Chapter 9 Evidence for *Chlamydia pneumoniae* Infection in Asthma

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Introduction

According to the British Thoracic Society, "asthma is a common and chronic inflammatory condition of the airways whose cause is not completely understood" [1]. Two important clinical characteristics of asthma are: 1) *reversible airway obstruction*, usually manifested by complaints of episodic cough, wheeze, shortness of breath and/or chest tightness, and 2) *bronchial hyperreactivity to a variety of stimuli* including aeroallergens, irritants, cold air, exercize and respiratory infections which can exacerbate or trigger asthma symptoms in susceptible individuals [2]. Asthma is a common medical condition affecting approximately 5-10% of children and adults worldwide [3] and is an important cause of morbidity in all age groups and mortality especially in the elderly [4].

An important recent advance in the understanding of asthma pathophysiology is the recognition that asthma, even in its earliest stages, is associated with chronic inflammation of the airways [5]. Bronchial inflammation appears to be necessary but not sufficient to produce asthma symptoms, which seem to occur only in susceptible individuals, possibly related to genetic acquisition of bronchial hyperreactivity [6]. Thus, asthma may be succinctly characterized as a chronic inflammatory condition of unknown etiology [2].

Because its underlying causes are unknown, asthma must be regarded as a syndrome, not a disease. Although IgE-mediated childhood allergy to common aeroallergens (mites, molds, plants, animal dander, etc.) has become synonymous with asthma in the public consciousness, this type of *allergic* or *atopic* asthma represents only one of several recognized asthma syndromes [7]. A substantial amount of asthma first becomes apparent in adulthood [8], and adult-onset asthma is not uniformly associated with IgE-mediated positive skin test reactions to common aeroallergens [9]. A significant but incompletely quantified proportion of childhood asthma is also not strongly associated with atopy [7].

Burrows et al. [9] recently reported that skin test-negative adult-onset asthma is nevertheless associated with increased levels of serum IgE and blood eosinophils, leading these investigators and others [10, 11] to postulate the existence of a missing antigen (not included in the batteries of common aeroallergens used for skin testing), potentially responsible for producing increased serum IgE levels and eosinophilia in "nonatopic" (skin test negative) asthma patients. Evidence supports the concept that a final common pathophysiologic pathway for both atopic and non-atopic asth-