Chapter

Atmospheric Pollution and Atopic Dermatitis

Gael Ananfack, Mazou Ngou Temgoua and Joel Noutakdie Tochie

Abstract

Atopic dermatitis is a frequent allergic dermatological disorder seen frequently in childhood. Affected patients often have a genetic predisposition and other atopic diseases like asthma, hay fever and allergic rhinitis. There are several triggering factors for atopic dermatitis among which the most recently established one is atmospheric or air pollution. The latter is due to the increased in industrialization in cities with the emission of waste products in the atmosphere as air pollutants. The role played by these pollutants in the pathogenesis of atopic dermatitis still remains largely unclear. This chapter elucidates the relationship between atmospheric pollution and atopic dermatitis.

Keywords: atopic, dermatitis, air, pollution, effects

1. Introduction

Atopic dermatitis (AD) is a chronic inflammatory dermatosis of multifactorial aetiologies. It is a common disease that frequently occurs in childhood. A rising prevalence rate of AD over the past six decades has been reported to be mainly due to several environmental factors [1, 2]. In the interplay between the interactions predisposing genes, environmental factors, impaired skin barrier integrity, skin microbiota, and immune deregulation are at the core of the pathogenesis of AD [3]. Established risk factors for AD include a regular diet rich in fresh fruits and fish during pregnancy leading to AD in offspring of these women during childhood. Avoiding such a diet by pregnant women has been demonstrated to reduce both the prevalence and incidence of AD in children and adolescents [4–6]. Furthermore, it has been reported that a family history of asthma, hay fever, or eczema is associated with AD in childhood and this risk increases if both parents have eczema [7]. Persons of similar racial and genetic background are at an increased risk of AD in metropolitan areas compared with countryside individuals [8]. Urbanization and industrialization often occur together. Industrialization in urban areas is often associated with atmospheric or air pollution which can be mild to severe depending on the degree of industries startups in the town or city concerned. The air pollution stemming from these industries has recently been positively correlated to the development of AD. However, the relationship between atmospheric or air pollution and AD still largely remains to be elucidated. With the several advancements made in industrialization, there is emerging of some chronic diseases which share

the same pathophysiological mechanisms with AD. Thus it is important to evaluate the contribution of atmospheric pollution in the growing burden of AD.

2. Atmospheric or air pollution and chronic inflammatory diseases

Although air pollution is well known to be harmful to the lung and airways, it can also damage most other organ systems of the body. Pollution affects the immune system and is associated with allergic rhinitis, hypersensitivity disorders, and autoimmune diseases. The lung has a large surface area that comes into contact with a myriad of antigens. It sensitization effect and antigen-presenting system are quite efficacious and this consequently makes individuals susceptible to autoimmune diseases. The pollution of air markedly contributes to illnesses such as systemic lupus erythematosus and rheumatoid arthritis [9]. A Canadian study found increased odds of having a diagnosis of a rheumatic disease following an increased exposure to ambient particulate matter with an aerodynamic diameter < 2.5 mm ($PM_{2.5}$) exposure [10]. Air pollutants have also been described to trigger or exacerbate diseases like juvenile idiopathic arthritis, but the impaired autoimmunity related to exposure to air pollution has largely been understudied. Inflammation in the bloodstream in response to air pollutants has been found to cause systemic vascular (including cerebral vascular) dysfunctions. Studies on animals observed that inhaled ultrafine particles from the atmosphere into the nostrils then get in contact with the neighbouring olfactory nerve and later to the central nervous system, particularly to the brain leading to inflammatory and oxidative stress responses [11]. In all the organs that are affected by air pollution, the skin is one of the most frequently involved leading to atopic skin disease.

3. Atmospheric or air pollution and atopic dermatitis

3.1 Evidence

Since almost one-third of patients with AD develop this skin disorder within their first year of life, it may be important to consider the impact of prenatal exposure to air pollution. In a study published almost a decade ago and involving 469 pregnant women, prenatal exposure to fine air pollutants (fine air particulate matter—$PM_{2.5}$) with subsequent postnatal exposure to the same air pollutants and cigarette smoke had their children followed up every three months for a year [12]. The prevalence of AD during the first year of life increased by two-fold [12]. Likewise, a Swedish study showed an association between AD and lower ventilation in the houses, in particular, in the child's bedrooms [13]. Furthermore, a German study found an association between indoor renovation activities (painting, furniture, and floor covering) and the antenatal period, infantile period and early childhood and lifetime prevalence of AD, likely in connection with high levels of volatile organic compounds (VOCs) [14]. More still, a study of 317,926 Taiwanese children found a significant positive association between traffic-related air pollutants such as carbon monoxide, nitrogen oxides and AD in both sexes [15]. In the same vein, a study conducted on 4907 French children found associations of both history and lifetime of AD with urban air pollutants such as NOx, CO, NO_2, PM10, and benzene pollutants [16].

A USA population-based study found the prevalence of childhood AD to be associated with mean annual NO_2, sulfur dioxide (SO_2), and sulfur trioxide (SO_3) [17]. A German birth cohort study found that the rates of AD in the first children

aged one to six years old was positively correlated with the degree of home proximity to motorable roads [18]. Also, the distance from home to the closest road was used as the main indicator of air pollution due to road traffics. The highest odds of AD occurred in children <50 m from the main road [18]. The authors postulated that residents living closer to heavy traffic are exposed to both higher amounts of and more toxic air pollutants that are freshly emitted from vehicles moving on these roads. Two longitudinal studies assessed the relationship between outdoor pollution and childhood AD symptom severity. A South Korean study of 41 children aged 8–12 years collected symptom diaries for 67 days and found significant associations between pruritus severity and daily ambient air particulate matter concentrations [19]. A longer-term study of 22 Korean children using symptom diaries for 18 months also found associations of AD symptoms with levels of outdoor air pollutants [20]. From the aforementioned data, it is clear that outdoor and indoor air pollution can cause, trigger and/or exacerbate AD.

3.2 Pathophysiology

As an allergic disease, the triggers of AD may originate from indoor and/or outdoor environmental factors and can interact with the skin by binding to the stratum corneum, becoming metabolized, or even penetrating the epidermis and entering systemic circulation through dermal capillaries [21]. The biomechanical effect of particulate matter is not entirely clear. It contains a myriad of toxic substances such as tobacco and alloy smoke, polycyclic aromatic hydrocarbons, organic compounds, nitrates, sulphates and metals. These particulates have the capacity to cross through the skin, the respiratory tract and blood placental barrier. They also have a slow index of sedimentation. Hence, they remain as air suspension over a longtime where they have dust mites and pollen 'carrier' effects due to their protein linking ability. When pregnant women are exposed to polycyclic hydrocarbons, this has several adverse effects to the foetas. These negative foetal effects include the formation of free radicals, activation of apoptosis, and the production of IgE and cytokines. Postnatal exposure to air pollutants increases the effects of prenatal exposure and has been implicated in lesions to the skin barrier, with a resulting inflammatory process [12, 22, 23]. There are likely multiple mechanisms for the harmful effects of different air pollutants. A study of skin biopsies from 75 AD patients found an association between severe AD and reactive oxygen species-related damage. This finding is in favour of the hypothesis that reactive oxygen species originating from environmental exposures cause oxidative stress damage to proteins in the stratum corneum [24]. Even short-term exposure to NO_2 or volatile organic compounds (VOC) caused significantly increased trans-epidermal water loss (TEWL) in both healthy individuals and those with AD [23, 25]. VOC may also contribute to T helper 2 (Th-2) polarization, suggesting potentially direct effects of pollutants on the immune system [26].

4. Public health implications

Air pollution is a public health problem today. Its ill-health effects are increasing worldwide. Assessing these effects may be difficult because the source of air pollution varies between communities and household situations. Governments should, therefore, put in place measures to reduce environmental air pollution in the aforementioned high-risk areas (e.g. those living close to the roadside) and people-centered measures such as facemasks which can reduce inhaled particulates. For instance, wearing personal respirators such as facemasks while being active in central Beijing

reduced the blood pressure (BP) and heart rate variability, and markers associated with cardiovascular morbidity [27]. The beneficial effects of the use of personal respirators on cardiovascular parameters markers were almost immediate and lasted during the entire exposure time [28]. Air purifiers also reduce air particulate matters. Air purification for just 48 hours significantly decreased $PM_{2.5}$ and reduced circulating inflammatory and thrombogenic biomarkers as well as systolic and diastolic BPs.

5. Conclusion

Atopic dermatitis is a growing disease; the risk factors are numerous and include air pollution. Air pollutants act by several mechanisms including the synthesis of reactive oxygen species which will cause a weakening of the skin barrier and thus exposes individuals to various degrees of atopic dermatitis. The increasing urbanization and development of countries that increase air pollution will probably aggravate this disease. Air pollution has a proven effect on the burden of AD. This should sensitize the general population especially AD patients and public health authorities in particular about the impact of air pollution on pollution health, especially dermatology health.

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