Introduction

Asthma is a clinical syndrome characterized by episodic reversible airway obstruction, increased bronchial reactivity, and airway inflammation. Asthma is now recognized to result from complex interactions among inflammatory cells, their mediators, airway epithelium and smooth muscle, and the nervous system. In susceptible individuals, these interactions can lead to symptoms of breathlessness, wheezing, cough, and chest tightness, ***(Wang et al., 2005)***.

In children, exposure to dust mites within the first year of life is associated with later development of asthma and, possibly, atopy. The level of IgE is associated with the prevalence and severity of airway hyperreactivity and asthma, ***(Brussee et al., 2005)***. Mite and cockroach antigens are common, and exposure and sensitization has been shown to increase asthma morbidity. Removing even one of many allergens can result in clinical improvement, ***(Kidon et al., 2005)***.

The mammalian immune system is comprised of T lymphocytes, cells that help fight off infection. One type of T lymphocyte is the T helper, which is categorized as either a Th_1 anti-inflammatory cell or a Th_2 pro-inflammatory cell. A healthy immune system modulates the balance of Th_1 and Th_2 production, ***(Bochner & Busse, 2005)***.

The Th_1 cells recognize new foreign particles, fight off the invaders, and "remember" what the enemies look like for future reference. In learning about the enemy, Th1 memory cells also make interferon gamma (IFN-γ), which inhibits formation of interleukin-4 (IL-4), a *cytokine* responsible for the asthmatic response. Cytokines such as

IFN-g and IL-4 are cells which signal proteins that elicit necessary immune responses from other cells. On the other hand, Th_2 inflammatory response prevents more allergic particles from entering the lungs. The Th1 cells prevent the overproduction of Th2 cells, which causes lung inflammation, ***(Custovic et al., 2005)***.

Corticosteroids are immunosuppressing agents and, thus, can decrease inflammation with particular efficacy in skin eruptions and bronchospasm. Inhaled corticosteroids are key in controlling inflammation of bronchial airways and nasal mucosa, ***(Anand & Routes, 2004)***.

McMillan et al., (2005), reported that budesonide administration reduced airway hyper-reactivity and leukocyte infiltration in association with a decrease in production of the Th2 mediators, IL-4, IL-13 and eotaxin-1. A reduction in peribronchiolar collagen deposition and mucus production was observed. Moreover, the administration of Budesonide modulates the progression of airway remodeling following prolonged allergen challenge via regulation of inflammation and active TGF-β signaling.