

Correspondence

Chlamydial infection and asthma: Is early allergic sensitization the only mechanism?

To the editor:

I would like to clarify a statement made by Horvat et al¹ in their interesting article describing results of experimental *Chlamydia muridarum* lung infection of neonatal, infant, and adult mice. They reported that neonatal and infant but not adult chlamydial infection enhanced allergic responses to ovalbumin (increased mucous-secreting cell numbers, IL-13 expression, and airway hyperresponsiveness) whereas neonatal chlamydial infection attenuated and infant infection augmented other ovalbumin-induced features of allergic inflammation (eosinophilia, T_H2 cytokine and dendritic cell responses). In addition, neonatal but not later infection appeared to damage alveoli in an allergen-independent manner. They concluded that early life chlamydial respiratory infections altered lung structure and function and increased the severity of airway sensitization to allergens later in life. They emphasized the potential clinical importance of these findings by stating that “asthma typically originates in early-life.”

Although no one would dispute the importance of childhood asthma, the authors' statement that “the majority of adult asthma originates in child-hood”¹ is not conclusively supported by their 3 cited references.²⁻⁴ One reference was a nonpopulation-based study reporting on infants hospitalized for bronchiolitis that was not relevant.² Another of the references was a well conducted population-based prospective study that followed children only to the age of 26 years.³ The third citation⁴ was a review that included a relevant study⁵ that actually contradicted the belief that most adults with active asthma had acquired the condition as children. In the national British cohort study, Strachan et al⁵ reported in their Table 4 that 507 (65%) of 776 adults with wheezing at age 33 years first became symptomatic after the age of 23 years. The only reliable way to determine the relative contributions of childhood-onset asthma (COA) and adult-onset asthma (AOA) to prevalence and morbidity in adulthood is to conduct population-based, lifelong prospective observational studies. In the absence of this type of study, the next best approach is to review studies that report the relative proportions of COA and AOA at various adult ages.⁶ Such a review indicates that AOA is at least as prevalent as COA, and that AOA is more severe, less likely to remit, and associated with greater mortality than

COA.⁶ Although COA certainly has higher incidence than AOA, there are many more years of adult life during which to accrue cases of AOA.

These comments are not intended to belittle the importance of childhood asthma. In regard to possible chlamydial pathogenesis, the article of Horvat et al¹ is extremely important because it indicates that a *Chlamydia* species can modulate the host immune response to produce an atopic predisposition, at least in a mouse model. Rather, the purpose of these comments is to illustrate that adult-onset asthma and nonallergic mechanisms of action may also be important. Emerging evidence suggests that *Chlamydia*-triggered, nonallergic immunopathologic host responses relevant to asthma (and chronic obstructive pulmonary disease) may include IL-8, neutrophilic asthma, and lung remodeling via inflammatory damage from heat shock protein 60.⁷ Clarification of asthma epidemiology may enhance opportunities for prevention and cure of this illness that affects all age groups.

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