

Home Exposures to Environmental Tobacco Smoke and Allergic Symptoms among Young Children in Singapore

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Key Words

Asthma • Allergy • Preschool children • Environmental tobacco smoke • Home exposures

Abstract

Background: Research relating environmental tobacco smoke (ETS) exposures have focused on childhood asthma. There have been fewer studies with conflicting results performed on associations of ETS exposures with allergic symptoms. We are interested to see if ETS exposures in the homes are associated with allergic symptoms among preschool children in Singapore where public smoking is banned.

Methods: A cross-sectional study adopting an expanded and modified ISAAC (International Study on Asthma and Allergies in Childhood) questionnaire for the evaluation of asthma and allergies was conducted on 6,794 children attending 120 randomly selected child care centers. Specific information on demographics and ETS exposures was obtained. Adjusted prevalence ratios (PRs) and 95% confidence intervals (CIs) were determined using Poisson multivariate regression with a log-link function and robust variance estimates as recommended for cross-sectional studies. **Results:** The response proportion was 70.0%, and 4,759 children from 97 centers participated. After adjusting for covariates, it was

found that home ETS exposure was associated with increased risks of current symptoms of rhinitis (PR 1.23; 95% CI 1.01–1.50) and rhinoconjunctivitis (PR 1.79; 95% CI 1.26–2.54). These associations followed dose-response trends with respect to number of cigarettes smoked or smokers in the homes. Home ETS exposures were also associated with higher PRs of wheeze, nocturnal cough and doctor-diagnosed asthma. Compared with paternal smoking, higher risks of the above outcomes were found for maternal smoking. **Conclusion:** Home ETS exposure is a risk factor associated with rhinitis and asthma among preschool children.

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Introduction

Environmental tobacco smoke (ETS) is a highly prevalent respiratory irritant, and the adverse effects of its exposure mirror those associated with active smoking [1]. There is convincing evidence that exposure to ETS is causally associated with an increased prevalence of asthma and wheeze in children and that exposure to ETS aggravates asthma in childhood [1, 2].

While most of the works relating ETS exposures have focused on asthma and wheeze in children, there have

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been very few studies performed on the associations with rhinitis and eczema symptoms. Studies relating ETS exposure and rhinitis symptoms mostly showed no or negative associations. Raheison et al. [3] documented that there was no association between rhinitis and parental smoking. In a cross-sectional study among 6-year-old Japanese children [4], ETS exposure was related to a significantly lower prevalence of allergic rhinitis (prevalence odds ratio, POR, 0.83; 95% CI 0.77–0.89). The review by Strachan and Cook [5] judged that there was no evidence of a causal relationship with allergic rhinitis. In their review, only 1 cross-sectional survey from Turkey reported a marginally statistically significant positive effect for clinical allergic rhinitis. Recently, Biagini et al. [6] reported that ETS exposure was associated with an increased risk of developing allergic rhinitis at age 1 (OR 2.7; 95% CI 1.0–6.8) and rhinitis symptoms during the first year (OR 1.9; 95% CI 1.1–3.2) for US infants. Stazi et al. [7] reported higher odds of rhinitis symptoms (POR 2.2; 95% CI 1.2–4.1) with maternal smoking for Italian children.

For eczema symptoms, Raheison et al. [3] reported no association between eczema and parental smoking. The protective effects of ETS exposures to allergic symptoms were observed in a Swedish study among 7,844 3-year-old children [8]; the authors reported reduced effect estimates for atopic eczema (POR 0.8; 95% CI 0.7–0.9) obtained for children exposed in late pregnancy compared with those that are nonexposed. Similarly, the smoking associations with eczema were significantly low (POR 0.8; 95% CI 0.7–0.9) in a study among South Australian preschool children [9]. Although the review by Strachan and Cook [5] primarily deals with the association between allergic sensitization and ETS exposure, this may be different from rhinitis and eczema symptoms. Indeed, Biagini et al. [6] and Stazi et al. [7] have demonstrated that there was no association between ETS and sensitization despite showing increased odds of rhinitis symptoms with ETS exposure.

In Singapore, public smoking is prohibited due to legislative ruling, which could have implications on smoking in the homes. Although some studies associating ETS exposures with allergic symptoms have clearly differentiated whether the exposures occur in the homes or outdoors, others have not [10, 11]. This ambiguity may result in exposure misclassification which could explain the conflicting evidences found in the studies above. The aim of this study is to investigate the associations of ETS exposures in the homes with allergic symptoms among preschool children in Singapore.

Materials and Methods

A cross-sectional study adopting an expanded and modified questionnaire for the evaluation of asthma and allergic symptoms was conducted. From the database of the Ministry of Community Development, Youth and Sports including 687 child care centers (CCCs) in Singapore, 120 CCCs (18%) were randomly selected. The questionnaires were distributed to the preschool children aged between 1.5 and 6 years based on the CCC registers and collected by the respective class teachers after up to 3 reminders. The parents or guardians of the children attending these CCCs were the respondents for the questionnaire survey. For asthma and allergic symptoms, the written questionnaire of the International Study of Asthma and Allergies in Childhood (ISAAC) was used [12]. Definitions of health outcomes are presented in table 1.

Exposure assessment of ETS was based on questionnaire information on exposure in the homes. The primary variable of interest was 'ETS', defined as daily smoking in the homes in the presence of children by either mother or father. Other ETS-related variables were also determined from the questionnaires which include the following: maternal ETS, mother who smokes in the home daily in the presence of children; paternal ETS, father who smokes in the home daily in the presence of children; both ETS, mother and father who smoke in the home daily in the presence of children; maternal ETS number, number of cigarettes which the mother smokes in the home each day (0, 1–10, >11 cigarettes); paternal ETS number, number of cigarettes which the father smokes in the home each day (0, 1–10, >11 cigarettes); and ETS ppl., number of people who smoke in the home daily (0, 1, 2 or more).

For potential covariates, the questionnaire included questions on the child's age, gender (male/female), ethnicity (Chinese/Indian/Malay/others), food allergy (doctor-diagnosed food allergy), preterm birth (born at least 1 month before due date), breastfeeding (breastfed for at least 3 months), maternal and paternal atopy (mother or father ever diagnosed with asthma/allergic rhinitis/atopic dermatitis), pet exposure (presence of cats or dogs or furry animals), housing type (public apartments/private apartments/single or terrace houses), socioeconomic status (SES), respiratory infections (usually have a cough with cold or flu in the past 12 months) [13], home dampness (visible damp stains or molds on the floor, walls or ceilings), home wall paper (yes/no), home traffic density (light/medium/heavy) and home air conditioning (yes/no). The SES of the children was assessed by questions on household income [14]. Since the children attend CCCs and are exposed to potential confounders in that environment, we evaluated CCC dampness and ventilation exposures via inspection [15].

All statistical analyses were conducted using the SAS software version 9.1. The data were initially analyzed by cross-tabulation and evaluated where appropriate using the χ^2 or Fisher's exact tests. In comparing the prevalence of asthma and allergies among different ETS exposure groups, computation of the prevalence ratios (PRs, relative risk) and 95% confidence intervals (95% CIs) using Poisson regression with robust variance estimates to adjust for overdispersion in the data was performed as recommended for cross-sectional studies [16, 17]. Poisson regressions were calculated utilizing the GENMOD procedure with a logarithmic link function. Potential covariates were included in the final multivariate models as covariates if they satisfied a change in estimate

Table 1. Prevalence of personal characteristics and health outcomes of the 4,759 children

Outcomes/characteristics	Description ^a	Children	Prevalence %	Response proportion ^b , %
<i>Personal characteristics</i>				
Mean age $\pm$ SD, years		4.08 $\pm$ 1.24		
Gender	Male	2,474	53.6	97.4
	Female	2,138	46.4	
Race	Chinese	3,778	81.9	97.4
	Malay	393	8.5	
	Indian	233	5.1	
	Others	209	4.5	
Type of housing	Public apartments	3,832	82.7	97.9
	Private apartments	580	12.5	
	Single/terrace houses	221	4.8	
SES	Total monthly income ^c <SGD 2,000 ^d	804	18.0	94.4
	Total monthly income SGD 2,000 to <4,000	1,463	32.7	
	Total monthly income SGD 4,000 to <6,000	967	21.6	
	Total monthly income >SGD 6,000	1,236	27.7	
Food allergy	Has a doctor diagnosed this child to be allergic to any food?	264	5.7	98.0
Maternal atopy	Has the mother ever been diagnosed with asthma/allergic rhinitis/atopic dermatitis?	626	14.1	97.1
Paternal atopy	Has the father ever been diagnosed with asthma/allergic rhinitis/atopic dermatitis?	557	12.6	96.7
Preterm birth	Was the child born at least 1 month before his/her due date (premature)?	414	8.9	98.0
Breastfeeding	Was this child breastfed for at least 3 months?	1,061	32.4	95.7
<i>Asthma and allergic outcomes</i>				
Wheeze	Has your child had wheezing or whistling in the chest in the last 12 months?	749	15.8	99.5
Doctor-diagnosed asthma	Has your child ever had asthma?	206	4.4	95.8
Nocturnal cough	In the past 12 months, has your child had a dry cough at night apart from a cough associated with a cold or chest infection?	1,280	28.2	95.9
Rhinitis	In the past 12 months, has your child ever had a problem with sneezing or a runny or blocked nose when he/she did not have a cold or a flu?	1,214	25.6	99.5
Rhinoconjunctivitis	In the past 12 months, has this nose problem been accompanied by itchy or watery eyes?	367	7.8	99.5
Eczema	Has your child had an itchy rash which was coming and going for at least 6 months in the past 12 months?	602	12.7	99.5
Flexural rash	Has this itchy rash affected the folds of the elbows or behind the knees or in front of the ankles or under the buttocks or around the neck, cheeks, ears or eyes in the past 12 months?	520	11.0	99.5

^a All symptoms are within the past year except for asthma; ^b response proportion to each item; ^c before tax; ^d USD 1 = SGD 1.59 in June 2006.

or significance test of the covariate criterion. For change in estimate, the covariate will be included in the multivariate model if the crude and adjusted PRs differ by $\geq 10\%$ [18]. For significance test of the covariate, the covariate will be included if the adjusted estimate of effect has a p value ≤ 0.20 [19]. We assessed the goodness of fit of the final models by calculating the deviance defined as twice the difference between the maximum achievable log likelihood and the log likelihood at the maximum likelihood esti-

mates of the regression parameters. Test results showed that the null hypothesis, i.e. that the fitted final models are appropriate, was not rejected ($p > 0.05$). For purposes of comparison with existing literature (see Discussion), we computed the adjusted POR using logistic regression for the associations between wheeze and ETS variables. Statistical significance was assessed using a 2-sided test at a 5% level of significance.

Table 2. Prevalence (%) of childhood asthma and allergies by ETS variables in the exposed and reference groups

	Maternal ETS		Paternal ETS		Both ETS		ETS	
	no	yes	no	yes	no	yes	no	yes
Wheeze								
Yes (749)	97.6 (718)	2.4 (18)	88.1 (650)	11.9 (88)	98.0 (722)	2.0 (15)	83.9 (617)	16.1 (118) ^a
No (3,985)	98.0 (3,849)	2.0 (78)	89.9 (3,513)	10.1 (396)	98.3 (3,860)	1.7 (67)	87.0 (3,393)	13.0 (509)
Doctor-diagnosed asthma								
Yes (348)	97.1 (332)	2.9 (10)	85.7 (293)	14.3 (49) ^a	97.1 (332)	2.9 (10)	82.5 (282)	17.5 (60) ^a
No (4,188)	98.0 (4,055)	2.0 (83)	89.9 (3,706)	10.1 (418)	98.3 (4,069)	1.7 (69)	86.8 (3,571)	13.2 (544)
Nocturnal cough								
Yes (1,280)	97.1 (1223)	2.7 (34)	87.4 (1,095)	12.6 (158) ^a	97.8 (1,229)	2.2 (28)	82.7 (1,037)	17.3 (217) ^a
No (3,262)	98.1 (3,168)	1.9 (61)	90.3 (2,910)	9.7 (312)	98.4 (3,177)	1.6 (53)	87.9 (2,823)	12.1 (389)
Rhinitis								
Yes (1,214)	97.4 (1,164)	2.6 (31) ^a	88.4 (1,054)	11.6 (139)	97.6 (1,168)	2.4 (29) ^a	84.3 (1,006)	15.7 (187) ^a
No (3,520)	98.1 (3,403)	1.9 (65)	90.0 (3,104)	10.0 (345)	98.5 (3,414)	1.5 (53)	87.2 (3,004)	12.8 (440)
Rhinoconjunctivitis								
Yes (367)	96.9 (349)	3.1 (11)	86.0 (313)	14.0 (51) ^a	97.5 (352)	2.5 (9)	80.7 (272)	19.3 (70) ^a
No (4,367)	98.0 (4,218)	2.0 (85)	89.9 (3,850)	10.1 (433)	98.3 (4,230)	1.7 (73)	87.0 (3,718)	13.0 (557)
Eczema								
Yes (602)	98.3 (585)	1.7 (10)	91.3 (543)	8.7 (52)	98.5 (586)	1.5 (9)	88.0 (523)	12.0 (71)
No (4,132)	97.9 (3,982)	2.1 (86)	89.3 (3,620)	10.7 (432) ^a	98.2 (3,999)	1.8 (73)	86.2 (3,487)	13.8 (5,560)
Flexural rash								
Yes (520)	98.6 (507)	1.4 (7)	92.2 (474)	7.8 (40)	98.8 (508)	1.2 (6)	89.1 (458)	10.9 (56)
No (4,214)	97.9 (4,060)	2.1 (89)	89.3 (3,689)	10.7 (444) ^a	98.2 (4,074)	1.8 (76)	86.2 (3,552)	13.8 (571)

Figures in parentheses are number of children. The total number and percentages may not add up owing to some missing information.

^a χ^2 or Fisher's exact test; $p < 0.05$ comparing prevalence of symptoms by exposure to ETS variables.

Results

From the 120 CCCs randomly selected, 97 agreed to participate (80.8%). From the total 6,794 children, 4,759 returned the questionnaire (response proportion 70.0%). Table 1 shows the prevalence of various symptoms among the children, with 4.4% of the children having doctor-diagnosed asthma. Among asthma and allergic symptoms, nocturnal cough (28.2%) and rhinitis (25.6%) were the most commonly reported symptoms; 15.8% had wheezing and 11.0% had eczema.

Table 2 shows the prevalence of wheeze, asthma, nocturnal cough, rhinitis, rhinoconjunctivitis, eczema and flexural rash in the exposed and reference groups. In general, the prevalence of children exposed to paternal smoking (10.2%) is higher than that of children exposed to maternal smoking (2.0%). The occurrences of any and both parents smoking in the presence of their children appear in 13.2 and 1.7% of the population, respectively.

A significantly higher prevalence ($p < 0.05$) of rhinitis was observed for maternal and both parents smoking while higher prevalences were noted for asthma, nocturnal cough and rhinoconjunctivitis for paternal smoking

(table 2). Lower prevalences of eczema and flexural rash were observed for paternal smoking ($p < 0.05$). Significantly, higher prevalences were found for asthma and rhinitis symptoms with children exposed to any of the parents smoking ($p < 0.05$).

After adjustment for covariates (table 3), increased risks of wheeze, doctor-diagnosed asthma, nocturnal cough, rhinitis and rhinoconjunctivitis were observed among children exposed to ETS. Risks for these health outcomes were higher for children exposed to maternal ETS compared with paternal ETS. High adjusted PRs were found for the associations of rhinitis and rhinoconjunctivitis among children exposed to either maternal and/or ETS variables (1.92–1.88 and 2.24–2.02). No significant associations were found for any ETS variables with eczema or flexural rash.

The effects of the estimated number of cigarettes smoked in the homes on symptoms of asthma and rhinitis among the children are given in table 4. Adjusted PRs for asthma and rhinitis parameters increased significantly with increasing number of cigarettes which the mothers and fathers smoke in the homes in a dose-response manner. Compared with unexposed children, exposed

Table 3. Associations of childhood asthma and allergies with ETS variables

	Maternal ETS		Paternal ETS		Both ETS		ETS	
	no	yes	no	yes	no	yes	no	yes
Wheeze ¹	1.00	1.58 (1.09–2.29)	1.00	1.14 (1.02–1.28)	1.00	1.45 (0.94–2.25)	1.00	1.51 (1.23–1.85)
Doctor-diagnosed asthma ²	1.00	1.37 (0.85–2.23)	1.00	1.08 (0.90–1.29)	1.00	1.27 (0.73–2.20)	1.00	1.35 (1.01–1.80)
Nocturnal cough ³	1.00	1.17 (0.90–1.51)	1.00	1.12 (1.06–1.19)	1.00	1.15 (0.86–1.53)	1.00	1.32 (1.15–1.51)
Rhinitis ⁴	1.00	1.92 (1.46–2.52)	1.00	1.03 (0.91–1.17)	1.00	1.88 (1.37–2.56)	1.00	1.23 (1.01–1.50)
Rhinoconjunctivitis ⁵	1.00	2.24 (1.29–3.92)	1.00	1.07 (0.85–1.34)	1.00	2.02 (1.05–3.92)	1.00	1.79 (1.26–2.54)
Eczema ⁶	1.00	1.14 (0.72–1.82)	1.00	0.94 (0.78–1.13)	1.00	1.05 (0.62–1.78)	1.00	0.97 (0.75–1.25)
Flexural rash ⁷	1.00	1.19 (0.71–1.99)	1.00	0.97 (0.80–1.19)	1.00	1.14 (0.64–2.01)	1.00	0.93 (0.70–1.24)

Data are adjusted PRs, with 95% CIs in parentheses.

¹ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, food allergy, respiratory infections, home dampness, home air conditioning, home pets, CCC dampness and CCC ventilation strategy.

² Adjusted for age, gender, race, familial atopy, breastfeeding, preterm birth, food allergy, respiratory infections and home wall paper.

³ Adjusted for age, race, familial atopy, breastfeeding, preterm birth, food allergy, home dampness, respiratory infections and CCC dampness.

⁴ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, food allergy, respiratory infections, home dampness,

home air conditioning, home wall paper, home carpet, home traffic density, CCC ventilation strategy and CCC dampness.

⁵ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, preterm birth, food allergy, respiratory infections, home dampness, home air conditioning, home traffic density, CCC ventilation strategy and CCC dampness.

⁶ Adjusted for SES, housing type, food allergy, familial atopy, breastfeeding, respiratory infections, home wall paper and CCC dampness.

⁷ Adjusted for SES, housing type, food allergy, familial atopy, breastfeeding, respiratory infections, home wall paper and CCC ventilation strategy.

Table 4. Associations of childhood asthma and allergies with intensity of ETS exposures

	Number of cigarettes which the mother smokes in the home			Number of cigarettes which the father smokes in the home			Number of people who smoke in the home		
	None	1–10 cigarettes	>11 cigarettes	None	1–10 cigarettes	>11 cigarettes	None	1 smoker	>1 smoker
Wheeze ¹	1.00	1.37 (1.03–1.82)	1.51 (0.88–2.60)	1.00	1.07 (0.86–1.32)	1.57 (1.21–1.90)	1.00	1.12 (0.98–1.28)	1.26 (0.97–1.63)
Doctor-diagnosed asthma ²	1.00	1.38 (0.61–3.15)	1.89 (0.52–6.86)	1.00	1.31 (0.83–2.06)	1.78 (1.06–2.99)	1.00	1.14 (0.94–1.38)	1.30 (0.89–1.90)
Nocturnal cough ³	1.00	1.03 (0.95–1.12)	1.06 (0.90–1.26)	1.00	1.30 (1.02–1.66)	1.42 (1.04–1.96)	1.00	1.13 (1.04–1.23)	1.29 (1.09–1.52)
Rhinitis ⁴	1.00	1.57 (0.94–2.64)	3.18 (1.05–6.74)	1.00	1.25 (0.94–1.65)	1.49 (1.06–2.11)	1.00	1.07 (0.89–1.28)	1.51 (1.16–1.96)
Rhinoconjunctivitis ⁵	1.00	1.12 (0.90–1.38)	1.24 (0.81–1.91)	1.00	1.28 (0.88–2.63)	1.57 (0.92–2.70)	1.00	1.38 (1.08–1.76)	1.90 (1.16–3.10)
Eczema ⁶	1.00	1.03 (0.90–1.18)	1.06 (0.81–1.40)	1.00	0.98 (0.68–1.42)	0.96 (0.46–2.01)	1.00	1.03 (0.89–1.20)	1.07 (0.79–1.45)
Flexural rash ⁷	1.00	1.04 (0.89–1.21)	1.08 (0.80–1.46)	1.00	1.02 (0.69–1.52)	1.05 (0.48–2.30)	1.00	1.05 (0.89–1.24)	1.10 (0.79–1.55)

Data are adjusted PRs, with 95% CIs in parentheses.

¹ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, food allergy, respiratory infections, home dampness, home air conditioning, home pets, CCC dampness and CCC ventilation strategy.

² Adjusted for age, gender, race, familial atopy, breastfeeding, preterm birth, food allergy, respiratory infections and home wall paper.

³ Adjusted for age, race, familial atopy, breastfeeding, preterm birth, food allergy, home dampness, respiratory infections and CCC dampness.

⁴ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, food allergy, respiratory infections, home dampness, home air con-

ditioning, home wall paper, home carpet, home traffic density, CCC ventilation strategy and CCC dampness.

⁵ Adjusted for age, gender, race, SES, housing type, familial atopy, breastfeeding, preterm birth, food allergy, respiratory infections, home dampness, home air conditioning, home traffic density, CCC ventilation strategy and CCC dampness.

⁶ Adjusted for SES, housing type, food allergy, familial atopy, breastfeeding, respiratory infections, home wall paper and CCC dampness.

⁷ Adjusted for SES, housing type, food allergy, familial atopy, breastfeeding, respiratory infections, home wall paper and CCC ventilation strategy.

children were at an increased risk of wheeze (PR 1.57; 95% CI 1.21–1.90), asthma (PR 1.78; 95% CI 1.06–2.99), nocturnal cough (PR 1.42; 95% CI 1.04–1.96), rhinitis (PR 1.49; 95% CI 1.06–2.11) and rhinoconjunctivitis (PR 1.57; 95% CI 0.92–2.70) if they were exposed to fathers smoking more than 11 cigarettes daily. The risks were higher for children exposed to mothers smoking more than 11 cigarettes daily: wheeze (PR 1.51; 95% CI 0.88–2.60), asthma (PR = 1.89; 95% CI: 0.52–6.86), nocturnal cough (PR 1.06; 95% CI 0.90–1.26), rhinitis (PR 3.18; 95% CI 1.50–6.74) and rhinoconjunctivitis (PR 1.24; 95% CI 0.81–1.91). The strength of the association between ETS and asthma and rhinitis symptoms also increased as the number of household smokers increased (table 4).

Discussion

The results of this study provide strong evidence of an association between ETS exposures within homes with asthma and rhinitis symptoms among Singaporean pre-school children attending CCCs aged 1.5–6 years. Despite that the data procured were not substantiated by clinical procedures such as skin prick testing, exercise challenge and lung function tests, previous surveys have found that the ISAAC written questionnaire was reasonably sensitive and specific for bronchial hyperresponsiveness [12, 20]. Home ETS exposure was also assessed using questionnaire responses considering exposure status based on questionnaire responses which have been found to provide reasonably valid estimates of exposure in studies elsewhere [1, 21–23]. The effects of potential confounders other than the exposures of interest were controlled in the multiple regression analysis using Poisson regression with a log-link function [17]. It has been reported that for cross-sectional data, estimating the OR by logistic regression may cause a biased perception if interpreted as a relative risk when the disease under investigation is not rare [16, 17].

ETS is known to contain a complex concoction of compounds which can be irritants, agents of inflammation and respiratory toxins that adversely affect immune function, lung growth, airway function and the respiratory epithelium broadly through several pathophysiologic pathways [1]. Investigations on the effect of ETS have been carried out using epidemiologic, murine models as well as human experimental studies. While a review of earlier epidemiologic studies on allergies has been inconclusive [3–7], the latter two types of studies can explain the findings of this present investigation. It is assumed

that ETS acts not as an allergen but rather as an adjuvant which facilitates sensitization. Early researchers have hypothesized that ETS effects are primarily mechanical, i.e. it may damage the respiratory epithelium, and this damage may itself increase the likelihood that allergens will reach immunoreactive cells [24]. Recent experimental studies on humans in an environmentally controlled chamber revealed that ETS can interact with allergen to modulate immune responses in the upper airway [25]. They demonstrated that short-term exposure of allergic subjects to ETS led to an enhanced allergic response characterized by specific allergic antibody (IgE) production against an inhaled protein allergen and the local formation of a Th2-type cytokine milieu (increased IL-4, IL-5 and IL-13 and decreased interferon- γ production), characteristic of an active allergic response.

In keeping with the above, this present study provides new evidence of statistically significant associations of exposure to ETS in the homes with increased risk of rhinitis and rhinoconjunctivitis symptoms in tropical pre-school children. Further, the high adjusted PRs suggest that the likelihood of exposure-symptoms associations cannot be explained by error, bias or confounding is greater. Also, the dose-response relation was evident in that the risk of these symptoms increased as the number of household smokers and cigarettes increased. Now, epidemiologic literature on causality has focused on potential causality using a set of criteria recommended for its assessment [1, 26, 27]. Indeed, this study has fulfilled 3 important criteria: strength of association, dose-response relationship and biologic plausibility.

The literature on the relation of ETS exposure and asthma symptoms gives conflicting results with regard to sex differences in susceptibility [28], with some studies showing a greater effect of ETS exposures on boys. In this study, we did not find any interaction between ETS exposure and sex in the health outcomes association evaluated in each multivariate model. Further, susceptibility to ETS exposure has been found for children with a parental history of asthma and childhood allergy, which are strong risk factors for asthma [29, 30]. In our study, the association between ETS exposure and the health outcomes was influenced by parental history (table 5), where a higher burden of most symptoms was found for children with parental atopy. Similar to our findings, Agabiti et al. [31] reported that current smoking was related to asthma among children in the presence of parental asthma history, but only to wheeze in the absence of parental history. Other data addressing the possible interaction of ETS exposures and parental atopy in relation to allergic symptoms are rare.

Table 5. Associations of childhood asthma and allergies with ETS exposure stratified according to parental atopy

	Parental atopy (no)		Parental atopy (yes)	
	ETS (no)	ETS (yes)	ETS (no)	ETS (yes)
Wheeze ¹	1.00	1.65 (1.27–2.15)	1.00	1.27 (0.91–1.75)
Doctor-diagnosed asthma ²	1.00	1.32 (0.87–1.99)	1.00	1.45 (0.94–2.15)
Nocturnal cough ³	1.00	1.29 (1.08–1.54)	1.00	1.36 (1.11–1.68)
Rhinitis ⁴	1.00	1.26 (0.99–1.61)	1.00	1.40 (1.04–1.90)
Rhinoconjunctivitis ⁵	1.00	1.51 (0.92–2.49)	1.00	1.97 (1.23–3.17)
Eczema ⁶	1.00	0.78 (0.53–1.14)	1.00	1.25 (0.88–1.77)
Flexural rash ⁷	1.00	0.70 (0.45–1.10)	1.00	1.32 (0.91–1.93)

Data are adjusted PRs, with 95% CIs in parentheses.

¹ Adjusted for age, gender, race, SES, housing type, breastfeeding, food allergy, respiratory infections, home dampness, home air conditioning, home pets, CCC dampness and CCC ventilation strategy.

² Adjusted for age, gender, race, breastfeeding, preterm birth, food allergy, respiratory infections and home wall paper.

³ Adjusted for age, race, breastfeeding, preterm birth, food allergy, home dampness, respiratory infections and CCC dampness.

⁴ Adjusted for age, gender, race, SES, housing type, breastfeeding, food allergy, respiratory infections, home dampness, home air con-

ditioning, home wall paper, home carpet, home traffic density, CCC ventilation strategy and CCC dampness.

⁵ Adjusted for age, gender, race, SES, housing type, breastfeeding, preterm birth, food allergy, respiratory infections, home dampness, home air conditioning, home traffic density, CCC ventilation strategy and CCC dampness.

⁶ Adjusted for SES, housing type, food allergy, breastfeeding, respiratory infections, home wall paper and CCC dampness.

⁷ Adjusted for SES, housing type, food allergy, breastfeeding, respiratory infections, home wall paper and CCC ventilation strategy.

The prevalence of maternal and paternal smoking found in this study was 4.1 and 27.5%, respectively. This is comparable with an earlier survey in Singapore reporting prevalences of smoking women at 3.5% and of men at 24.2% [32]. Cook and Strachan [33] reported a summary OR for maternal ETS and wheezing of 2.0 (95% CI 1.7–2.3) which is slightly higher than the calculated POR estimate of this study (for comparative purposes) of 1.8 (95% CI 1.0–3.2), but the confidence intervals show considerable overlap. On the other hand, the summary OR of paternal ETS was reported to be 1.2 (95% CI, 0.9 to 1.5), while this study was higher at 1.6 (95% CI 1.2–2.2), although an overlap of confidence intervals could also be noted. For ETS, they reported a summary OR of 1.5 (95% CI 1.3–1.8) that is comparable with the POR estimate of this study of 1.7 (95% CI 1.3–2.1). In general, the effect estimates of the associations of ETS with wheeze reported in this study were comparable with the estimates via metaanalyses provided by Strachan and Cook [33].

In this study, maternal ETS confers a higher risk of asthma and rhinitis symptoms of children compared with paternal ETS. There are 3 possible reasons for this observation: (1) it reflects the higher ETS exposure since mothers have more intense contact with their children in homes compared with fathers; (2) nonsmoking mothers may exert some influence in protecting the child against ETS exposure due to the father's smoking, and (3) the im-

portance of prenatal exposure as an independent variable in developing symptoms after the child has been born [34]. Depending on which symptom is considered, the risk was both lower and higher for both ETS when compared with just maternal or paternal ETS. The reasons for this are not clear and could be partly attributed to the behavior of the parents [35].

The World Health Organization estimates around half of the children in the world to be exposed to ETS, mostly in their homes [11]. As children within this age group in Singapore were most likely not exposed in the CCCs or in the public due to the strict legislative ban on public smoking and regular health promotion campaigns, home exposure is possibly the dominant source of ETS. Reducing home ETS exposure may offer the most promising opportunity for reducing morbidity because this risk factor is potentially modifiable. Hence, reducing the burden of asthma and allergic symptoms may require a stronger focus on smoking cessation in the homes where, if effective, one could gain significant public health benefits. Indeed, this underlines the importance of sending a message about the health risks of ETS exposure in the homes.

In a political setting where public smoking is banned, assessments of ETS exposures and symptoms by parental reports can have some implications on the reporting bias. However, we do not believe that bias from differential

misclassification of ETS exposure explains our findings. It can be expected that more parents would not want to be seen as flouting the law and thus report that they are smoking within the privacy of their homes. Under these conditions, it can be anticipated that the proportions of exposure misclassification are likely to be the same (non-differential) for symptomatic children as for the healthy children. In keeping with this, it is also likely that the parents of symptomatic children would underreport symptoms which would bias our findings toward the null. However, if this is the case, it would not account for our findings concerning the dose-response relationships between ETS exposure intensity and asthma and rhinitis symptoms.

Although we have achieved 70% response, even a very high proportion may not preclude bias if the nonparticipation subjects differ from the participating ones. From the randomly selected nonrespondents ($n = 220$), more were somewhat likely to be males (58 vs. 53%) and Chinese (87.0 vs. 81%). As a greater odds of asthma and allergies in young Singapore children is associated with males and Chinese [14], the PRs associated with gender and race might be underestimated causing a less than adequate adjustment for these confounders in the analyses. However, we believe this is likely to be minimal because in many cases, parents did not participate due to their inability to find time to complete the questionnaire. Further, community surveys in our population have reported that the non-Chinese is more likely to consent to surveys than the Chinese. This suggests that poorer responses are a reflection of cultural and socioeconomic characteristics rather than a health-related bias [36].

The strengths of this study include its population-based sampling strategy and large sample size. Since more than 90% of the children in Singapore attend CCCs [36], our results are likely to be representative of the country. This study specifically addresses exposures to ETS in the homes and whether it occurs in the presence of children. This design eliminates confounding issues of lower exposures by higher ventilation rates (smoking outdoors) and avoidance behavioral patterns [10, 37] which might lead to exposure misclassification. We lack information to assess the magnitude of misclassification of symptom status from parental recall, awareness of potential hazardous environmental influences or medical practice which would lead to preferential reporting of symptoms, and therefore, stronger associations with the exposures of interest. Nevertheless, this was unlikely because the study was conducted without any specific concerns about home exposures [15]. Furthermore, it is unlikely that our findings are the result of a spurious association that arose from consistent variations in medical practice across the island, as the quality of medical practice tends to be uniform in this small island republic.

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