Asthma, Bronchial Hyper-responsiveness and *Chlamydophila (Chlamydia) pneumonia* Infection in Adult Thai Population

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Background: *The associations between Chlamydophila (Chlamydia) pneumonia infection and chronic asthma or bronchial hyper-responsiveness (BHR) have been inconclusive.*
Objective: *We aimed to determine the association between C. pneumonia infection and asthma as well as BHR in the adult Thai population.*
Material and Method: *This nested case-control study retrieved the data from a nation-wide Respiratory Health Survey (2001-02) in the adult population (age 20-44 year) in Thailand. Each subject underwent questionnaire interview, spirometry, bronchoprovocative test, skin prick test for common aeroallergens and venous blood collection. Subjects with BHR (n = 79) including those with asthma (n = 52), were randomly selected as cases. Subjects without BHR or asthma were also randomly selected as the control (n = 137). We used the stored serums for the C. pneumonia serologic assay including IgA, IgG and IgM by microimmunofluorescence (MIF) technique.*
Results: *There is no significant relationship between chronic Chlamydia infection (IgG $\geq$ 1:512 and IgA $\geq$ 1:40) and BHR or asthma. Higher IgM was found in subjects with BHR when compared with the control group (p = 0.04). The IgM titer $\geq$ 1:10 was associated with BHR with borderline significance (odds ratio 1.98; 95% CI 0.98-4.00; p = 0.05). Logistic regression analysis revealed no evidence of confounding effects for age, sex and atopy. However, mite allergy seems to be an effect modifier of the relationship between the recent Chlamydia infection and BHR.*
Conclusion: *The present study does not support the hypothesis about the association between persistent C. pneumonia infection and chronic asthma. However, the recent infection may be related with bronchial hyper-responsiveness particularly in those without allergy to house dust mite.*

Keywords: *Adult, Asthma, Bronchial Hyper-reactivity, Chlamydophila pneumonia*

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Over the last decade, there has been substantial evidence of the association between asthma and the infection of some intracellular pathogens especially *Chlamydophila (Chlamydia) pneumonia*. Chlamydial infection has been shown as the precipitating factor for the exacerbation of asthma as well as the risk for developing persistent disease. The association between *C. pneumoniae* and bronchial hyper-responsiveness has also been observed.

A case of acute *C. pneumonia* infection initiating severe chronic asthmatic bronchitis was reported in 1989[1]. It was then reported that 4 of 19 patients with acute *C. pneumonia* infection developed newly diagnosed asthma after illness suggesting that *C. pneumonia* infection may be able to initiate

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asthma[2]. In epidemiological studies, the increase in *C. pneumonia* infection has been found correlated with an increase in asthma[3-5]. There has also been evidence that adult onset asthma is associated with chronic *C. pneumonia* infection[6,7]. The relationship of bronchial hyperresponsiveness and *C. pneumonia* has also been postulated[8,9]. The severity of asthma was also found to be associated with high antibody titers against *C. pneumoniae*[10].

The serological study by ELISA method in blood donor subjects revealed a high prevalence of *C. pneumonia* infection in Thailand[11].

The aim of this study was to determine the association between *C. pneumonia* infection and asthma as well as bronchial hyper-responsiveness in the adult Thai population.

Material and Method

This nested case-control study retrieved the data from a previous Respiratory Health Survey during the year 2001-02[12]. The cross-sectional survey was done in adult population (20-44 year of age) at national level over 5 geographical regions including Bangkok, central, southern, north and north-eastern regions. The sampling was done using proportional multi-staged stratified randomization technique. Household registration was used as a sampling frame. Demographic and clinical data including risk factors of bronchial hyper-responsiveness and asthma were collected and recorded. The data includes sex, age, occupation, socio-economic status, asthma-related symptoms, age at onset, smoking, etc. The results of spirometric measurement and methacholine challenge test were also recorded, as well as the results of skin prick test for common aeroallergens including house-dust mite, cockroach, cat, dog, careless weed, kapok and mixed mold.

The survey yielded that the prevalence of subjects with bronchial hyper-responsiveness (BHR) was 3.98% and the prevalence of definite asthma (reversible airflow obstruction or BHR with asthmatic symptoms within last 12 months) was 2.91%[12]. The serum of all subjects in the study population was collected and stored at -20°C. We therefore used the serum for the serological assay for *C. pneumonia* antibody response in this study.

Calculated sample size under the assumption of 4% of exposure (chronic chlamydial infection) in the control group, with relative risk 5.0[8], 95% confidence interval, 80% power and 2:1 proportion yielded the numbers of 70 cases and 140 controls. Subjects with definite asthma were randomly selected as the study group. The control group is randomly selected from the same cohort with matching for age group and geographical regions. The subjects in the control group were without any criteria for definite asthma or bronchial hyper-responsiveness. Subjects whose serum was not available were excluded from the study.

The serum of subjects who are selected as case and control will be blindly assayed for *C. pneumonia* antibodies including IgA, IgG and IgM. Sera collected from patients were kept at -20°C until tested. Microimmunofluorescence (MIF) test kits (Focus Technologies, California, USA) for detection of IgG, IgM, and IgA antibodies to *C. pneumonia* were performed with serial dilutions of patients' sera. *C. pneumonia* MIF substrate slides have 12 test wells, each with three distinct antigen spots (*Chlamyophila pneumonia, Chlamydia trachomatis* and *Chlamydia psittaci* elementary bodies) and a yolk sac control spot. Endpoint titers of antibody were determined by the last dilution of sera that showed apple green fluorescence under the immunofluorescence microscope. Possible chronic infection is defined if high titer of IgG ($\geq$ 1:512) with high titer of IgA ($\geq$1:40) are demonstrated.

The study proposal including the patient consent forms have been approved by the Faculty of Medicine Siriraj Hospital's Ethical Committee.

Statistical methods

Chlamydial infections were categorized by the titer of antibodies. Chronic infection was defined by the criteria as mentioned. Comparisons of the serological results between case and control groups were done by Chi-square test and odds ratios with 95% confidence interval were presented. Unpaired t-test and Mann-Whitney U-test were also used to compare continuous variables as parametric and non-parametric methods, respectively. Stepwise logistic regression method was used for multivariate analysis.

Results

The demographic data of the study population is shown in Table 1. There is no significant difference between the control and BHR or asthma group except for atopy which has been already known to be associated with asthma or bronchial hyper-responsiveness. The geometric means of Chlamydia antibody titers were compared as demonstrated in Table 2. Higher IgM was found in subjects with BHR compared with the control group ($p = 0.04$). The antibody titers were categorized

Table 1. Demographic data

	Control (n = 137)	BHR (n = 79)		Asthma (n = 52)	
			p-value*		p-value*
Age, mean (SE)	32.7 (0.6)	32.9 (0.8)	0.89	33.5 (1.0)	0.47
Male, n (%)	55 (40.2)	31 (39.2)	0.90	21 (40.4)	0.98
Atopy, n (%)	56 (42.1)	59 (74.7)	<0.001	41 (78.9)	<0.001
Ever smoke, n (%)	39 (28.5)	30 (38.0)	0.15	21 (40.4)	0.12
Regions Central	42 (30.7)	24 (30.4)	0.85	14 (26.9)	0.73
North-eastern	26 (19.0)	17 (21.5)		13 (25.0)	
Southern	16 (11.7)	12 (15.2)		8 (15.4)	
Northern	47 (34.3)	22 (27.9)		14 (26.9)	
Bangkok	6 (4.4)	4 (5.1)		3 (5.8)	

***** Chi-square test except age for which un-paired t-test was used

Table 2. Comparisons of geometric means of *Chlamydophila pneumonia* antibodies*****

	Control (n = 137)	BHR (n = 79)		Asthma (n = 52)	
			p-value†		p-value†
IgM	5.5 (5.3-5.8)	6.0 (5.5-6.5)	0.04	5.9 (5.4-6.5)	0.15
IgG	39.2 (31.2-49.1)	43.6 (31.6-60.2)	0.52	45.3 (30.6-66.9)	0.52
IgA	14.0 (12.4-15.9)	15.3 (12.9-18.2)	0.34	15.4 (12.5-18.9)	0.35

***** Data are presented as geometric mean (95% confidence interval)
† Mann-Whitney U test

and the analysis of the association with BHR and asthma are shown in Table 3 and 4. There is no significant relationship between chronic Chlamydia infection as defined by high titer of IgG ($\geq$ 1:512) with high titer of IgA ($\geq$ 1:40) with BHR or asthma. The IgM titer $\geq$ 1:10 was found associated with BHR with borderline significance (odds ratio 1.98; 95% CI 0.98-4.00; p = 0.05). The comparison between the control and asthma group demonstrates no significant relationship.

Further statistical analysis of the association between Chlamydia IgM antibody and BHR adjusted for atopic status was performed using logistic regression analysis. The results are shown in Table 5. There are no differences between the odds ratio adjusted for age, sex, atopy or mite allergy and the crude odds ratio. There is thus no evidence of the confounding effect among the risk factors. However, the odds ratios seem different between subjects with and those without house dust mite allergy (1.08 vs. 3.17) as shown in Table 6. Although the p-value for effect modification was not significant, this may be due to inadequate sample size for the subgroup analysis.

We compared the geometric means of *C. pneumonia* antibodies in the subgroup of those with BHR according to the degree of PC_{20} to indicate the relationship of the infection and the severity of BHR. The results are shown in Table 7.

Discussion

The relationships between persistent *C. pneumonia* infection and asthma or bronchial hyper-responsiveness are presently inconclusive. There has been evidence that *C. pneumonia* can produce persistent infection with pulmonary inflammation in animal models and can cause persistent or latent respiratory tract infection in humans[13]. *C. pneumonia* can multiply within human pulmonary macrophages and can induce the production of inflammatory mediators, including tumor necrosis factor-a (TNF-a), interleukin-1a (IL-1a), and IL-6 *in vitro*[14] Additionally, it was found that *C. pneumonia* caused ciliostasis of ciliated bronchial epithelial cells and epithelial damage could increase bronchial hyper-responsiveness by exposing patients to allergen sensitization[15]. It is thus possible

Table 3. *Chlamydophila pneumonia* antibodies and bronchial hyperresponsiveness (BHR)

		Control (n = 137)	BHR (n = 79)	Odds ratio (95% CI)	p-value*
IgM	< 1:10	117	59		
	≥ 1:10	20	20	1.98 (0.98-4.00)	0.05
IgG	< 1:32	52	29		
	≥ 1:32	85	50	1.05 (0.59-1.87)	0.86
IgA	< 1:32	107	58		
	≥ 1:32	30	21	1.29 (0.68-2.46)	0.44
Chronic†	No	132	74		
	Yes	5	5	1.78 (0.50-6.40)	0.37

* Chi-square test
† Chronic = subjects with IgG ≥1:512 and IgA ≥ 1:40

Table 4. *Chlamydophila pneumonia* antibodies and asthma

		Control (n = 137)	Asthma (n = 52)	Odds ratio (95% CI)	p-value*
IgM	< 1:10	117	40		
	≥ 1:10	20	12	1.76 (0.78-3.93)	0.17
IgG	< 1:32	52	19		
	≥ 1:32	85	33	1.06 (0.55-2.06)	0.86
IgA	< 1:32	107	37		
	≥ 1:32	30	15	1.45 (0.70-2.99)	0.32
Chronic†	No	132	48		
	Yes	5	4	2.20 (0.56-8.61)	0.25

* Chi-square test
† Chronic = subjects with IgG ≥ 1:512 and IgA ≥ 1:40

Table 5. Logistic regression analysis of the association between bronchial hyperresponsiveness and *Chlamydophila pneumonia* IgM antibody

	Odds ratio for IgM ≥ 1:10	p-value*	p-value for effect modification*
Crude	1.98 (0.98-4.00)	0.05	
Adjusted for age	2.00 (0.99-4.04)	0.06	0.66
Adjusted for sex	1.98 (0.99-3.97)	0.05	0.47
Adjusted for atopy	2.04 (0.97-4.28)	0.06	0.77
Adjusted for mite allergy	2.08 (1.01-4.28)	0.047	0.15

* Likelihood ratio test

Table 6. The association between bronchial hyperresponsiveness and *Chlamydophila pneumonia* IgM antibody according to allergic status to house dust mite

	Non-allergic to mite		Allergic to mite	
	Control	BHR	Control	BHR
IgM < 1:10	79	23	34	36
IgM ≥ 1:10	13	12	7	8
Odds ratio (95% CI)	3.17 (1.27-7.89)		1.08 (0.35-3.30)	
p-value*	0.01		0.89	

* Mantel-Haenszel test

Table 7. Comparison of geometric means of *Chlamydophila pneumonia* antibodies according to the degree of bronchial hyperresponsiveness

	PC_{20} > 4 mg/mL (n = 43)	$PC_{20} \leq$ 4mg/mL (n = 33)	p-value*
IgM	5.7 (5.2-6.2)	6.4 (5.5-7.5)	0.20
IgG	51.9 (33.6-80.2)	34.8 (21.2-57.1)	0.22
IgA	14.8 (11.9-18.3)	16.0 (11.9-21.5)	0.85

* Mann-Whitney U-test

that chronic or latent *C. pneumonia* infection may contribute substantially to the development of stable or persistent asthma.

However, the results of this study do not support the hypothesis which posts the association between chronic chlamydial infection defined by IgA antibody and bronchial hyper-responsiveness or asthma. This finding is consistent with other recent studies. It was reported that chlamydial infection is not a major risk for asthma in children and young adults[16]. It was also found that there was no association between Chlamydia serology and asthma in newly asthmatic children[17]. No statistical differences for the serological results between the asthma and control groups was reported[18]. Moreover, a study in a cohort of 1,211 children revealed that the incidence of asthma and allergic rhinitis in *C. pneumonia* infected subjects were lower[19]. It was recently reported that subjects with acute *C. pneumonia* infection do not subsequently develop bronchial hyper-responsiveness[20].

The present study nevertheless found that subjects with bronchial hyper-responsiveness (BHR) had higher IgM antibody response to *C. pneumonia* than the control. This finding is consistent with a previous study reporting the association of recent Chlamydial infection and bronchial hyper-responsiveness[8]. We also found that the relationship of high IgM antibody and bronchial hyper-responsiveness was present specifically in subjects without allergy to house dust mite (Table 6). There have been reports about the relationship of chlamydial infection and nonatopic asthma specifically. A previous study revealed a strong relationship between chlamydial IgG antibody and nonatopic asthma[21]. It was recently reported from a longitudinal study that chronic *C. pneumonia* infection accelerates the loss of lung function significantly in subjects who contracted new nonatopic asthma[22]. It was also reported that the association between chlamydial IgG antibody and wheezing was restricted to girls without atopic sensitization[23]. Repeated infections early in life were also found strongly associated with the prevalence of asthma and current wheeze at school age, particularly among those with nonatopic asthma[24] It may be inferred that *C. pneumonia* infection can lead to the development of bronchial hyper-responsiveness by pathways other than those with atopy. This may involve a mechanism for the exacerbation of asthma due to chlamydial infection. However, the association between high IgM antibody and asthma in this study was not statistically significant (Table 4). It is likely that the bronchial hyper-responsiveness which developed after *C. pneumonia* infection is transient because there was no evidence of the relationship of persistent infection as defined by high IgA antibody and bronchial hyper-responsiveness or asthma in this study. Airway responses to respiratory infections have been studied. Allergic individuals experience greater changes in airway responsiveness than do non-allergic individuals after naturally acquiring common colds[25]. It was also reported that allergic individuals had a significantly greater change in BHR during experimental rhinovirus infection as compared with normal subjects[26]. The result of present study, therefore, is in contrast with those since we found the association of chlamydial infection and BHR only in the subjects without allergy to house-dust mite.

The mechanism for the development of BHR induced by chlamydial infection is still not clear. *C. pneumonia* has been found to cause ciliostasis in bronchial epithelial cells *in vitro*[15]. This effect appears to be a specific property of *C. pneumonia* since *C. trachomatis* had no similar effect. *C. pneumonia* also induces pro-inflammatory cytokine synthesis in human peripheral mononuclear cells and alveolar macrophages[27,28].

The association of high IgM antibody and BHR in those without allergy to house dust mite in this study is still inconclusive and may only be found by chance. The interactions between IgM antibody and atopic status or the allergy to other aeroallergens were not revealed. Further studies with a specific design are thus needed to clarify this relationship.

The severity of asthma was found to be associated with high antibody titers against *C. pneumonia* in the previous study[10]. We therefore compared the geometric means of chlamydial antibodies in those with bronchial hyper-responsiveness with different levels of PC_{20} as shown in Table 7. No statistical significant difference was demonstrated.

In conclusion, the present study does not support the hypothesis of the association between persistent *C. pneumonia* infection and chronic asthma. However, recent infection may be related with bronchial hyper-responsiveness particularly in those without allergy to house dust mite.

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โรคหืด ภาวะหลอดลมไวเกิน และการติดเชื้อ Chlamydophila (Chlamydia) pneumonia ในประชากรผู้ใหญ่ในประเทศไทย

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***ภูมิหลัง**: ปัจจุบันยังไม่ทราบแน่ชัดถึงความสัมพันธ์ระหว่างการติดเชื้อ Chlamydophila (Chlamydia) pneumoniae กับโรคหืดและ/ภาวะหลอดลมไวเกิน*

***วัตถุประสงค์**: คณะผู้นิพนธ์ได้ทำการศึกษาครั้งนี้เพื่อศึกษาถึงความสัมพันธ์ระหว่างการติดเชื้อ C. pneumoniae กับโรคหืดและภาวะหลอดลมไวเกินในประชากรผู้ใหญ่ในประเทศไทย*

***วัสดุและวิธีการ**: เป็นการศึกษาแบบ nested case-control โดยนำข้อมูลจากการสำรวจสุขภาพระบบการหายใจในประชากรไทย ระหว่างปี พ.ศ. 2544-45 โดยได้ทำการสำรวจในประชากรอายุ 20-44 ปี ประชากรในกลุ่มตัวอย่างทุกรายได้รับการสัมภาษณ์ประวัติ การตรวจสมรรถภาพปอด การทดสอบความไวหลอดลม การทดสอบภาวะภูมิแพ้ทางผิวหนัง และการเก็บตัวอย่างเลือด คณะผู้นิพนธ์ได้สุ่มเลือกประชากรกลุ่มศึกษา ได้แก่ ผู้ที่มีภาวะหลอดลมไวเกิน (จำนวน 79 ราย) รวมทั้งประชากรที่ได้รับการวินิจฉัยว่าเป็นโรคหืด (จำนวน 52 ราย) และประชากรกลุ่มเปรียบเทียบ ได้แก่ ผู้ที่ไม่มีภาวะหลอดลมไวเกินหรือโรคหืด (จำนวน 137 ราย) จากนั้นทำการตรวจหาแอนติบอดีชนิด IgA, IgG และ IgM ต่อเชื้อ C. pneumonia ด้วยวิธี microimmunofluorescent (MIF) โดยใช้ซีรัมของกลุ่มตัวอย่างที่เก็บไว้*

***ผลการศึกษา**: ไม่พบความสัมพันธ์ระหว่างการติดเชื้อ C. pneumonia ชนิดเรื้อรัง (IgG ≥ 1:512 และ IgA ≥ 1:40) กับภาวะหลอดลมไวเกินหรือโรคหืด พบแอนติบอดีชนิด IgM ในประชากรกลุ่มที่มีภาวะหลอดลมไวเกิน สูงกว่าในประชากรกลุ่มเปรียบเทียบ (p = 0.04) ระดับไตเตอร์ของ IgM ≥ 1:10 อาจมีความสัมพันธ์กับภาวะหลอดลมไวเกิน (odds ratio 1.98; CI 0.98-4.00); p = 0.05) การวิเคราะห์แบบ logistic regression ไม่พบว่า อายุ เพศ หรือ ภาวะภูมิแพ้เป็นปัจจัยรบกวน (confounding factor) อย่างไรก็ตาม พบว่าภาวะภูมิแพ้ต่อไรฝุ่นอาจเป็น effect modifier ต่อความสัมพันธ์ระหว่างการติดเชื้อ chlamydia ในระยะเวลาไม่นานกับภาวะหลอดลมไวเกิน*

***สรุป**: การศึกษานี้ไม่สนับสนุนสมมติฐานความสัมพันธ์ระหว่างการติดเชื้อ C. pneumonia ชนิดเรื้อรังกับโรคหืด อย่างไรก็ตาม การติดเชื้อในระยะเวลาไม่นานอาจสัมพันธ์กับภาวะหลอดลมไวเกิน โดยเฉพาะประชากรที่ไม่มีภาวะภูมิแพ้ต่อไรฝุ่น*