

Role of Interleukin-10 in COPD in Tobacco Smokers with Chronic Ozone Exposure

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a common, usually slowly developing and progressing condition affecting people in all parts of the world. Major causes of this disease include prolonged exposure to inhaled air pollutants, causing chronic inflammation, leading to pulmonary tissue destruction and remodeling. Tobacco smoke is an important and most recognized airway irritant linked to development of COPD. Other air pollutants, such as ground-level ozone, have been also linked to development of inflammatory lung diseases. Release and action of anti-inflammatory mediators, such as pleiotropic cytokine interleukin (IL)-10 may reduce the inflammatory response and protect against tissue injury. Role and effects of IL-10 on development and progression of COPD secondary to tobacco smoking and prolonged exposure to ground-level ozone have not been studied. Anti-inflammatory effects of IL-10 may significantly slowdown the development and progression of COPD in some sub-populations of patients due to its pleiotropic expression. Further studies could eventually help to develop effective treatment for some COPD patients.

Keywords

Smoking, Tobacco, Ozone Exposure, COPD, Interleukin-10.

Chronic obstructive pulmonary disease (COPD) is a common slowly developing and progressing respiratory disease affecting lives of many people around the world. Chronic and repeated exposure to various inhaled irritants is known to play an important role during development and progression of COPD [1,2]. The process of COPD development and progression involves activation of different types of immune cells by multiple endogenous pro-inflammatory mediators, such as pro-inflammatory cytokines interleukin (IL)-1 β , IL-6, tumor necrosis factor (TNF) α , IL-8, IL-17, IL-18, IL-32 and interferon (IFN)- γ , resulting in chronic inflammation, tissue injury and remodeling. Multiple evidence indicated involvement of pro-inflammatory cytokines during development and progression of COPD [3-16]. Tobacco smoking is well established cause of this chronic pulmonary disease [17,18].

Ozone is a powerful oxidant causing damage to respiratory tissues. This ground-level pollutant is produced by reaction of sunlight on air containing hydrocarbons and nitrogen oxides. High levels

are produced in heavily populated urban areas around the world. Evidence indicates that long-term exposure to ground-level ozone and the air pollutants that produce it significantly correlate with development of pulmonary diseases. People living in urban areas with high ozone levels had increased risk of dying from lung disease [19,20].

IL-10 is a pleiotropic anti-inflammatory cytokine produced by activated monocytes, macrophages, helper T-cells, and B-cells, reducing production of IL-6 and TNF- α [21]. Intratracheal instillation of recombinant IL-10 in Sprague-Dawley rats before ozone (O₃) exposure significantly reduced O₃-induced PMN infiltration and pulmonary hyperpermeability responses [22]. The exact mechanism of relationship between IL-10 and other inflammatory mediators and downstream molecular events is not known. Polymorphisms in the human IL-10 gene have been associated with the development of asthma [23]. Another experimental study provided more evidence that IL-10 protects against O₃-induced lung inflammation, and also that IL-10 deficiency enhanced O₃-induced neutrophilic inflammation and injury at the centriacinar region of the lung [24]. Role of IL-10

during development and progression of COPD secondary to tobacco smoking and prolonged exposure to ground-level ozone has not been studied. Studies are needed in this area. Anti-inflammatory effects of IL-10 may significantly slow down the development and progression of COPD in some sub-populations of patients due to its pleiotropic expression. Further studies could eventually help to develop effective treatment for some COPD patients.

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