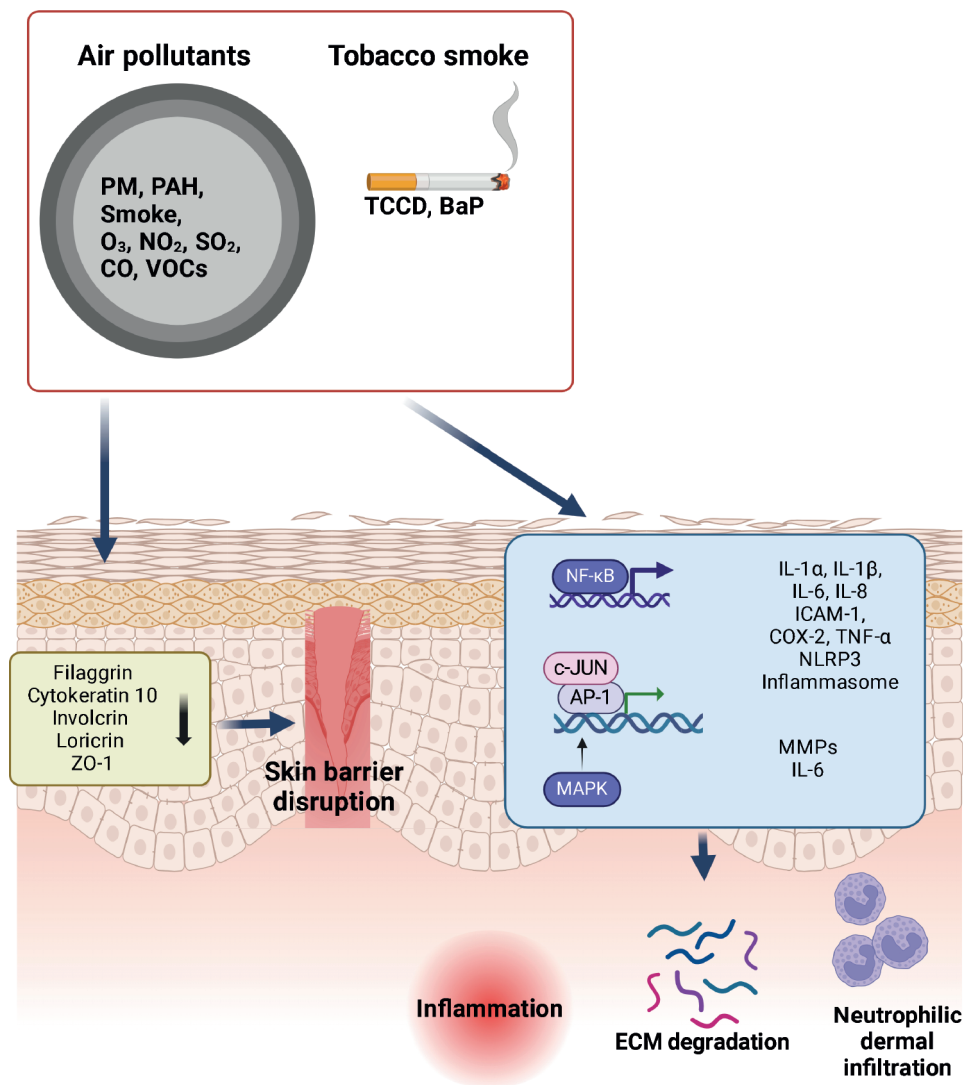


Fig. 4. Impacts of air pollutants on skin barrier and immunity. Created with BioRender.com.

PM: particulate matter, PAH: polycyclic aromatic hydrocarbon, VOC: volatile organic compound, TCCD: 2,3,7,8-tetrachlorodibenzodioxin, BaP: benzo[a]pyrene, AhR: aryl hydrocarbon receptor, IL: interleukin, ICAM: intercellular adhesion molecule, COX-2: cyclooxygenase-2, TNF-α: tumor necrosis factor-α, NLRP3: NLR family pyrin domain containing 3, MMP: matrix metalloproteinase, ECM: extracellular matrix.

Additionally, fibroblasts and keratinocytes exposed to PM can activate the MAPK pathway through phosphorylation of extracellular signal-regulated kinase, c-Jun N-terminal kinase, and p38^{35,40,61,65-69}. MAPK activation induces the activation of activator protein 1 (AP-1) and the transcriptional regulation of matrix metalloproteinases (MMPs), which is also linked to skin aging⁷⁰. Numerous studies have reported elevated levels of MMPs in response to PM exposure^{56,7176}. Furthermore, pollutant-induced ROS and activation of AhR amplify these inflammatory pathways^{37,68,77-80}.

Clinically, PM exposure leads to epidermal thickening and dermal inflammatory cell infiltration⁷³. Prolonged PM_{2.5} exposure is

related to severe inflammation, characterized by extensive neutrophil infiltration, which can exacerbate inflammatory skin conditions⁸¹. These findings imply a potential relationship between PM exposure and AD⁷³.

AIR POLLUTANT-RELATED SKIN DISEASES

Skin aging

Skin aging results from 2 overlapping mechanisms, intrinsic and extrinsic aging. Intrinsic aging is chronological aging, which

naturally affects all body tissues and organs over time, and is influenced by genetic factors. In contrast, extrinsic aging is driven by environmental factors, known as skin exposomes, such as sunlight, air pollution, cigarette smoke, and diet. This type of aging manifests as coarse wrinkles, solar elastosis, and pigmentary disorders⁸². Among these exposomes, sunlight is the most recognized, leading to the concept of photoaging. However, recent research has also identified both outdoor and indoor air pollution as underrated environmental risk factors for extrinsic skin aging (Fig. 5)⁸³.

Studies suggest strong correlations between air pollution and skin aging, particularly in pigmentary changes and wrinkles⁸⁴. A cohort study supported that PM exposure was associated with skin aging, as measured by the SCIENXA score, with heightened facial pigment spots by over 20% in areas with high PM concentrations⁸³. In another study from Taiwan, PM₁₀ and PM_{2.5} were

associated with pigmentation, particularly in individuals over 45 years of age. Similarly, a study from China found that women exposed to higher levels of PM_{2.5} showed more age-associated pigmentation (senile lentigines) on the face and hands⁸⁵. PM_{2.5} levels have also been found to be associated with increased wrinkles⁸⁶. Another study from China proved that indoor cooking with solid fuels increased wrinkles on the hands and cheeks⁸⁷. Additionally, in a study performed among the Taizhou cohort from China, PM_{2.5} was correlated with pigmentation on the forehead and wrinkles on the upper lips.

Air pollutants contain diverse compounds, each contributing differently to skin aging⁸⁴. For instance, exposure to PM_{2.5} adversely affects tight junctions, keratins, and filaggrins, resulting in elevated skin permeability and signs of premature aging⁸⁴. PM-induced ROS also heightens the expression and activity of MMP, specifically MMP-1 and MMP-3, expediting skin aging by

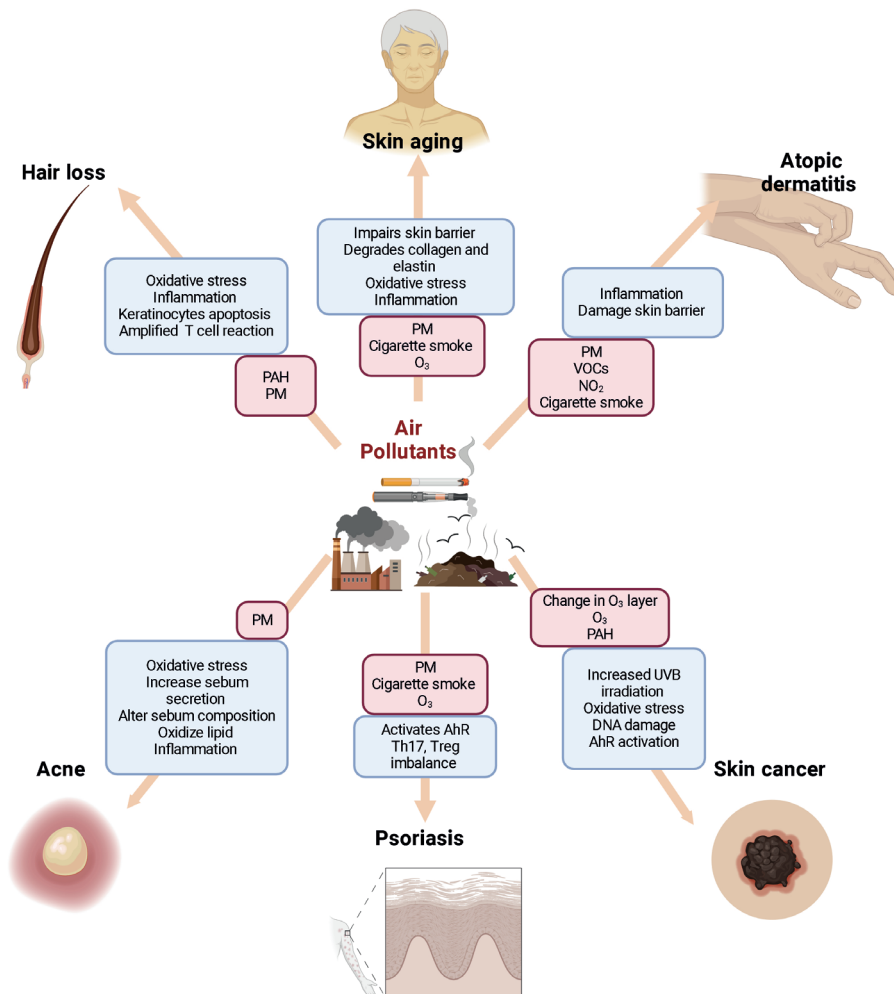


Fig. 5. Effects of air pollutants on various skin disorders and their potential mechanisms. Created with BioRender.com.

PM: particulate matter, PAH: polycyclic aromatic hydrocarbon, VOC: volatile organic compound, AhR: aryl hydrocarbon receptor, Th17: T helper 17, Treg: regulatory T cell, UVB: ultraviolet B.

degrading collagen and elastin. Previous research has also indicated that PM triggers IL-1 α and IL-1 β expression in keratinocytes via the p38 MAPK pathway. These ILs upregulate the expression of MMP-1 and COX-2 in human dermal fibroblasts, contributing to dermal collagen disruption and wrinkle formation⁶⁸. Moreover, exposure to PM elevates transforming growth factor- β levels in the skin and downregulates the synthesis of collagen type 1 α chain (COL1A1, COL1A2), as well as elastin by fibroblasts. These changes contribute to wrinkle formation^{84,88}. PM_{2.5} is also associated with cellular senescence relating to AhR-induced ROS generation. Scavenging ROS mitigates PM_{2.5}-mediated cellular senescence by regulating DNA and histone methylation⁸⁸. Moreover, PM particles are capable of carrying organic chemicals, metals, and PAH that can penetrate the skin and accelerate aging. Specifically, PAHs activate AhR in melanocytes and keratinocytes and synergistically interact with ultraviolet A (UVA), inducing skin pigmentation⁷⁶. AhR signaling in melanocytes and keratinocytes is important for skin pigmentation as evidenced by an AhR-deficient mice where there was a significantly weaker pigmentation compared with that in the wild-type mice.

Cigarette smoking is another well-known factor contributing to accelerated aging that causes deep wrinkles, skin dryness, sagging, and premature graying. It induces oxidative stress, resulting in elevated trans-epidermal water loss (TEWL), lipid peroxidation, cell death, and connective tissue degradation through MMP-1 and MMP-3⁷⁶. Similar to PM, cigarette smoke interacts synergistically with UV radiation, leading to disruptions in the epidermal barrier, increased erythema, and reduced skin elasticity⁸⁹. In addition to the topical effects, inhalation of cigarette smoke has systemic impacts. Long-term exposure alters the composition and distribution of elastin and microfibrils in the dermis, increasing skin stiffness⁹⁰.

O₃ also interacts with the skin and contributes to aging. A study involving 2 German cohorts of older adults assessed the impact of 5-year exposure to tropospheric O₃ on skin aging, revealing a positive correlation between coarse wrinkles and O₃ exposure⁹¹. O₃ induces oxidative stress, promotes lipid peroxidation, activates AhR, and depletes skin levels of vitamins C and E^{92,93}. Additionally, O₃ exhibits a synergistic interaction with UVA, amplifying oxidative stress in the skin and promoting nuclear factor erythroid 2-related factor 2 expression in keratinocytes, which leads to skin inflammation, wrinkle formation, and pigmentation⁹⁴. Research has also demonstrated that O₃ activates NF- κ B, increasing MMP-9 and COX-2 whereas, downregulates collagen-1 and collagen-3 in the dermis, contributing to the development of wrinkles and senile lentigines^{8,95}.

Finally, a cross-sectional study from China evaluated the impact of indoor air pollution on skin aging and found that cooking with solid fuels was associated with skin aging, with more pronounced wrinkles on both the face and hands, highlighting the role of indoor air pollution in the skin aging process⁸⁷.

AD

AD is characterized by eczema and pruritus, typically manifesting in early life stages, including infancy and childhood⁹⁶. The pathophysiology of AD involves compromised epidermal barrier integrity and immune system imbalance⁹⁶. Globally, the incidence of AD is rising⁹⁶. Although the correlation between air pollution and AD, particularly in highly polluted areas, has been a subject of investigation. The relationship remains uncertain⁹⁷. This uncertainty may be attributed to various factors, including environmental interactions that increase susceptibility to allergy-related diseases following exposure to air pollutants. However, research has shown that levels of PM_{2.5}, PM₁₀, toluene, and VOCs are higher on days when individuals with AD report symptoms compared to days without symptoms⁹⁸. Furthermore, evidence suggests that exposure to PM_{2.5}, PM₁₀, and NO₂ in the first trimester, is linked to an increased likelihood of AD^{99,100}. Moreover, both active and passive smoking have been positively correlated with AD prevalence in adults^{101,102}.

Air pollutants may promote AD by impairing the skin barrier, which increases allergen sensitization and causes irritations¹⁰³. Studies have suggested that formaldehyde, VOCs, and NO₂ may impair the skin barrier in individuals with AD by increasing the TEWL^{104,105}. Elevated TEWL, which suggests compromised skin barrier functionality, could increase the sensitivity of individuals to airborne allergens^{106,107}. Moreover, a compromised epidermal barrier facilitates PM infiltration. Prolonged and frequent exposure to PM could induce skin irritation, resulting in epidermal thickening and dermal inflammation⁷³.

Additionally, air pollutants have been demonstrated to increase cytokine levels including IL-1 α , IL-1 β , TNF- α , and IL-8, which are linked to elevated AD risk⁶². They are also essential in the onset and exacerbation of AD lesions and symptoms by activating AhR signaling pathways. These pathways trigger oxidative stress and initiate inflammation, further impairing the barrier function^{108,109}. Elevated AhR expression has been observed in AD¹¹⁰. Furthermore, air pollution may alter the skin microbiome, potentially influencing both the incidence and severity of AD¹¹¹. Finally, environmental conditions such as humidity and temperatures can amplify the harmful effects of various atmospheric particles and gaseous air pollutants on AD¹¹².

Acne

The pathogenesis of acne involves a complex interplay of factors, including hormonal imbalance, genetic predisposition, and environmental factors, resulting in comedone formation and subsequent inflammation¹¹³. Research has highlighted a association between air pollution and acne exacerbation. For instance, a time-series study in Beijing found that elevated levels of ambient PM and NO₂ were related to an increase in acne cases¹¹⁴. Similarly,

Liu et al.¹¹⁵ reported that higher levels of these pollutants were linked to more frequent daily visits for acne vulgaris. The current literature suggests that ambient air pollution may aggravate acne by altering lipid composition or biophysical functions of the skin and triggering inflammation.

First, PM can alter skin function by modifying its lipids, DNA, and/or proteins through oxidative stress¹¹⁴, leading to lipid oxidation in the stratum corneum, characterized by elevated levels of oxidized squalene and diminished linoleic acid concentrations²³. Additionally, studies have indicated that air pollution promotes sebum excretion, decreases vitamin E and squalene in the sebum, and elevates both lactic acid and erythema index in facial skin²⁴.

Additionally, ROS and AhR activation are thought to be implicated in inflammatory response induction^{116,117}. Previous research examining the impact of PM exposure on acne-like inflammation using *Cutibacterium acnes* and *Staphylococcus aureus* peptidoglycan-treated human epidermal keratinocytes found elevated levels of proinflammatory cytokines, COX-2, TLR4, and phosphorylated NF- κ B. These findings suggest that PM exposure could exacerbate acne by promoting inflammatory responses¹¹⁸.

Beyond PM, cigarette smoke can also be the pro-inflammatory cytokine IL-1 α and to oxidize comedones in patients with acne¹¹⁹. Similarly, PAHs, including BaP, promote acneiform eruptions by enhancing IL-8 secretion, a cytokine highly expressed in inflammatory acne lesions^{37,120}. Moreover, TCDD, a potent lipophilic compound derived from PAHs, has been associated with chlor-acne, categorized as an occupational dermatosis¹²⁰.

Psoriasis

Psoriasis is a skin disorder with systemic inflammation in which IL-17 and Th17 cells play essential roles in disease pathophysiology^{121,122}. Research suggests that cigarette smoke may exacerbate psoriasis severity in a dose-dependently¹²³. Additionally, a recent study found a correlation between air pollution exposure and increased psoriasis activity, along with a higher likelihood of flares¹²⁴.

The prevailing theory holds that the activation of AhR by PM and O₃ could potentially impact Th17 cell differentiation^{41,125}. For instance, diesel exhaust, an important source of PM, interacts directly with AhR, facilitating the rapid differentiation of Th17 cells⁴¹. AhR, which regulates Th17 differentiation and Treg cells, can induce Th17 polarization and activation in response to PM_{2.5}, thus modulating T-cell activity and exacerbating Th17-related diseases⁴¹. Furthermore, the co-localization of AhR with its nuclear transporter, ARNT, in the lower epidermal layers of acute psoriatic lesions supports the involvement of AhR pathways¹¹⁰. Therefore, air pollutants may play a significant role in Th17 differentiation through AhR, which is considered pivotal in psoriasis development.

Hair loss

Hair is thought to be a reliable indicator for assessing exposure to environmental pollutants¹²⁶. Studies analyzing mercury, lead, heavy metals, and PM_{2.5} levels in hair samples from people in mining areas have provided insights into how air pollutants affect hair health^{127,128}. Air pollutants can accumulate on the scalp and hair, potentially penetrating deeper skin layers through mechanisms such as transcutaneous absorption or via hair follicles⁸³. Research shows that particles <1.5 μ m in size can enter hair follicles¹²⁹.

PAHs, often found on PM surfaces, can stimulate ROS production and trigger inflammatory skin responses, potentially leading to hair loss¹³. Tobacco smoke, which contains PAHs, exhibits oxidative properties and has been linked to early-onset androgenetic alopecia¹³⁰. Additionally, another study suggests that PAHs accelerate the ultrastructural degradation of hair fibers, altering hair quality and surface properties¹³¹. In experimental studies, cigarette smoke exposure to mice led to alopecia, graying hair, reduced subcutaneous tissue thickness, decreased hair follicles, and damage to hair bulb cells in affected areas¹³².

Additional data suggest that PM can lead to keratinocytes apoptosis within hair follicles, potentially inhibiting hair growth¹³³. A recent study linked flare-ups in alopecia areata with increased PM₁₀ concentrations¹³⁴. Evidence suggests that PM could elicit an amplified reaction from T cells by inducing oxidative stress within hair follicles.

Skin cancer

A correlation has been observed between rising cancer incidence and the presence of air pollutants¹³⁵. Pollutants (PM, PAHs, O₃, VOCs, and heavy metals) have shown to promote skin tumor formation. Evidence suggests that PM₁₀ exposure can increase skin cancer risk¹³⁶. A 10 μ g/m³ increase in PM₁₀ was correlated with increased relative risk of non-melanoma skin cancer up to 52%¹³⁵. PAHs also significantly contribute to the development of skin cancer, likely related to the increased UV irradiation resulting from O₃ layer depletion, combined with the synergistic effects of these air pollutants with carcinogenic potential¹³⁵.

First, air pollution-driven changes in the O₃ layer have led to more UV irradiation reaching the earth's surface, thereby increasing skin cancer risk¹³⁷. A 1% decrease in the O₃ layer thickness is estimated to correspond with a 2% rise in UVB irradiation-associated melanoma cases¹³⁸. Similarly, this level of O₃ layer reduction is associated with a 3%–4% increase in squamous cell carcinoma (SqCC) incidence¹³⁹. In contrast, elevated levels of O₃ in the human environment can also induce oxidative stress. This leads to inflammation and compromising the epidermal barrier integrity, thereby increasing cancer susceptibility¹⁴⁰.

Second, air pollutants directly contribute to tumor formation by binding to cellular DNA through PAH-derived epoxides

and diols¹⁴¹. Furthermore, the activation of the AhR and subsequent promotion of downstream signals owing to chronic skin exposure to PAHs and UV radiation can contribute to skin cancer development¹⁴². Oxidative stress and genotoxicity caused by these pollutants may be facilitated through interactions with AhR¹⁴³. Furthermore, studies have shown that animals lacking Langerhans cells (LCs) are less prone to cancer. LCs mediate the metabolic conversion of PAHs to a prooncogenic intermediate, elevating mutagenesis rate and increasing DNA damage in the epidermis, which ultimately contributes to SqCC development¹⁴⁴.

CURRENT THERAPEUTIC AND COSMETIC INTERVENTIONS

The impact of air pollution-associated skin diseases is expected to increase in the coming years. Although advocating for cleaner air, renewable energy, and environmental protection remains the primary solution, individual protection against the harmful effects of pollution is equally important. This underlines the critical need for integrative research on the mechanisms on how air pollution

affects the skin, with the goal of formulating effective preventive and therapeutic approaches for susceptible individuals.

PM has been detected within hair follicles, even on skin with an intact barrier⁷³. As mentioned, particles <1.5 µm in diameter can enter into hair follicles and, through this transfollicular pathway, reach viable cells in deeper skin layers¹²⁹. Studies have also indicated that certain air pollutants can be dermally absorbed, with dermal absorption rates often matching or surpassing those of inhalation¹⁴⁵. Additionally, pollutants may also reach the skin through the circulatory system. Therefore, protective measures should address both external and internal exposure pathways^{146,147}. **Table 1**^{63,65,67-69,76,148-163} summarizes the current therapeutic interventions to prevent or mitigate air pollution effects on the skin.

CONCLUSION

Air pollution is a notable global issue that contributes to and worsens various skin diseases. We conducted extensive research to elucidate the mechanisms by which air pollutants, particularly PM, affect the skin and to identify therapeutic or preventive strategies

Table 1. Summary of current therapeutic and cosmetic interventions to prevent or mitigate the effects of air pollutants on skin

Dietary antioxidants	- Antioxidant supplements: vitamins B3, C, and E; omega-3; β-carotene; selenium; coenzyme Q10; polypodium leucotomos; green tea; and lipoic acid¹⁴⁸ - Marine-derived compounds^{67,149} - Epigallocatechin gallate from green tea⁶⁵ - Fermented fish oil from mackerel⁶³ - 7,3',4'-trihydroxyisoflavone from soybeans¹⁵⁰ - Mediterranean diet: fruits, vegetables, whole grains, legumes, olive oil, fish, and poultry¹⁵¹
Topical antioxidants	
Vitamin C	- Reduces free radical damage, increases collagen synthesis, and decreases pigmentation¹⁵² - Pre-treatment with vitamin C compound mixture prevents O₃-induced oxidative damage in human keratinocytes¹⁵³
Niacinamide (Vitamin B3)	- Helps repair damaged DNA - Niacinamide effectively inhibits PM_{2.5}-induced ROS generation and also impedes PM_{2.5}-induced oxidation of lipids, proteins, and DNA⁶⁹
Phenolic compounds	- Attenuate cellular oxidative stress induced by PM - Quercetin and revarastrol¹⁵⁴
Phytosterols	- Afzelin attenuates inflammation through MAPKs/AP-1 signaling pathways¹⁵⁵ - Reduce ROS generation, inflammation, and PM-induced cellular damage^{152,156} - Fucosterol (phytosterol from brown seaweed) attenuates the harmful effects in both keratinocytes and fibroblasts caused by PM⁷⁶
Saponin compounds	- Ginsenoside Rb1 counteracts ultraviolet B radiation-induced oxidative stress in the skin, inhibits inflammation, and expedites wound healing¹⁵⁷⁻¹⁶⁰ - Ginsenoside Rb1 protects against PM_{2.5}-induced skin damage by inhibiting mitochondria and endoplasmic reticulum stress-dependent apoptosis¹⁶¹
Others	
E/Z-2-benzylidene-5,6-dimethoxy-3,3-dimethyl-indan-1-one	- AhR antagonist - Inhibits AhR activation in human skin exposed to air pollution - Suppresses the activation of genes associated with wrinkle formation and carcinogenesis (CYP1, COX-2, MMP-)¹⁵¹
Negative air ions	- Downregulate both mRNA and protein levels of pro-inflammatory cytokines induced by PM¹⁶² - Reduce intracellular ROS - Inhibit p38 MAPK activation - Deactivate activator protein 1 (c-FOS and c-JUN)⁶⁸
Tapinarof	- Topical therapeutic AhR modulator¹⁶³

PM: particulate matter, ROS: reactive oxygen species, MAPK: mitogen-activated protein kinase, AP-1: activator protein 1, AhR: aryl hydrocarbon receptor, COX-2: cyclooxygenase-2, MMP: matrix metalloproteinase.

to mitigate air pollution-induced skin damage. Recent studies have assessed the molecular mechanisms that highlight the central role of oxidative stress. Further research is required to develop safe and effective therapeutic and preventive interventions.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

DATA SHARING STATEMENT

Data available on request due to privacy/ethical restrictions.

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