Introduction

Asthma is a chronic disease characterized by airways hyper responsiveness, airways inflammation, airways remodeling and reversible airways obstruction. Airway structural cells, recruited inflammatory cells and many mediators such as cytokines, chemokines and adhesion molecules are involved in the pathogenesis of asthma. Although asthma is treatable in most, but not all patients by currently available drugs, no treatment is preventive or curative and the disease has reached epidemic proportions worldwide and its incidence is continuing to increase. Many thousands have chronic, severe asthma and suffer daily symptoms making it imperative that we continue to improve our understanding of mechanisms of asthma particularly related to airway inflammation and remodeling, the hallmarks of asthma, and to identify new therapeutic targets **(Fang et al., 2008).**

Anti-inflammatory therapy, usually in the form of oral or inhaled corticosteroids, is currently recommended to treat the bronchial inflammation **(NHLBI, 2003).** Corticosteroids reduce peripheral-blood eosinophilia and microvascular leakage caused by inflammatory mediators, inhibit inflammatory cell influx and decrease inflammation-mediated bronchial hyperreactivity. The vast majority of asthmatics patients can be managed with inhaled corticosteroid therapy, but the most severely affected patient require the addition of oral steroids to control asthmatic symptoms. Patients whose asthma remains poorly controlled despite high dose oral steroid therapy present a difficult therapeutic problem **(NHLBI,1997).**

Mycoplasma pneumoniae (M.pneumoniae) infection has been implicated as a possible mechanism leading to or exacerbating underling chronic pulmonary diseases as bronchial asthma. It play a role in the pathogenesis of asthma beyond simple and acute exacerbation as it can be detected by PCR and or culture more often from the airways of patients with chronic, stable asthma than from matched control patients **(Marc et al.,2000).**

Lung abnormalities including reduced pulmonary clearance and airway hyperresponsiveness may persist for weeks to months after an infection with M.pneumonia is known to induce a number of inflammatory mediators implicated in the pathogenesis of asthma that may play a role in exacerbation, which often include wheezing **(Koh et al.,2001).**

Aim of work

Evaluation of a possible association between Mycoplasma pneuomoniae infection and childhood asthma