# INCIDENT WHEEZING AND PREVALENT ASTHMA HAVE DIFFERENT SEROLOGIC PATTERNS OF "ACUTE" CHLAMYDIA PNEUMONIAE ANTIBODIES IN ADULTS

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**Objectives**: (1) To determine whether incident wheezing and prevalent asthma have different patterns of acute antibody in patients diagnosed with C. pneumoniae (Cpn) infection and (2) to measure Cpn-specific IgA antibody over time in these patients.

**Methods**: A case series of adult outpatients are reported who (1) were evaluated during either their first ever episode of (incident) wheezing or during chronic established (prevalent) asthma and who (2) were diagnosed with Cpn infection on the basis of either (i) acute antibody as indicated by the presence of IgM, a four-fold rise in titer against either IgM or IgG, or an elevated IgG antibody titer of 1:512 or greater in the MIF test (n=15), (ii) one or more positive cultures (n=3) or (iii) both culture positivity and acute antibody (n=2). Cpn-specific IgA antibody was measured after absorption of IgG. IgA was reported as detectable if a titer of 1:16 or greater was measured. Serologic testing was performed without knowledge of the clinical history. Asthma diagnosis was supported by spirometric testing. Median followup was 10 months (range 1 month to 7 years).

**Results**: Of 159 patients with wheezing who had Cpn antibodies measured, 20 (13%) were diagnosed with Cpn infection: 10 patients (mean age 45, 5M/5F) had incident wheezing and 10 (mean age 55, 7M/3F) had prevalent asthma.

All 10 incident wheezers had acute primary (IgM antibody, n=8) or secondary (four-fold or greater titer rise without IgM, n=2) Cpn infection. Six incident wheezers (60%) subsequently developed asthma (5) or chronic bronchitis (1).

Seven patients with prevalent asthma had persisting high IgG antibody titers of 1:512 or greater without an IgM response or a four-fold titer change (n=7), 2 others were persistently culture-positive and had stable antibody titers of 1:128 and one patient was culture positive and had an antibody titer of 1:16.

Culture positivity correlated with acute antibody in incident wheezing (n=2) but did not correlate with acute antibody in prevalent asthma (n=3).

Cpn-specific IgA antibody has been uniformly and persistently detected in those tested to date (8 of 20), including in the 2 persistently culture-positive patients with IgG titers of 1:128. All incident wheezers who developed chronic disease also developed persistent IgG antibody titers (and persistent IgA in those tested).

Cpn-specific IgA antibody has also been detected in 42 (44%) of 96 other patients with acute asthmatic bronchitis or chronic asthma who had IgG antibody titers of less than 1:512 and thus did not meet serologic criteria for acute infection.

**Conclusions**: (1) Serologic patterns of Cpn infection in asthma are dependent on the stage of disease. (2) In this study, culture positivity in the absence of acute antibody suggests chronic infection rather than lack of sensitivity of the MIF test to detect acute infection. (3) Detection of Cpn-specific IgA antibody following the acute phase of incident wheezing and during prevalent asthma may be a marker for chronic chlamydial infection. (4) Detectable Cpn-specific IgA antibody was associated with almost half (50 of 104) of adult asthma in this study.