Home Exposures, Parental Atopy, and Occurrence of Asthma Symptoms in Adulthood in Southern Taiwan*

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Objective: **Parental atopy and environmental exposures at home have been recognized risk factors for adulthood asthma. However, the relative contributions of specific risk factors and the overall contributions of heredity or home exposure remain unexplored. The purpose of this study was to identify predictors and estimate the population attributable risk (PAR) of each exposure for typical asthma symptoms among 26- to 50-year-old Taiwanese. We also investigated whether an interactive effect existed between parental atopy and home exposures on the occurrence of asthma symptoms in adulthood.**

Design: **A cross-sectional study with retrospective components.**

Setting: **Elementary and middle schools in Southern Taiwan.**

Subjects: **Between March and October 2004, we conducted a cross-sectional survey among schoolchildren's parents from 94 elementary and middle schools in Southern Taiwan. The main outcome measure was typical asthma-like symptoms occurring within the preceding 5 years. Information on hereditary and home exposures was collected by using a self-administered questionnaire.**

Results: **After excluding unqualified questionnaires, data from 24,784 subjects were left for analysis. New-onset asthma was reported for 0.83% of male (n = 80 of 9,662) and 1.36% of female subjects (n = 206 of 15,122). Besides parental atopic factors, environmental tobacco smoke or pet avoidance and visible mold on walls at home were independently associated with the occurrence of asthma symptoms. Mutually adjusted models produced statistically significant associations between any home exposure (odds ratio [OR], 1.80; 95% confidence interval [CI], 1.08 to 3.23; PAR, 28.04%), parental atopy (OR, 4.47; 95% CI, 3.47 to 5.75; PAR, 31.38%), and new-onset asthma. However, there was no interaction between parental atopy and home exposures.**

Conclusions: **Home exposures and parental atopy both increased the risks of new-onset asthma in adulthood but did not show an interactive effect. These two exposure categories approximately contributed equally to the adulthood asthma.** ***(CHEST 2006; 129:300–308)***

Key words: adult; asthma; home exposure; gene-environmental interaction; parental atopy; population attributable risk

Abbreviations: CI = confidence interval; ETS = environmental tobacco smoke; OR = odds ratio; PAR = population attributable risk

During the last quarter century, evidence has accumulated that the prevalence and incidence of asthma among adults has increased in many countries.[1–4] The changing pattern of disease has not been fully explained, in part because of an incomplete understanding of its pathogenesis. The changes have been too rapid to be accounted for by changes

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in gene frequencies. It is also unlikely that they can be totally accounted for by changes in either diagnostic patterns or in reporting attitude of asthmatic symptoms by the general population.[5] They do, however, suggest a role for environmental exposure in the etiology of this evolving epidemic.[6]

Our knowledge about the influence of home environments on respiratory symptoms has improved. Up to the present, the literature is rather consistent regarding its importance in childhood asthma, but there is sparse information on the situation in adults. Adult-onset asthma and child-onset asthma are quite different in clinical characteristics and pathogenesis.[7,8] However, the role of genetic predisposition in adult-onset asthma is also less clear than in child-onset asthma.[8] Both genetic and environmental factors are believed to contribute to adulthood asthma.[9–11] Epidemiologic evidence concerning the relative contributions of environmental stimuli and genetic propensity to adult-onset asthma has been insufficient and warrants further investigation.

In Taiwan, the best of our knowledge, only one epidemiologic report[12] has addressed adulthood asthma in the general population. We conducted the present prevalence study to investigate the relationship between parental atopy, home environmental factors, and the occurrence of typical asthma symptoms among 26- to 50-year-olds. The population attributable risk (PAR) was estimated and compared for each exposure category. We also tested the hypothesis that the joint effect of genetic predisposition to asthma and home environmental exposure on the risk of new-onset adulthood asthma is greater than expected on the basis of their independent effects.

Materials and Methods

Population and Study Design

Southern Taiwan, the southernmost part of Taiwan Island, comprises five administrative districts: Tainan City, Tainan County, Kaohsiung City, Kaohsiung County, and Pintung County. The overall population and territory of this area in 2002 were 5,501,747 people and 7,914 square kilometers. Between March and October 2004, we conducted a cross-sectional, school-based survey for respiratory diseases and symptoms in middle- and elementary-school children and their parents. Twenty of the 189 middle schools and 74 of the 627 elementary schools in Southern Taiwan were randomly selected and investigated. Classroom incentives but not individual incentives were used to encourage participation. Stratified sampling by grade was applied in each school. The study protocol was approved by the Respiratory Health Screening Steering Committee of the Taiwan Department of Health and by the Institutional Review Board at our university hospital. It also complied with the principles outlined in the Helsinki Declaration.[13] We collected data after informed consent was obtained from parents for their own participation. Each completed questionnaire was reviewed and verified by a researcher for quality control.

Definition of Asthma

The questionnaire we used for parents' respiratory health was a Chinese language version translated and modified from the questionnaire of the American Thoracic Society and the Division of Lung Diseases (ATS-DLD-78).[14,15] The validity of the revised questionnaire was evaluated by a pulmonologist and two epidemiologists before the study was conducted. Prior to the core questionnaire, several typical asthma-like symptoms, such as wheezy or whistling in the chest, nocturnal dyspnea, and night cough unassociated with a cold, were described in detail. The definition of asthma used in this study was determined by a positive response to the question, "Have you ever experienced the aforementioned asthma-like symptoms?" If the subject answered "yes," he/she would be further asked, "At what point in your life did the symptoms first appear?" Those who reported an onset of symptoms within 5 years were defined as new-onset asthma subjects.

Genetic and Environmental Determinants

The English literature on asthma determinants was reviewed to identify the hereditary and environmental risk factors for adulthood asthma. We focused on modifiable factors in the home environment that could be investigated by questionnaire survey. These included cockroaches, water damage, visible mold on walls, incense burning, pet ownership, carpet use, and environmental tobacco smoke (ETS) exposure. Information on history of ETS or pet avoidance was also requested. Parental atopy was a measure of genetic predisposition and was defined as a history of maternal or paternal asthma, allergic rhinitis, or allergic eczema. To adjust for possible confounding, we also included host-related variables: sex, age, education level, family income, and smoking status. Unfortunately, neither blood samplings nor skin tests were performed in our large study design.

Statistical Analysis

Older people might be more likely to have asthma-like symptoms.[12] Therefore, we decided to omit subjects > 50 years old from our data set because of the difficulty in distinguishing between asthma and COPD.[16,17] Bivariate analyses were conducted to determine associations with new-onset adulthood asthma. We developed multivariate logistic regression models containing all predictors with positive effects to asthma occurrence. Reduced models were then created by removing exposure variables that were not positively associated with asthma or $p > 0.15$ in the full models.

PARs were also calculated to estimate the contribution of various risk factors for new-onset asthma. PAR represents cases that would be prevented if the subjects were not exposed to specific agents or risk factors. PAR was calculated using the formula $P(R-1)/[P(R-1)+1]$, where P is the prevalence of the exposure, and R is the relative risk due to the exposure.[18]

Individual and joint effects of home environment and parental atopy on new-onset adulthood asthma were estimated using indicator variables created for each category, omitting the hypothesized low-low risk category. Estimates for each of the three exposure categories with the reference group were derived from the same logistic regression model, after adjustment for confounders. Statistical significance was set at $p < 0.05$ based on a two-sided calculation.

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Results

Our study surveyed the parents of 35,682 children from 94 elementary and middle schools. A total of 9,990 subjects were excluded from our study due to refusal participation, inadequate demographic information, or missing responses to the key questions. Data from 25,692 subjects remained satisfactory, with an overall response rate of 72.1%. Most of the participants were between 26 and 50 years old. We omitted 908 subjects > 50 years old; 24,784 subjects (9,662 male and 15,122 female) were left for further analysis. In our population, 895 cases of asthma were identified; in these cases, 286 subjects (32.0%) reported symptoms onset within the preceding 5 years. Overall, 0.83% of the male and 1.36% of the female subjects had new-onset asthma (Table 1). In Taiwan in 2002, there were approximately 4.66 million men and 4.54 million women between 26 and 50 years of age, so this prevalence corresponds to approximately 100,400 cases of new-onset asthma. Table 1 also shows the characteristics of the study subjects. Female, younger subjects, lower education level, lower family income, and former smoking were found to be associated with the occurrence of adulthood asthma (Table 2).

After adjustment for host factors such as sex, age, education level, family income, and smoking status, we found paternal and maternal asthma and allergic rhinitis/atopic eczema to be significantly related to new-onset adulthood asthma (Table 3). Among these parental atopic factors, maternal asthma showed the strongest effect, with an odds ratio (OR) of 11.61 (95% confidence interval [CI], 8.07 to 16.42; PAR, 15.97%). Among home environmental factors, subjects who had ever avoided ETS and pets were 1.60 times and 3.21 times, respectively, more likely to have asthma develop in the preceding 5 years. Visible mold on walls at home was independently associated with the occurrence of asthma symptoms in adulthood (OR, 1.49; 95% CI, 1.09 to 2.01; PAR, 7.74%). The presence of cockroaches, dogs, birds, and ETS exposures at home were also positively associated with adulthood asthma but did not reach statistical significance (Table 3).

While mutually adjusted models were applied, we found a statistically significant association between home exposure—defined as cockroaches, visible mold on walls, dogs, or ETS or pet avoidance—and new-onset asthma (OR, 1.80; 95% CI, 1.08 to 3.23). Parental atopy—defined as parental asthma and allergic rhinitis/atopic eczema—was also noted as a predictor of new-onset asthma (OR, 4.47; 95% CI, 3.47 to 5.75) [Table 3]. The PARs were 28.04% for home environmental factors and 31.38% for parental atopic factors. For all the hereditary and environmental factors we identified, the total PAR was 59.42% of our population. Of the estimated 100,400 cases of new-onset adulthood asthma in 26- to 50-year-old Taiwanese residents, we estimated that approximately 31,500 excess cases of new-onset asthma were attributable to hereditary factors. Home environmental factors accounted for around 28,200 excess cases, which was consistent with 5,640 excess cases annually occurring in Taiwan.

Compared with subjects of the reference category, subjects with home exposure but without parental atopy were 1.61 times more likely to have adulthood asthma developed in the preceding 5 years. Those with parental atopy alone were 3.26 times as likely. The adjusted OR of new-onset adulthood asthma was 7.32 (95% CI, 4.01 to 14.99) in subjects with both atopic heredity and home exposures (Table 4). There was no interaction of family history of atopy and environmental exposure at home (p value of the interaction term > 0.05). The combined effect of these two factors was approximately as expected on the basis of their independent effects in the multiplicative scale ($1.61 \times 3.26 \fallingdotseq 7.32$).

Discussion

Our large cross-sectional questionnaire survey among 26- to 50-year-old adults provides evidence of the effects of hereditary and environmental factors on the occurrence of adulthood asthma in Taiwan. Although our study was cross-sectional, we analyzed data by case-control study method, which was very efficient compared with a cohort study yielding a similar amount of information. Our data corresponded to a follow-up of 24,784 adults for 5 years, and the incidence of typical asthma symptoms was calculated as approximately 2.4 cases per 1,000 person-years.

The present study demonstrated that both home exposures and parental atopy increased the risk of asthma symptoms but did not show an interactive effect. These two exposure categories approximately contributed equally to new-onset adulthood asthma. We also found approximately 5,640 excess asthma cases annually attributable to home exposures. The effect of eliminating these home environmental factors, if they are indeed causal, would have a profound impact on hospitalization rates, clinic and emergency department visits,[19] or even some nonmedical costs such as work loss and early retirement in adults.[20]

Questionnaires have been widely used to assess the prevalence of asthma. The previous Taiwanese study[12] reported the lifetime asthma prevalence for patients > 18 years old was 7.8%. However, we only found 3.6% adults had asthma-like symptoms in our

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Table 1—*Distribution of Potential Risk Factors and Prevalence of Asthma in the Study Population

Risk Factors	Subjects, %	Subjects, No.	Asthma Onset Within 5 Years (n = 286), %	Asthma Onset > 5 Years Ago (n = 609), %	No Asthma (n = 23,889), %
Sex					
Male	38.98	9,662	0.83	2.43	96.74
Female	61.02	15,122	1.36	2.47	96.16
Age, yr					
26–30	3.95	978	2.35	2.86	94.79
31–35	19.34	4,793	1.44	2.36	96.20
36–40	37.81	9,371	1.41	2.44	96.15
41–45	29.35	7,273	0.71	2.28	97.00
46–50	9.56	2,369	0.42	3.08	96.50
Education†					
Elementary school or less	6.63	1,593	1.63	3.01	95.35
Middle school	22.41	5,384	1.10	2.02	96.88
High school	49.47	11,886	1.14	2.39	96.47
College or beyond	21.49	5,163	1.12	2.81	96.07
Family income, $†‡					
0–400,000	40.27	9,307	1.21	2.28	96.51
400,001–600,000	28.25	6,528	1.18	2.39	96.43
≥ 600,001	31.48	7,276	1.15	2.56	96.29
Smoking status†					
No	75.28	18,182	1.23	2.35	96.42
Current	24.21	5,848	0.89	2.53	96.58
Former	0.51	122	2.46	3.28	94.26
Asthma in father†					
No	97.56	23,707	1.07	2.20	96.73
Yes	2.44	592	4.39	10.64	84.97
Asthma in mother†					
No	97.80	23,698	0.95	2.19	96.85
Yes	2.20	532	9.59	11.84	78.57
Allergic rhinitis/atopic eczema in father†					
No	93.06	22,612	1.03	2.23	96.74
Yes	6.94	1,687	2.85	4.74	92.41
Allergic rhinitis/atopic eczema in mother†					
No	93.76	22,719	1.04	2.28	96.67
Yes	6.24	1,511	2.65	4.24	93.12
Cockroaches seen monthly at home, No.†					
0	17.17	4,149	0.89	2.19	96.91
1–2	41.48	10,026	1.09	2.21	96.70
≥ 3	41.36	9,996	1.33	2.68	95.99
Water damage at home, d/yr†					
0	89.32	21,643	1.13	2.33	96.54
1–7	9.91	2,402	1.17	3.21	95.63
≥ 8	0.76	185	2.70	1.62	95.68
Walls with visible mold at home, No.†					
0	82.67	19,965	1.06	2.27	96.67
1	11.57	2,793	1.68	2.90	95.42
≥ 2	5.76	1,391	1.73	3.45	94.82
Incense burning at home†					
No	42.53	10,267	1.23	2.50	96.27
Yes	57.47	13,873	1.12	2.38	96.50
Dog at home†					
No	80.28	19,415	1.11	2.43	96.46
Yes	19.72	4,769	1.38	2.41	96.20
Cat at home†					
No	97.74	23,638	1.15	2.41	96.44
Yes	2.26	546	1.65	3.30	95.05
Bird at home†					
No	96.45	23,325	1.16	2.40	96.45
Yes	3.55	859	1.40	3.26	95.34
Carpet use at home†					
No	89.99	21,845	1.17	2.44	96.39
Yes	10.01	2,430	0.95	2.30	96.75
ETS at home, No. of cigarettes/d†					
0	56.53	13,497	1.07	2.16	96.78
1–20	40.70	9,717	1.22	2.76	96.02
≥ 21	2.77	661	1.66	2.87	95.46
ETS avoidance†					
No	93.34	21,704	0.95	2.13	96.91
Yes	6.66	1,549	3.62	5.81	90.57
Pet avoidance†					
No	91.53	21,481	0.93	2.10	96.97
Yes	8.47	1,989	3.47	5.83	90.70

*Some percentages do not total 100 because of rounding.
†No. of subjects does not add up to total number because of missing data.
‡New Taiwan dollars per year ($1 US = $32 New Taiwan).

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Table 2—*ORs of New-Onset Adulthood Asthma in Relation to Personal Characteristics, Parental Atopy, and Home Environmental Factors*

Risk Factors	OR	95% CI
Sex		
Male	1.00	
Female	1.66	1.28–2.16
Age, yr		
26–30	1.00	
31–35	0.60	0.38–0.99
36–40	0.59	0.39–0.95
41–45	0.30	0.18–0.50
46–50	0.18	0.08–0.36
Education		
Elementary school or less	1.00	
Middle school	0.66	0.42–1.07
High school	0.69	0.46–1.07
College or beyond	0.68	0.43–1.11
Family income, $*		
0–400,000	1.00	
400,001–600,000	0.97	0.72–1.30
≥ 600,001	0.95	0.72–1.26
Smoking status		
No	1.00	
Current	0.75	0.50–1.03
Former	2.05	0.50–5.48
Asthma in father		
No	1.00	
Yes	4.67	3.02–6.92
Asthma in mother		
No	1.00	
Yes	12.39	8.92–16.91
Allergic rhinitis/atopic eczema in father		
No	1.00	
Yes	2.90	2.10–3.94
Allergic rhinitis/atopic eczema in mother		
No	1.00	
Yes	2.63	1.85–3.65
Cockroaches seen monthly, No.		
0	1.00	
1–2	1.22	0.85–1.80
≥ 3	1.51	1.06–2.20
Water damage, d/yr		
0	1.00	
1–7	1.04	0.69–1.51
≥ 8	2.41	0.85–5.32
Walls with visible mold, No.		
0	1.00	
1	1.61	1.15–2.19
≥ 2	1.66	1.06–2.48
Incense burning		
No	1.00	
Yes	0.91	0.72–1.16
Dog		
No	1.00	
Yes	1.25	0.94–1.64
Cat		
No	1.00	
Yes	1.45	0.69–2.66
Bird		
No	1.00	
Yes	1.22	0.64–2.09
Carpet use		
No	1.00	
Yes	0.81	0.51–1.21
ETS, No. of cigarettes/d		
0	1.00	
1–20	1.16	0.91–1.48
≥ 21	1.58	0.80–2.80
ETS avoidance		
No	1.00	
Yes	4.06	2.98–5.43
Pet avoidance		
No	1.00	
Yes	4.00	3.01–5.26

*New Taiwan dollars per year ($1 US = $32 New Taiwan).

population. Because our samples did not include those > 50 years old, we reduced the possibility of information bias from COPD, which seems undifferentiated with asthma by questionnaire survey.[16,17] In our result, only 497 subjects (55.5%) with typical symptoms had ever been recognized as having asthma by physicians. This finding suggests that asthma is underdiagnosed and needs more attention in Taiwan. Therefore, we used prevalence of typical asthma-like symptoms as our outcome measurement in determining risk factors in further analyses.

Adult women had a higher rate of new-onset asthma, which was consistent with other studies[2,21] in western countries. Asthma appeared to affect underprivileged populations disproportionately in United States[22] and Sweden.[2] In our population, subjects with lower family income or education level were more likely to have new-onset asthma (Table 2). Although not statistically significant, former smoking was also a positive predictor of new-onset asthma in our population, which was consistent with the findings in recent studies.[2,23] Because all these factors were potential confounders in risk factor analyses, they were controlled as covariates.

Research has showed that current exposure to indoor ETS might increase the risk of adult-onset asthma.[9,24,25] In a Canadian study[26] of 20- to 44-year-olds, the risk of asthma was related to current pet ownership at home, with an OR of 1.6 for cats (95% CI, 1.1 to 2.4) and for dogs (95% CI, 0.9 to 2.9). Subjects with typical asthma symptoms might reduce ETS and pet exposures. Although selection bias might be introduced and would mask the relationship,[25] especially in cross-sectional design, our results showed positive risks of current ETS and pet exposures to the new-onset asthma (Table 3). History of ETS and pet exposures at home would also increase the risk of adulthood asthma. Jaakkola et al[9] noted a dose-dependent effect with cumulative ETS exposure on incident asthma. They also found adults who had had pets > 12 months would also have significant risks.[10] We used the answers from history of ETS and pet avoidance, partly reflecting past exposure at home. Subjects who had ever avoided ETS and pets were 1.60 times and 3.21 times, respectively, more likely to have typical asthma symptoms develop in the preceding 5 years (Table 3). We also found relatively higher risks of new-onset asthma in adults with past ETS and pet exposures than in those with current exposures, which was consistent with other studies from Sweden[27] and Finland.[10]

Incense burning and carpet use at home had negative effects on the occurrence of adulthood asthma (Table 2), which was consistent with our previous asthma study in children.[28] The possible

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Table 3—*ORs With 95% CIs, Mutually Adjusted ORs, and PARs for Parental Atopy and Home Environmental Factors Associated With New-Onset Adulthood Asthma

Risk Factors	Prevalence, %	OR	95% CI	OR§	95% CI	PAR, %	Adjusted OR	95% CI	PAR, %
Parental atopy									
Asthma in father	2.44	2.63†	1.55–4.24	2.63†	1.55–4.24	3.39			
Asthma in mother	2.20	11.61†	8.07–16.42	11.61†	8.07–16.42	15.97			
Allergic rhinitis/atopic eczema in father	6.94	2.41†	1.67–3.39	2.41†	1.67–3.39	8.55			
Allergic rhinitis/atopic eczema in mother	6.24	2.02†	1.36–2.90	2.02†	1.36–2.90	5.77			
Any parental atopy	15.43			4.55	3.54–5.85	34.09	4.47	3.47–5.75	31.38
Home environment									
Cockroaches	82.84	1.21‡	0.84–1.81	1.23‡	0.85–1.84	15.99			
Water damage	10.67	0.81‡	0.50–1.25						
Visible mold	17.33	1.48‡	1.07–2.01	1.49‡	1.09–2.01	7.74			
Dog	19.72	1.24‡	0.88–1.72	1.26‡	0.90–1.72	4.87			
Cat	2.26	0.86‡	0.30–1.93						
Bird	3.55	1.14‡	0.53–2.13						
ETS	43.47	1.02‡	0.77–1.34						
ETS avoidance	6.66	1.59‡	1.01–2.46	1.60‡	1.03–2.47	3.61			
Pet avoidance	8.47	3.27‡	2.16–4.84	3.21‡	2.13–4.73	14.98			
Any environmental factor‖	89.56			2.02	1.23–3.65	47.25	1.80	1.08–3.23	28.04

*All ORs are adjusted for sex, age, education, family income, and smoking status.
†Adjusted for other parental atopic factors.
‡Adjusted for other home environmental factors.
§Reduced regression model.
‖Only including factors listed in the reduced regression model.

explanation could be that incense and carpet use might be reduced by families with current asthmatics, which might provide protective effects for asthma through selection mechanisms, especially in cross-sectional study. We decided not to include them as covariates in further analysis because inclusion of these factors might have given significant associations, but they would have been difficult to interpret for causality.

Indoor dampness problems have been related to increased risk of asthma-related symptoms in adults.[11,24,25,29] A study[30] of day-care workers in Taiwan demonstrated an increased risk of wheezing in relation to visible mold (OR, 1.39) and water damage (OR, 1.32). Another Taiwanese study[31] of office workers showed a similar OR for chest tightness. Home dampness may favor the presence of house-dust mites. Verhoeff et al[32] reported that visible mold and water damage were associated with increased sensitization to both dust mites and mold. Reported visible mold and water damage are useful indicators for home dampness. In our study, visible mold on walls at home was found to be independently associated with the occurrence of asthma symptoms in adulthood after controlling for other home environmental exposures (Table 3), which was consistent with other population-based studies from Sweden[24] and United States.[25]

In Taiwan, cockroaches and dust mites are popular allergens for asthmatics. Lin et al[33] noted that sensitization to cockroach allergens and house dust mites was associated with lower pulmonary functions. In the United States, Litonjua et al[34] demonstrated that indoor dust concentrations of cockroach allergen were significantly associated with recurrent asthmatic wheezing. Rosenstreich et al[35] also showed that exposure to high levels of cockroach allergen in the home was strongly associated with increased hospitalizations and other asthma morbidity among asthmatics. From our preliminary study,[28] visible

Table 4—*Joint Effects Between Home Environment and Parental Atopy on New-Onset Adulthood Asthma*

Variables	New-Onset Adulthood Asthma, %	No Asthma, %	OR*	95% CI
Home environment (−)/parental atopy (−)	0.46	98.54	1.00	
Home environment (+)/parental atopy (−)	0.82	97.28	1.61	0.89–3.28
Home environment (−)/parental atopy (+)	1.37	91.78	3.26	0.89–9.84
Home environment (+)/parental atopy (+)	3.45	90.97	7.32	4.01–14.99

*All ORs are adjusted for sex, age, education, family income, and smoking status.

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cockroaches at home was significantly related to the occurrence of childhood asthma and possessed the highest attributable risks (7.96% for boys and 19.27% for girls) among home environmental factors. Our data were consistent with these findings in that visible cockroaches at home was positively associated with new-onset adulthood asthma and had a PAR of 15.99% because the prevalence of exposure is very high (Table 3).

A family history of allergic diseases was associated with an increase risk of asthma, suggesting that genetic factors play a central role in the development of asthma.[9–11,36] Some genetic markers could impose susceptibility to the effects of environmental factors. Population-based studies[9–11] have found that parental asthma was also a strong determinant of asthma development in adulthood. We used a parental history of allergic diseases as a measure of the genetic propensity of asthma. Our study demonstrated that, while mutually adjusted models were applied, both parental atopy—defined as parental asthma and allergic rhinitis/atopic eczema—and home environmental factors—defined as cockroaches, visible mold on walls, dogs, or ETS or pet avoidance—were found to be significant predictors for new-onset adulthood asthma (Table 3). These two exposure categories approximately contributed equally, with PAR of 28.04% for home exposures and 31.38% for parental atopy. In a twin-family study[36] from Finland, 87% of the variation in liability to adolescent asthma was explained by genetic factors, which suggested that a family history of asthma was stronger than other risk factors. The present data were also inconsistent with our previous finding on childhood asthma that parental atopy plays the most important role rather than other indoor and outdoor environmental factors.[28] A different pathogenetic mechanism should be considered between child-onset and adult-onset asthma.[7] Further studies are warranted to identify the relative contributions of environmental stimuli and genetic propensity in various types of asthma.

When we tested the hypothesis that the joint effect of hereditary propensity to atopy, representing genetic constitution, and home exposures on the risk of adulthood asthma is greater than expected on the basis of their independent effects, we found no interaction effect between the exposure categories. The combined effect of these two factors was approximately as expected on the basis of their independent effects in the multiplicative scale (Table 4), which was consistent with a Finnish study[10] discussing the interactive effect between pet ownership and parental atopy. Parental atopy would not modify the effects of home exposures on the risk of new-onset adulthood asthma.

It is difficult to target asthma prevention efforts. Only 38.6% of the individuals with typical asthma symptoms in our analysis had a parental history of atopy, and only 9.0% of those with parental atopy had asthma symptoms. If interventions target only families with a history of atopy, then $> 60\%$ of the subjects who experience asthma symptoms will be neglected, and only 9% will potentially benefit. Home environmental exposures showed substantial effects on adulthood asthma in our study. Reduction in certain home exposures, especially visible cockroaches or mold on the wall, could prevent the occurrence of asthma symptoms considerably. It seems easier to eliminate such exposures on a national scale than to attempt to counter hereditary factors. Additional research is necessary to prove that the elimination of home environmental exposures will result in lower rates of adulthood asthma.

Our study has some limitations. Bias could also be introduced if differential changes of environmental exposures showed, such as relocation of families. Because we were unable to measure personal environmental exposures or sensitization to various allergens, such as dust mites, fungi, or cockroaches, we might have underestimated the effects of these home exposures to asthma. Another potential source of bias was in the interpretation of parental history for atopy/asthma as an indication of a genetic predisposition to adulthood asthma. Although the importance of parental history as a predictor of disease has been demonstrated,[9–11,36] not every individual in the family inherits the allergic tendency. Therefore, in the presence of a true association, misclassification of exposure that was random with respect to other study variables would weaken the observed association rather than lead to false-positive inferences. More complete personal risk factors, however, were very difficult to obtain in such a large-scale survey. Investigators decided not to try to obtain more personal information, such as childhood infections, hygiene, dietary habits and obesity, because it would have resulted in a lower participation rate and would have introduced greater bias in the study. The number of covariates was therefore limited, as in many other large-scale studies.

Subjects with asthma symptoms may be more prone to report their exposures, especially if the exposure has been discussed in association with asthma. Although information about exposures was collected in a uniform manner, we could not fully exclude the possibility that case subjects and controls provided differential information. However, this study was conducted in a random population sample in a region where no specific concerns over exposures in the home environment had been raised. The questions about exposures in the home environment

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were embedded in a questionnaire including questions on respiratory symptoms. This does not exclude the possibility that over-reporting can to some extent be responsible for the associations found. On the other hand, there is a possibility of underreporting of exposures among the cases due to avoidance, as reporting of exposures may be influenced by present habits.

In conclusion, we identified a number of hereditary and home environmental factors associated with the occurrence of asthma symptoms among 26 to 50-year-old Taiwanese adults. Home exposures and parental atopy approximately contributed equally but did not show an interactive effect on new-onset asthma. Data from this study may serve as baseline data for the comparison of future prevalence surveys of adulthood asthma in Taiwan. Further efforts to improve the problem of underdiagnosis are also warranted. Exposure to home environmental factors increased the risk of asthma in adults regardless of the coexisting hereditary factors. The present findings suggest that public health policy for eliminating certain home exposures are needed, which could have large effects not only on public health but also on medical costs in Taiwan.

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