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Air Pollution and its Effects on Autoimmune Diseases

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Environmental exposures, genetic talent, and epigenetic agents are considered as effective factors in the emerging of autoimmune diseases (ADs) [1-3]. ADs are specified by the loss of self-tolerance and unfit to the generation of autoantibody and immune-mediated tissue destruction [4]. Almost 5% of the world's population is affected by this disease which is the fourth leading cause of disability in women [1, 5].

Studies have shown that the prevalence of ADs is growing worldwide [6]. ADs include a wide range of illnesses containing systemic lupus erythematous (SLE), rheumatoid arthritis (RA), multiple sclerosis (MS), type 1 diabetes mellitus (T1DM), and etc [4, 7]. Autoimmune diseases, the most important challenges of medicine, cause chronic disability and mortality in individuals with pulmonary and cardiovascular diseases [8].

ADs have a powerful genetic history involved [9, 10], but the impact of environmental agents must not be minimized [11]. Studies have reported that environmental agents are about for 40-70% of all Ads [8, 12]. As the leading environmental risk factor; air pollution causes and intensifies a number of illnesses [13, 14]. Human activities such as transporting, manufacturing, smoking, and agriculture as well as natural sources such as forest fires and volcanic eruptions cause air pollution [15]. Anthropogenic and geogenic pollutants are caused by a combination of nitrogen dioxide [NO_2], sulfur dioxide [SO_2], ozone [O_3], and carbon monoxide [CO] along with particulate matter (PM) [16]. In this regard, PM can be sorted according to the particles' origin, including chemical (hydrocarbons), metallic (nickel, iron), mineral (silica, quartz), and biological (pollen and endotoxins) sources [17, 18].

Recent studies have reported that air pollution is involved in the increase of ADs. The role of polluted air in initiate ADs marks the reaction of PM available in air pollution with the immune system in the lungs and the effects of long-term inflammation [19]. Based on the main evidence, air pollution can generate an imbalance of T cell, generation of pro-inflammatory cytokines, airway injury, oxidative stress and methylation changes to the beginning and exacerbation of ADs [20]. Further studies carried out in vitro and in vivo with emerging molecular biologic methods will be used to assess potential main effects of air pollutants and the mechanisms related to the initiation and the aggravation of these diseases [21].

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If the causal relation between polluted air exposure and ADs is defined, even if air pollution involves only a little section of these diseases, the effect of air pollution on the global population will be significant. Therefore, it is important to improve public health policies to decrease exposure to air pollution, and to further study its molecular and cellular methods involved in ADs.

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