



Air pollution and skin diseases: Adverse effects of airborne particulate matter on various skin diseases

Kyung Eun Kim^a, Daeho Cho^{a,*}, Hyun Jeong Park^{b,**}

^a Department of Life Science, Sookmyung Women's University, Seoul 140-742, Republic of Korea

^b Department of Dermatology, Yeouido St. Mary's Hospital, The Catholic University of Korea, Seoul 150-713, Republic of Korea

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ABSTRACT

Environmental air pollution encompasses various particulate matters (PMs). The increased ambient PM from industrialization and urbanization is highly associated with morbidity and mortality worldwide, presenting one of the most severe environmental pollution problems. This article focuses on the correlation between PM and skin diseases, along with related immunological mechanisms. Recent epidemiological studies on the cutaneous impacts of PM showed that PM affects the development and exacerbation of skin diseases. PM induces oxidative stress via production of reactive oxygen species and secretion of pro-inflammatory cytokines such as TNF- α , IL-1 α , and IL-8. In addition, the increased production of ROS such as superoxide and hydroxyl radical by PM exposure increases MMPs including MMP-1, MMP-2, and MMP-9, resulting in the degradation of collagen. These processes lead to the increased inflammatory skin diseases and skin aging. In addition, environmental cigarette smoke, which is well known as an oxidizing agent, is closely related with androgenetic alopecia (AGA). Also, ultrafine particles (UFPs) including black carbon and polycyclic aromatic hydrocarbons (PAHs) enhance the incidence of skin cancer. Overall, increased PM levels are highly associated with the development of various skin diseases via the regulation of oxidative stress and inflammatory cytokines. Therefore, anti-oxidant and anti-inflammatory drugs may be useful for treating PM-induced skin diseases.

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1. Introduction

Particulate matter (PM), which includes the harmful suspended contaminants in the air, is generally encompassed in air pollution [65,27]. The airborne PM is a complex mixture including particulate contaminants (smog, tobacco smoke, soot, etc.), various types of dust, biological contaminants (pollen, house dust mite allergens, etc.), and gaseous contaminants (exhaust gas from traffic or hood, etc.). It comprises sulfates,

* Correspondence to: D. Cho, Department of Life Science, Sookmyung Women's University, Chungpadong 2-ka, Yongsan-ku, Seoul 140-742, Republic of Korea.

** Correspondence to: H. J. Park, Department of Dermatology, Yeouido St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Youngdeungpo-ku, 62 Yeouido-dong, Seoul, 150-713, Republic of Korea.

E-mail addresses: cdhkor@sookmyung.ac.kr (D. Cho), hjpark@catholic.ac.kr (H.J. Park).

¹ Hyun Jeong Park and Daeho Cho contribute to this work equally.

nitrates, and carbon compounds in the atmosphere [65,27]. As a result of rapid industrialization and urbanization, environmental pollution is becoming a severe public problem worldwide. In particular, airborne PM in the ambient atmosphere is highly associated with the incidence of respiratory and cardiovascular diseases and with increased mortality [39,10,87]. The World Health Organization (WHO; Fact Sheet N 313, 2014) has established that premature death by air pollution occurs with cardiovascular diseases, respiratory diseases, and lung cancer at rates of 80%, 14%, and 6%, respectively. The ambient PM is the most intimate element of the human health impacts. Recent increased PM concentration in the air pollution is correlated with the increased mortality and morbidity. It affects human health even at low concentration. Due to the significant impacts of small PM, WHO has suggested a guideline for limited concentration of PM. PM₁₀ must not exceed 20 $\mu\text{g}/\text{m}^3$ (annual mean) and 50 $\mu\text{g}/\text{m}^3$ (24-h mean). In the case of PM_{2.5}, the mean of concentration must not exceed 10 $\mu\text{g}/\text{m}^3$ per year and 25 $\mu\text{g}/\text{m}^3$ for 24 h.

Airborne PM is classified as particulate matter, fine particulate matter, and ultrafine particles depending on the aerodynamic diameter of particles. Particles that are less than 10 μm are called the particulate matter (PM₁₀) and are inhalable. PM₁₀ is composed of inhalable particles from dusts, industrial emissions, and traffic emissions [27]. Inhalation of PM₁₀ is highly related to various respiratory diseases. Infiltration of PM₁₀ into the lungs results in systemic immune responses, including enhanced lung inflammation due to increasing various pro-inflammatory cytokines such as interleukin (IL)-1, IL-6, IL-8, and monocyte chemoattractant protein-1 (MCP-1) from lung epithelial cells and macrophages. The lung inflammations cause various diseases of the lung and other inflammation-related conditions [29,27].

In the 1990s, a smaller PM particle size (less than 2.5 μm) was discovered and defined as fine PM (PM_{2.5}). Fine PM is primarily comprised of organic carbon compounds, nitrates, and sulfates [17,95]. Ambient PM_{2.5} is increasingly present in the surrounding air and is significantly associated with human health and especially respiratory tract diseases because PM_{2.5} can reach bronchial tubes and deep lung. In addition, PM_{2.5} is associated with the exacerbation of cardiovascular diseases and systemic inflammation [24,16,80]. A cohort study in which participants were followed for decades has discovered that compared with larger particles, smaller particles like PM_{2.5} have much more adverse effects on human health [23].

Recently, ultrafine particles (UFPs) have been found in abundance in urban air and defined as a new particle type. UFPs are less than 100 nm in diameter and have greater potential for adverse effects to human health than PM_{2.5}, because UFPs can penetrate into the blood stream and accumulate in the lung and other organs of the body [23,28]. As well as the size, the composition of UFPs is also important with regard to its impact on human health. Both indoor and outdoor UFPs are primarily from cigarette smoke, engines, cooking fumes, and industry. The most important sources of UFP are vehicular exhausts, especially diesel exhaust that includes black carbon, which contains large amounts of UFP [38] and is defined as a class one carcinogen by WHO. UFP, the smallest PM, result in chronic obstructive pulmonary disease (COPD) and asthma through their deposition in the lungs without the filtration through the nasal mucosa [3].

Although PM is divided into three different types based on the mean of aerodynamic diameter, PM₁₀ generally includes all fractions of PM₁₀, PM_{2.5}, and UFPs [27]. Deposition by inhalation is primarily in the upper respiratory tract (head and comparatively large conducting airway), lower respiratory tract (larynx, small airway, and alveoli), and deep lung (alveoli) for PM₁₀, PM_{2.5}, and UFPs, respectively [27]. These three categorized particulate matters are globally implicated in severe environmental pollution due to significant increases in urbanization and industrialization processes. Given the growing concern about environmental pollution, we have chosen to discuss how PM air pollution affects various diseases, and especially skin diseases.

2. Review

Skin is the largest organ in body, and acts as the first and most important defense barrier against environmental contaminants. Skin is always exposed to the contaminants, and various industrial chemicals can be absorbed into the skin. These absorbed compounds can cause local toxicity in the skin and systemic toxicity in other organs, although it may enter by percutaneous penetration. The severity of these toxicities depends on the age and medical history of skin diseases. Percutaneous penetration is significantly related with the integrity of the barrier, anatomic site, age, and properties of the contaminants. Therefore, children and patients with impaired skin barriers are easily affected by dermal exposure due to the increased absorption [88]. Skin consists of three main layers: epidermis, dermis, and hypodermis. The stratum corneum of the epidermis is the outermost skin layer, acting as the main functional barrier. A “brick and mortar” model describes pathways for skin penetration across the stratum corneum. According to the arrangement of the corneocytes, which are nonviable keratinocytes, there are four pathways for skin penetration, including mechanical delivery, an intracellular route, a transcellular route, and a transfollicular route [56,9]. Skin has numerous pores, which are decidedly larger than PM, there is no direct evidence that PM can penetrate into skin regardless of smaller size. However, it is undoubtedly reported that the particles can penetrate skin through hair follicles depending on their size, indicating the penetration of PM through transfollicular route [47]. Hair follicles interrupt the functions of stratum corneum barrier through the formation of intrusions in the stratum corneum, thus providing a route for penetration. In addition, chemical compounds are accumulated in the orifice of hair follicles. Because the hair follicles on the scalp and face, which are easily exposed to the environment, constitute approximately 10% of the total skin surface, the follicles allow greater absorption by the transfollicular route [9]. Also, there are many reports that PM contribute to various skin diseases, such as inflammatory skin diseases, skin aging, androgenetic alopecia (AGA), and skin cancer. In this article, we review what is known about the association between ambient PM and skin diseases.

2.1. Particulate matter air pollution and inflammatory skin diseases: atopic dermatitis, acne, and psoriasis

Recent epidemiological investigations into the effect of environmental contamination, especially ambient air pollution, on several skin diseases indicate that some PM affects the progression of inflammatory skin diseases, such as atopic dermatitis (AD), acne, and psoriasis [77, 94,83,34]. AD is a chronic and recurrent inflammatory skin disease with symptoms of itching and eczema that usually begins during infancy or childhood. The incidence of AD worldwide is on the rise [90,5]. Although the mechanism of AD is still not clear, environmental pollution have been implicated one of the complicating factors of AD with genetic predisposition and immunologic mechanisms [44,74,22,79]. PM adversely affects patients with allergic diseases, including asthma and AD [59,27]. Moreover, the increased concentration of PM in ambient air is strongly related to progression of AD in children. Long-term exposure to PM_{2.5} and nitrogen dioxide from vehicle exhaust is significantly associated with eczema and allergic sensitization [59]. Also, high levels of PM₁₀, PM_{2.5}, and UFP intensify AD symptoms, such as itching, among children with pre-existing AD [77]. These studies provide evidence that PM-exposed children have a high risk for developing AD and/or for exacerbation of pre-existing disease. Furthermore, short-term exposure of the UFP nitrogen dioxide mainly impairs the epidermal barrier function (e.g., transepidermal water loss), resulting in the exacerbation of AD symptoms [18]. Recent epidemiologic studies suggest that indoor and outdoor air pollution exacerbate AD symptoms [42,43]. In nine randomly selected kindergarteners, reduced PM₁₀ concentration following improvement of air quality considerably influenced symptoms of AD [42]. PM₁₀ concentration in the indoor air was reduced from $182.7 \pm 23.68 \mu\text{g}/\text{m}^3$ to $73.4 \pm 22.05 \mu\text{g}/\text{m}^3$ after

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