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The Association Between Air Pollutants and Psoriasis and Atopic Dermatitis: Epidemiological and Mechanistic Evidence

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ABSTRACT

Air pollution is a major global public health concern that affects the majority of the world's population. The effects of air pollution on the respiratory and cardiovascular systems have continuously been studied, but the effect of air pollution on the skin, and specifically inflammatory skin diseases is an area that's researched, yet needs more public awareness. The skin is the first barrier that protects us from the outside world, and this literature review examines how air pollution can penetrate this barrier and lead to two common inflammatory skin disorders, which are psoriasis and atopic dermatitis (eczema). This review was conducted by searching PubMed between January and March of 2026 using combinations of the terms, “psoriasis”, “atopic dermatitis”, “eczema”, “oxidative stress”, and “particulate matter”. Studies published within the past 10 years were prioritized and only English-language publications were used. Studies were excluded if they did not involve human subjects, or if pollution exposure was not a primary variable. Common air pollutants like particulate matter (PM), polycyclic aromatic hydrocarbons (PAHs), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3) can lead to oxidative stress if they enter the skin, which can lead to skin barrier damage and inflammatory responses. Epidemiological studies from regions with high pollution levels such as China, South Korea, and Europe demonstrate a strong link between exposure to pollutants and an increased amount of clinical visits to examine flares of these two skin diseases. Lastly, genetic susceptibility appears to play a role as well, and can increase the likelihood of flares developing. Throughout this review, findings are described as associations unless mechanistic evidence supports a causal pathway. As global air pollution levels continue to rise, recognizing its role as an environmental trigger for inflammatory skin diseases is essential to protect skin health and create effective treatments.

Keywords: Air pollution, psoriasis, eczema, atopic dermatitis, particulate matter, genetic susceptibility

INTRODUCTION

Air pollution is a major global public health issue that affects billions of people worldwide. The World Health Organization (WHO) defines air pollution as a contamination of the indoor or outdoor environment by a chemical, physical or biological agent that modifies the natural characteristics of the

atmosphere. This pollution can be created by household combustion devices, motor vehicles, forest fires, and industrial facilities (1).

WHO outlines four major air pollutants, which are particulate matter (PM), ground-level ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2) (1). In addition to the four major air pollutants that WHO outlines, the Clean Air Act also notes that carbon monoxide (CO) should be included in this category. Particulate matter (PM) is a major harmful air pollutant that is composed of a mixture of solid and liquid particles that vary in size (2). PM can be divided into two categories; fine, where particles are less than $PM_{2.5}$, and coarse, where particles have diameters between 2.5 and 10μm. Another type of air pollutant are polycyclic aromatic hydrocarbons (PAHs), and they often cling to $PM_{2.5}$ (3). CO is a colourless, odourless gas that is produced by the incomplete combustion of wood petrol, charcoal, and kerosene. Second, O_3 is actually at ground level and is formed through the reaction with gases in the presence of sunlight. NO_2 is a gas that is commonly released from the combustion of fuels from transportation and industries. Lastly, SO_2 is a colourless gas with a sharp odour and it is produced from the burning of fossil fuels (4).

WHO data shows that 99% of the global population breathes air that contains high levels of pollutants, and low and middle income countries suffer from the highest amounts of exposure. Ambient (outdoor) air pollution in cities and rural areas was estimated to cause 4.2 million premature deaths worldwide per year in 2019, and air pollution also leads to 147 million years of healthy life lost annually (5). Air pollution contributes to more deaths and reduced life expectancy particularly in highly industrialized and urban regions. While air pollution is most commonly associated with respiratory diseases, research in the past few decades has shown that it can have significant effects on the skin as well.

The skin acts as a protective barrier between the body and its external environment, and can be affected by common pollutants like NO_2 and SO_2 , which are capable of interacting with the skin surface and penetrating it through hair follicles or sweat glands due to their small size. PM, O_3, NO_2, and SO_2 are capable of triggering inflammatory skin diseases such as psoriasis and eczema.

25% of the individuals that suffer from skin diseases worldwide have common forms of inflammatory skin diseases such as psoriasis and atopic dermatitis. Research indicates that there is a strong genetic predisposition to both psoriasis and eczema. However, exposure to environmental factors such as pollution can worsen or trigger both conditions (5).

An estimated 60 million people have psoriasis worldwide (6). Psoriasis can be diagnosed by examining various symptoms, such as skin redness, discomfort, itching, the formation of thick scaling plaques, pain, soreness and bleeding. Not only are these symptoms a physical burden for affected individuals, but this burden may be even more significant if the patient has depression, anxiety, or stress. This can potentially lead to disfigurement and even impaired labor capacity in individuals that are greatly affected by this disease (7). In recent years there have been studies that show the association between long-term and short-term air pollution exposure and how this impacts the prevalence of psoriasis. These studies have been carried out in China (8), South Korea (9), and Italy (10) . These studies aim to investigate the

association between pollutant exposure and psoriasis outcomes, often using large population datasets to assess both short-term and long-term exposure effects.

The most common form of eczema is atopic dermatitis, which affects more than 200 million people worldwide. It is reported that 1-3% of adults and 15-20% of children are impacted by this condition (7). This can diminish the quality of life, cause large costs for patients, and alter social functioning. Symptoms include skin redness, swelling, scaling, and dryness. Atopic dermatitis often affects certain areas of the body, including skin folds, the face and neck, hands and wrists, as well as feet and ankles. There are topical anti-inflammatory ointments and creams that can be used to treat this condition for mild or moderate symptoms. Factors such as low humidity, living in urban areas, and genetic predisposition are all associated with the development of eczema; however, this review focuses on the role of air pollution in the development or triggering of eczema (11).

In this literature review, I will examine how different types of air pollution such as PM, NO_2, O_3, and PAHs contribute to the intensification of inflammatory skin diseases such as psoriasis and atopic dermatitis, or trigger flare-ups. Additionally, this paper will explore the mechanisms through which pollutants enter the skin barrier, induce oxidative stress, and activate immune pathways that can lead to these two skin conditions.

Despite a growing amount of research linking air pollution to inflammatory skin diseases, much of the existing literature focuses on either epidemiological associations or isolated biological mechanisms, while this review integrates both. Additionally, while respiratory and cardiovascular effects of air pollution are well established, its impact on skin health is comparatively less explored and recognized. This gap is particularly important given that the skin is the body's primary barrier to the environment and the growing levels of air pollution. This literature review aims to analyze current epidemiological and mechanistic evidence to provide a deeper understanding of how air pollutants are associated with the development and intensification of psoriasis and atopic dermatitis. By examining both population-level trends and underlying cellular pathways that may contribute to them, this paper highlights air pollution as a significant, yet often overlooked environmental factor.

This literature review was conducted using peer-reviewed articles obtained from databases such as PubMed, as well as reputable public health sources such as the World Health Organization (WHO). Common search terms included combinations of keywords such as "air pollution", "psoriasis", "atopic dermatitis", "eczema", "skin barrier", "oxidative stress", "polycyclic aromatic hydrocarbons", and "particulate matter". The included studies were published within the past 10 years, however a few earlier studies were included to provide foundational context for key biological mechanisms.

PSORIASIS

Air pollution has been increasingly recognized as a possible environmental factor that may lead to the development or exacerbation of psoriasis. This section will outline how pollutants such as PM, NO_2, SO_2

and PAH can interact with the skin and immune systems in ways that promote inflammation by signaling pathways. These pathways can cause reactive oxygen species (ROS) to form, which then cause damage in proteins and DNA. Additionally, epidemiological studies have shown that higher short and long-term exposure to air pollution has been associated with increased psoriasis visits to examine flares.

Cellular and Molecular Mechanisms Involved in Air Pollution Induced Psoriasis Development

PM is able to penetrate the skin through areas that have a disrupted barrier, through hair follicles, and sweat glands. PM can penetrate the skin barrier through disrupted stratum corneum. PM triggers psoriasis by inducing oxidative stress, damaging proteins and DNA (2).

Pollutants can damage the outer layers of the skin and can also cause irritation. PAHs often enter the skin when they are attached to particulate matter in air that is polluted. The aryl hydrocarbon receptor (AhR) is a protein inside of skin cells that connects chemical signals from the environment to the body's protective responses, and it does this to remove and mediate dangerous substances such as toxins, to activate the immune system, or to maintain normal cell balance (12). Once AhR is activated, it moves to the nucleus of the cell and joins another protein called aryl hydrocarbon receptor nuclear translocator (ARNT) and together they turn on specific genes. The genes that are activated produce enzymes that can break down toxic chemicals such as PAHs, but they can also create other harmful products such as ROS, and these cause oxidative stress. Reactive oxygen species can damage DNA and proteins (12).

ROS can increase Ca^{2+} levels inside cells, and higher calcium levels lead to keratinocytes maturing and differentiating. This leads to abnormal skin growth in psoriasis. ROS can also activate transcription factors like NF-kB and AP-1 which turn on genes that produce inflammatory cytokines, and these pathways are central to psoriasis inflammation (13). Transcription factors like NF-kB and AP-1 can release pro-inflammatory cytokines, for example, TNF-α. TNF-α is an important cytokine in psoriasis because it drives inflammation, consequently causing keratinocytes to produce more ROS and this creates a cycle. Inflammation produces ROS, the ROS damage cells, damaged cells release cytokines like TNF-α, and then these cytokines can cause more inflammation. This cycle can actually help maintain psoriasis lesions (15).

PM exposure can induce an interaction between the proteins TLR5 and NOX4, leading to the production of ROS and the activation of the interleukin (IL)- 6 cytokine. IL-6 causes inflammation, and promotes chronic plaque formation (2). A study by Grossman et al. (1989) examined psoriasis plaques from 35 patients and found very high levels of IL-6 in active psoriasis plaques. Keratinocytes contained IL-6, and this study found that IL-6 stimulates skin cell growth (14). Overall, IL-6 signaling that can ultimately be caused by PM, contributes to psoriasis by abnormal skin thickening and inflammation.

PM exposure can also damage parts of the cell such as the mitochondria and endoplasmic reticulum. PM can cause endoplasmic reticulum (ER) stress. The ER folds proteins inside the cell, which further increases ROS production (2). Therefore, PM doesn't only cause oxidative stress by triggering the AhR, but it can create oxidative stress in other areas and cause other types of damage to cells. Additionally, the mitochondria can be targeted by pollutants as well, such as PM2.5 (15). Once mitochondria are disrupted,

less ATP is generated, and more ROS are produced. When more ROS are generated, more inflammatory pathways will be triggered that lead to psoriasis or the exacerbation of it.

Along with PM's danger of being able to penetrate the skin, toxic heavy metals, PAHs, sulfates, and nitrates can be absorbed on the surface of suspended particulate matter in the air and cause the substance to be even more harmful. PM can also contain transition metals like iron or copper attached to their surface, which contribute to the production of ROS in cells (2). Transition metals such as iron or copper that are present in PM can generate ROS through Fenton reactions (16). The Fenton reaction is the reaction between iron and hydrogen peroxide, which generates hydroxyl radicals. There are ROS that are generated from the Fenton reaction, and these ROS can trigger pathways that lead to psoriasis and other inflammatory responses (17).

Ozone, another air pollutant, may not be directly linked to causing psoriasis, but it can cause inflammation. Ozone is able to induce oxidative stress, and can create products like peroxides and aldehydes that are able to damage the barrier of the epidermis (1).

These mechanisms demonstrate how air pollution can contribute to the development or exacerbation of psoriasis. Pollutants such as PM can penetrate the barrier with other toxic compounds including PAHs and transition metals attached to them which can induce oxidative stress. These substances can activate pathways such as AhR and stimulate inflammatory signaling pathways which may increase the production of ROS. An increased amount of ROS can damage parts of the cell, such as the mitochondria and ER. Elevated ROS levels can also promote the release of inflammatory cytokines like TNF-α and IL-6 which can lead to increased keratinocyte production and chronic inflammation. These studies help clarify how air pollutants can trigger psoriasis on a cellular level, but we also need to investigate how common this is in individuals and populations.

Epidemiological studies on air pollution and psoriasis

Many studies establish a link between air pollution and psoriasis. A study done in Beijing, China recorded 500,266 patient visits for psoriasis using large-scale healthcare database records. China is one of the most polluted countries in the world, due to their rapid industrial growth, large pollution, and dependence on fossil fuels. This study found that a same day increase of $PM_{2.5}$ concentrations was associated with a 0.29% increase in daily outpatient visits for psoriasis (18). While this percentage is small, it does suggest that elevated concentrations of $PM_{2.5}$ may be associated with exacerbations in psoriasis. Additionally, pollution exposure was estimated at the city level, which may not capture individual exposure variation that determines a patient's actual risk.

A different study that collected data at a dermatology clinic in Italy supported the results from the previous study, as the researchers noted that short-term air pollution may trigger psoriasis. Information from a total of 957 patients with psoriasis, with 4398 follow-up visits who attended the dermatologic clinic of the University Hospital of Verona for their examination were included in this study. It is important to note that concentrations of $PM_{2.5}$ and PM_{10} were significantly higher 60 days before the

psoriasis flares occur. The risk for having a more severe case of psoriasis was approximately 40-50% higher among patients in higher exposure groups for $PM_{2.5}$ and PM_{10} (10).

For someone who already has psoriasis, air pollution may amplify the abnormal immune response that causes thick and scaly skin patches. A study carried out in the UK revealed an increased risk of psoriasis with participants with higher genetic vulnerability and increased exposure to pollutants. This study also calculated a polygenic risk score (PRS) for psoriasis, where they combined genetic variants known to increase psoriasis risk. Participants who had a high genetic risk and were exposed to $PM_{2.5,}$ PM_{10}, and NO_2 were 2-4 times more likely to develop psoriasis compared to the low risk group. This study was composed of 474,055 individuals who were tracked over about 12 years, while air pollution was measured simultaneously (19).

While these studies were conducted across different geographic regions, they show consistent trends linking higher pollution exposure to increased psoriasis severity and clinical visits. Across these three studies, PM2.5 emerges as the most consistent risk factor, even though the magnitude of its association varies considerably. For example, the Beijing study reported smaller increases in daily visits, while the Italian cohort observed substantially higher risks of disease with prolonged exposure. This discrepancy likely reflects differences in outcome measures (outpatient visit counts vs flare severity) rather than a true difference between biological effects. This additionally highlights a broader inconsistency in how psoriasis outcomes are measured across epidemiological literature. The UK Biobank is the only one of the three that studies that highlights the importance of genetic susceptibility, and it suggests that pollution may have a stronger effect in individuals who are genetically predisposed. The UK Biobanks' conclusion that gene-environment interaction plays a strong role in this development may explain some of the variability in results seen between the Chinese and Italian results. Together, these findings indicate that while the association between air pollution and psoriasis is consistent, the strength of the relationship is influenced by exposure duration, outcome measurement, and individual genetic susceptibility.

Long-exposure to air pollutants such as $PM_{2.5}$, PM_{10}, NO_2, SO_2, nitrogen oxides and ground-level ozone are all associated with a higher risk of developing psoriasis. Short-term risk has also been associated with higher flares of psoriasis and outpatient visits for these flares. There are many epidemiological studies that show that clinical visits for psoriasis flares increase during times where air pollution levels are elevated. Additionally, genetic predisposition can play a large role in triggering these flares and may increase the likelihood of a psoriasis flare from pollution. Lastly, among the pollutants studied, $PM_{2.5}$ and PM_{10} appear to have the strongest and most consistent association with inflammatory skin diseases across different regions. This is likely due to its ability to penetrate the skin barrier and carry in other compounds that can lead to oxidative stress and trigger immune pathways.

AIR POLLUTION AND ATOPIC DERMATITIS (ECZEMA)

Eczema or atopic dermatitis usually manifests in earlier life stages, such as infancy and childhood, and it is common for young children to grow out of this skin disease as they age. Air pollution has reportedly contributed to the development and exacerbation of AD (20).

How air pollution triggers eczema

As with psoriasis, pollutants trigger eczema through an initial phase of oxidative stress and generation of ROS, but the downstream signaling differs substantially. Instead of driving keratinocyte proliferation as they do in psoriasis, pollutants impact the skin barrier and immune system.

Research has shown that with higher levels of PM2.5 and PM_{10}, more individuals with AD report symptoms. Air pollutants may promote AD by damaging the skin barrier, which can later cause irritations. Studies have suggested that volatile organic compounds, NO_2, and O_3 may impair the skin barrier in individuals with AD. When these pollutants come in contact with the skin, they can enter it through hair follicles, sweat glands, or damaged areas, the same way the pollutants that induce psoriasis enter the skin. Extended or frequent exposure to PM can not only induce skin irritation through mechanisms like oxidative stress, but it can result in epidermal thickening and dermal inflammation.

When the pollutants previously described enter the skin, they cause oxidative stress and ROS are created. These ROS can cause filaggrin downregulation, and consequently damage to the epidermis. This leads to the skin barrier becoming weaker and allows pollutants to enter the skin more easily. Fligarrin is crucial in maintaining hydration, and when pollution decreases filaggrin levels, water escapes more easily. The skin will become more dry, and this can be a major trigger for an eczema flare. Skin barrier damage also alters the skin microbiome and leads to an abnormal balance of skin bacteria. Barrier damage induced by pollution can cause more *staphylococcus aureus*, which is harmful bacteria, and lead to less staphylococcus epidermidis which is beneficial bacteria. These pollutants can also trigger Th2-mediated immune responses, and this can cause more allergic reactions. Th2 lymphocytes release cytokines such as IL-4 and IL-13. Th2 immune responses cause increased inflammation and can lead to an increased production of IgE antibodies. IgE is an antibody that is commonly involved in allergic reactions and higher levels of this can lead to increased itching and sensitivity to allergens (21).

Overall, these findings demonstrate that air pollution contributes to atopic dermatitis by disrupting the skin barrier, altering the microbiome, and activating inflammatory immune responses. While these biological mechanisms explain how pollutants trigger eczema on a cellular level, it is also crucial to examine how this can affect real-world populations.

Epidemiological studies on air pollution and eczema

Many epidemiological studies provide important evidence on the relationship between air pollution exposure and eczema by observing large populations, which has allowed researchers to identify patterns and how genetics may influence the development of eczema.

A study based on a UK Biobank that examined 500,000 participants found that the groups exposed to higher levels of air pollution had a higher risk of developing early onset atopic dermatitis compared to the group that was exposed to lower pollution levels. The group that had a low genetic risk and was exposed to high levels of air pollution were less affected than the group with a high genetic risk. These observations show that for someone who is genetically predisposed to atopic dermatitis, risk of developing AD when exposed to air pollution is higher.

A study performed in South Korea investigated the association between PM and other types of pollutants, and AD. The authors monitored levels of $PM_{2.5}$ and 10, SO_2, NO_2, and CO, and found that increases in levels of these pollutants were associated with a significant increase in monthly patient visits for AD. This study also covered a very large area as their statistics were from the seven largest cities in South Korea (9). South Korea is one of the most highly industrialized nations in the world, with Seoul being ranked number 13 for most polluted cities. The air quality index of other cities in South Korea ranges from 131-148 in the most polluted cities. Overall South Korea is ranked 59/138 in global air pollution (22). Similar to other studies discussed in this review, exposure was estimated at the city level rather than individually. This may understate the variability in pollutant exposure across participants in the study. South Korea has 85 cities and it is likely that pollution levels vary considerably between them. Every $10ug/m^3$ increase in $PM_{2.5}$ led to a 2.71% increase in patient visits for AD, every $10ug/m^3$ increase in PM10 caused a 2.01% increase in patient visits. Additionally, every 1ppb increase in SO_2 caused a 2.26% increase in visits, and every 100ppb increase in CO caused a 2.86% increase in visits. This study also looked at O_3 and NO_3, but found that these two pollutants were not associated with increased patient visits for AD (9).

China is an area of the world that has a dangerous abundance of air pollution. The highest PM concentrations are in Beijing, but significant levels of pollution extend throughout the country. $PM_{2.5}$ levels for roughly 28% of the population are classified as unhealthy, and PM_{10} levels are less severe. A study conducted in China evaluated dermatologic conditions of 20,138 students from all 34 provinces of China. Air pollution levels of O_3, CO, NO_2, SO_2, $PM_{2.5}$, and PM_{10} over the course of 6 years were obtained from a database and this data was linked to over 19,000 of the participant's hometowns. Results showed that students exposed to CO were 2.57 times more likely to develop AD (23). Students exposed to NO_2 and second-hand smoke were 2.4 times more likely to develop AD, which contrasts with the results from the previous study that was carried out in South Korea. The exact pollutants in second-hand smoke were not defined in this study. This highlights how lifestyle factors like second-hand smoke can be difficult to separate from ambient pollution effects in city-level studies. The smoke may induce additional inflammation that this study may have not separated from the effects of the NO_2. Overall, these findings suggest that when air pollution is combined with second-hand smoke, together these substances may amplify the inflammatory response that the skin has.

CONCLUSION

Air pollution has increasingly been recognized as an important environmental factor that can contribute to the development or exacerbation of inflammatory skin diseases such as psoriasis and atopic dermatitis. Pollutants such as PM, NO_2, O_3, SO_2, and PAHs can penetrate the skin barrier, induce oxidative stress which produces ROS that can damage cellular structures, weaken the epidermal barrier, and activate inflammatory signaling pathways.

In psoriasis, pollution often activates pathways such as AhR, NF-kB and cytokine signaling. This can lead to keratinocyte proliferation and chronic inflammation. In atopic dermatitis, pollutants can disrupt the skin barrier by reducing filaggrin levels and promoting Th2-mediated immune responses that increase inflammation and allergic sensitivity. In atopic dermatitis, air pollution can activate immune pathways and lead to oxidative stress. This can reduce filaggrin levels, impair the skin barrier, and promote Th2-mediated inflammation, which increases skin sensitivity, dryness, and allergic responses.

Epidemiological studies from regions with high levels of air pollution support these conclusions. Studies show that when pollution levels increase, so do associated disease flares and dermatologic visits. Additionally, genetic susceptibility is a factor that may interact with environmental exposure, and can increase the likelihood that pollutants will trigger disease in individuals that are at a higher genetic risk. Epidemiological studies demonstrate correlations between air pollution levels and inflammatory skin disease flares; however, mechanistic studies showing oxidative stress and immune activation, provide biological explanations that support a potential causal relationship.

With the rapid growth of industrialization and urbanization, it is crucial that the relationship between environmental pollution and skin health isn't overlooked. As treatments for these conditions are limited, further research should investigate methods to protect the skin barrier and reduce pollution.

While many studies demonstrate a strong association between different types of air pollution and inflammatory skin diseases, it is important to note other factors that may contribute to this relationship. As mentioned, genetic predisposition plays a significant role in this relationship. In addition to this, other factors that may alter this relationship include socioeconomic status, climate-related factors, and lifestyle factors. An individual who lives in a highly urbanized or industrial area is likely to experience higher pollution levels. Climate-related factors include temperature and humidity. Low humidity and cold weather are known triggers for eczema, and this was a factor that is not mentioned in a majority of studies. Lifestyle factors such as time spent outside and skincare could further influence this. If someone exercises outside a lot, they may be exposed to more air pollution. Therefore, while pollution is an important environmental trigger for psoriasis and eczema, there are other variables that should be accounted for when determining the reliability of this relationship.

Despite the strong associations identified, there are several limitations within the studies that are included in this review. Many epidemiological studies rely on pollution levels within an entire city and not in specific areas, which may reduce accuracy. Additionally, differences in how these skin conditions are evaluated and diagnosed could make it difficult to accurately compare results across studies. For example, some studies measure hospital visits, while others assess only self-reported symptoms, this could lead to potential inaccuracies. The epidemiological studies that have been done are in areas that contain high levels of air pollution, yet regions that have low levels of air pollution have not studied this relationship.There are also variables like second- hand smoke that are not taken into account in all studies, which could make them less comparable. Lastly, most studies focus on short-term associations, while the effects of long-term exposure to air pollution isn't understood as well.

Due to ethical constraints, controlled experimental exposure of harmful air pollutants in humans is not acceptable. However, there are alternative research designs that could strengthen the understanding of this relationship. For example, a study could track individuals over time and compare disease severity during periods of high versus low pollution exposure. The studies included in this review look at pollution exposure on a population level and not on an individual one. This means that two individuals living in the same area may experience significantly different exposure to air pollution depending on their daily activities.

Future research should aim to strengthen the causal understanding of this relationship, and experimental approaches are needed to complement findings from epidemiological studies. Controlled laboratory studies using cultured human skin cells or reconstructed skin models could directly examine how specific air pollutants affect oxidative stress, cytokine production, and the skin barrier. Intervention-based studies could also be conducted to determine whether antioxidant-based skincare treatments could help protect the skin of individuals that live in areas with high levels of air pollution

Ultimately, understanding the complex interaction between environmental exposure to air pollution, genetic susceptibility, and the immune response will be essential in developing more effective prevention strategies and treatments for these skin conditions.

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