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The 2016 Melbourne thunderstorm asthma epidemic: risk factors for severe attacks requiring hospital admission

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Contributions

FT conceived and designed the study, assisted by CM, MS, JAD, JL and MH. All the authors collected the data. FT, MH, JL, NHS, SP, and BE collated and analysed the data. MH wrote the first draft. All the authors wrote the manuscript and approved the final version.

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ABSTRACT

Background: The world's most catastrophic and deadly thunderstorm asthma epidemic struck Melbourne, Australia, on November 21st 2016.

Objective: Among thunderstorm-affected patients presenting to emergency rooms (ERs), we investigated risk factors predicting severe attacks requiring admission to hospital.

Methods: Thunderstorm-affected patients were identified from ER records at the eight major Melbourne health services and interviewed by telephone. Risk factors for hospital admission were analyzed.

Results: We interviewed 1435/2248 (64%) of thunderstorm-affected patients, of whom 164 (11.4%) required hospital admission. Overall, rhinitis was present in 87%, and current asthma in 28%. Odds for hospital admission were higher with increasing age (odds ratio 1.010, 95% CI 1.002, 1.019) and among individuals with current asthma [(adjusted odds ratio (aOR) 1.87, 95% CI 1.26, 2.78]. Prior hospitalization for asthma in the previous 12 months further increased the odds for hospital admission (aOR 3.16, 95% CI 1.63, 6.12). Among patients of Asian ethnicity, the odds for hospital admission were lower than for non-Asian patients (aOR 0.59, 95% CI 0.38, 0.94), but higher if born in Australia (OR=5.42, 95% CI 1.56, 18.83).

Conclusions: In epidemic thunderstorm asthma patients who presented to the ER, higher odds for hospital admission among patients with known asthma were further amplified by recent asthma admission, highlighting the vulnerability conferred by suboptimal disease control. Odds for hospital admission were lower in Asian patients born overseas, but higher in Asian patients born locally, than in non-Asian patients; these observations suggest susceptibility to severe thunderstorm asthma may be enhanced by gene-environment interactions.

Key words

Asthma; thunderstorm; epidemic; emergency; hospitalization; ethnicity; Asian; Indian; rhinitis.

Abbreviations

ER	emergency room
SD	standard deviation
aOR	adjusted odds ratio
GP	general practitioner
ACT	Asthma Control Test
ARIA	Allergic Rhinitis and its Impact in Asthma

Introduction

Epidemic thunderstorm asthma occurs when a thunderstorm triggers asthma exacerbations in multiple individuals. Such events strike unexpectedly and with devastating suddenness. The largest and most catastrophic of these occurred on November 21st 2016 in Melbourne, Australia (1,2,3). After the storm struck at 6 pm, more than 3500 patients across the state of Victoria presented in respiratory distress to Emergency Rooms (ERs), overwhelming ambulance and health services (4,5). Countless more individuals were managed in the community by general practices and pharmacies. Ten deaths have been traced to this epidemic, far exceeding global mortality rates of single reported deaths in three earlier epidemics (6,7,8).

The pathogenesis of thunderstorm asthma is multifactorial. Meteorological factors combine synergistically with high aeroallergen loads to trigger bronchospasm in susceptible individuals (9,10). Culprit allergens vary across the globe. The allergen implicated in epidemics occurring in South-eastern Australia is ryegrass pollen (6, 11,12) with all six previous Melbourne epidemics also occurring in November, during the peak of ryegrass pollination (6,13,14,15). The 2016 event also recorded extreme levels (102 grains/m3 of airborne pollen on the day (16,17). (Increased particulate matter (PM_{10}) concentrations at the time were associated with a gust front and constituted a raised dust event, most likely incidental (17).)

Individual susceptibility factors that have been identified in previous Australian epidemics are: allergic rhinitis; ryegrass pollen sensitization; pre-existing asthma; poor adherence to corticosteroid-based preventers; and, outdoor location at the time of the storm (11,12). The relevance of ryegrass pollen sensitization, seasonal allergic rhinitis, and outdoors exposure was again confirmed for the 2016 Melbourne epidemic, occurring in 100%, 99%, and 94%

respectively among patients from a single centre (16). However, the small size and restricted datasets of the cohorts limit their conclusions.

We recently reported that the 2016 Melbourne epidemic featured an ethnic predominance of Asian and Indian patients among ER presentations, hospital admissions, and fatal cases (17); possible explanations for this include higher rates of atopy, or socio-cultural barriers to medical care. Also, while the majority of thunderstorm asthma patients did not have a prior diagnosis of asthma, all patients admitted to intensive care had known asthma (17).

To further explore individual susceptibility to epidemic thunderstorm asthma, and in particular to severe acute attacks, we sought to identify risk factors for hospital admission from the ER. We studied patients who presented to ERs of the eight Melbourne metropolitan health networks and subsequently undertook structured telephone interviews.

Methods

Clinical follow-up

Hospital ERs were overwhelmed by thunderstorm asthma presentations on the 21st of November 2016, and no site was able to immediately organize appropriate follow-up for assessed patients.

The eight Melbourne metropolitan health services collaborated to develop a unified approach. Each Respiratory Department obtained full lists of all ER presentations with respiratory symptoms at their institution between the 21st and 23rd of November, 2017. Each patient's record was reviewed to confirm consistency with acute asthma and to exclude alternative diagnoses. A clinical questionnaire was developed. Medical staff and trained medical students under supervision telephoned all affected patients. This occurred 3-4 weeks after the

thunderstorm event between the 12th and 23rd of December, 2016. Based on telephone responses and according to individual local resources, patients were appropriately triaged to attend urgent clinic review.

Clinical telephone questionnaire.

There are no validated telephone questionnaires for use in thunderstorm asthma epidemics. A questionnaire was therefore devised to determine the following: whether patients were still symptomatic at the time of the phone call (high-risk: urgent review); age and gender; self-identified ethnicity; birthplace; whether they had perennial or seasonal rhinitis and its severity (18); their main location during the afternoon and evening (outdoors vs indoors; windows open or shut); whether they had previously been diagnosed with asthma, and; if known to have asthma, to what degree their asthma had been controlled prior to the thunderstorm event (based on the Asthma Control Test, used with permission) (19), use of inhaled corticosteroid preventers, possession of asthma action plans, and previous health care utilization; if not known to have asthma, whether they had symptoms suggesting undiagnosed asthma prior to the thunderstorm event; concurrent allergic conditions; and smoking status.

Data compilation, comparison, and analysis

Following the conclusion of the clinical triaging protocol, approval from all eight individual Institutional Review Boards was sought to retrieve, analyze, and combine data from multiple sites in order to inform government public health policy (4) and for scientific publication. Non-identifiable data were pooled from all sites for combined analysis. Baseline demographics of this cohort have been previously published (17).

Patient characteristics

The characteristics of patients discharged home from the ER were compared to those who were admitted to hospital.

Risk factors for hospital admission

Descriptive data are presented as frequencies and percentages for categorical data and mean (SD) for continuous. Demographic and clinical characteristics of those admitted to hospital and those discharged home from the ER were compared using t-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables.

Logistic regression methods were used to assess risk factors for outcomes defined as hospital admission (yes, no). Both unadjusted and age, sex adjusted models were fitted to each outcome. Multivariate logistic regression models with all possible risk factors, age and sex adjustments were also considered.

Interaction terms were included in the regression models if there were evidence of an effect modification from any of the risk factors. Interaction terms were considered statistically significant if $p < 0.1$.

Results are presented as odds ratios with 95% confidence intervals with $p < 0.05$ considered statistically significant. Analyses were performed using Stata, release 12.0 (StataCorp, College Station, TX).

RESULTS

Patients were identified and contacted as previously published (17). Of 2242 records of thunderstorm presentations, usable interview data were obtained for 1435 patients (64%).

[The other 807 patients were either uncontactable despite repeated phone calls (679), declined to participate (105) or provided uninterpretable data (23).] Of the 1435 patients analyzed, 164 (11.4%) required hospital admission, and the rest were discharged directly from the ER.

The patient cohort had a mean age of 32.0 years (standard deviation ±18.6 years) and a male predominance (56%, Table 1). Current smoking was uncommon (8%). As reported in our previous publication, Asian (Chinese, Vietnamese, East or South-East Asian- 22%) and Indian (includes Sri Lankan, Pakistani and Bangladeshi-17%) ethnicity were over-represented in this cohort, when compared to census-derived combined Asian and Indian ethnicity of 25% in the resident population (Table 1) (17). Exactly half the cohort was born in Australia. Sixty-eight percent reported exposure to the outdoors (either physically outdoors, or indoors with open windows).

Known current asthma was present in 28%, past asthma in 15%, and symptoms suggestive of undiagnosed asthma (wheeze, chest tightness or shortness of breath) in 26% (Table 2). Eighty-seven percent reported symptoms of seasonal allergic rhinitis (runny nose, sneezing, blocked itchy nose, watery itchy eyes) involving at least the springtime. Seventy-two percent had moderate or severe rhinitis symptoms based on established criteria (18). Self-reported eczema (28%) and food allergy (16%) were less common (Table 2).

Among patients with current asthma, symptoms were not well controlled in 55% (Table 3). Fifty-eight percent of those with current asthma did not have an action plan. Sixty-eight percent either were not prescribed a corticosteroid preventer prior to the thunderstorm, or were using this less than five days per week. Health care utilization among current asthma patients in the prior 12 months is summarized in Table 3, and indicated low rates of non-

urgent primary care review, and high rates of ER presentations and overnight hospital admissions.

Risk factors for hospital admission

Among the entire cohort, patients with known current asthma had increased odds of admission to hospital wards or intensive care [adjusted odds ratio (aOR) 1.87, 95% CI 1.26, 2.78,]. Increasing age and increasing numbers of years in Australia were also associated with higher odds of requiring hospital admission. Asian ethnicity appeared to reduce the odds of hospital admission (aOR 0.59,95% CI 0.38, 0.94, Table 4).

Because birthplace in Australia was near significance for an increased risk for hospital admission (aOR 1.44, p=0.05, Table 4) we further stratified data by place of birth (Table 5). Asian patients born overseas (first generation migrants) had a lower odds of admission (aOR=0.33, 95% CI 0.13, 0.85). An interaction analysis of Asian ethnicity and birth place showed a higher odds of admission for Asian patients born locally in Australia (aOR=5.42, 95% CI 1.56, 18.83).

Patients of Indian ethnicity did not have these risk-profile interactions with birthplace (data not shown).

Among patients with known current asthma, hospital admission in the prior 12 months was associated with higher odds of hospital admission for thunderstorm asthma (aOR 3.16, 95% CI 1.63, 6.12, Table 6). The possession of an asthma action plan increased the odds for hospital admission (aOR 2.7595% CI 1.51, 5.00), while self-reported food allergy reduced the odds for admission (aOR 0.31, 95% CI 0.13, 0.76) (Table 6).

Discussion

The 2016 Melbourne thunderstorm epidemic asthma event was the largest and most catastrophic to date (17). Among patients who presented to ER during the epidemic, our analysis indicates that age, current asthma, ethnicity, and birthplace, all influence the likelihood of a severe acute attack requiring hospital admission. These findings have wide-ranging practical implications for public health messaging, preventive health policy, and clinical management. They also extend our understanding of the pathophysiology of epidemic thunderstorm asthma and the underlying patterns of allergic disease.

Our initial investigation into the 2016 Melbourne epidemic suggested that patients of Asian (Chinese, Vietnamese, East or South-East Asian) and Indian subcontinental ethnicity were over-represented among ER presentations, among those admitted to hospital, and also among ten deaths related to the epidemic and under coronial investigation (17).

Conflicting factors around ethnicity may impact ER presentations for asthma. On one hand, ethnic minorities are often substantially composed of migrants, who usually have a lower prevalence of asthma (20,21,22). On the other hand, ethnic minorities face disparities in asthma burden (23), quality of asthma care (24,25), and even inclusion in asthma trials (26). Thus the previous observation that Asian and Indian minorities were over-represented in the recent thunderstorm asthma epidemic (17) could relate to social and cultural factors impeding effective planned asthma care, and predisposing to increased unplanned ER presentations.

This current analysis uncovers a more complex and nuanced relationship between ethnicity and thunderstorm asthma risk, due to an interaction with birthplace. We found that first-generation Asian migrants presenting to ER with thunderstorm asthma had lower odds for severe attacks requiring hospital admission when compared to patients of Caucasian, or other, ethnicity. This is consistent with the previously-described healthy migrant effect (20). We additionally found, however, that Asian patients born locally in Australia had higher odds for

241 severe attacks requiring hospital admission. This is unlikely to be explained by socio-cultural
242 factors, since second and subsequent-generation Asian migrants might be expected to have
243 overcome socio-cultural barriers to good health care. Australian-born status would be
244 expected to confer protection from admission rather than increased risk. We therefore
245 postulate that the increased odds for hospitalization among Australian-born Asians are more
246 consistent with a gene-environment interaction and conceivably incremental sensitization to
247 rye grass pollen.

248 It is known that second generation migrants are often reported to have a higher prevalence of
249 asthma than first generation migrants (20). This has been specifically shown among Asian
250 immigrants in Melbourne (27). In the same study, the prevalence of atopy and allergic rhinitis
251 in Asians, whether born overseas or in Australia, exceeded that observed in non-Asians (27).
252 Against this background, first generation Asian migrants may have an (initially) dormant
253 genetic susceptibility toward greater severity of thunderstorm-related asthma attacks, which
254 becomes activated in subsequent generations by early life environmental exposures occurring
255 in the host country.

256 In further support of this hypothesis, multiple ethnic-specific gene-environment interactions
257 relating to atopic disease have been reported (28). Differences in asthma genetics are also
258 present between Chinese and other populations (29). Epigenetic mechanisms such as DNA
259 methylation may explain how environmental conditions influence the expression of allergy
260 and asthma genes (30). Our findings are also concordant with the pattern of non-respiratory
261 allergic conditions in Australia, where Asian-born children have a lower prevalence of nut
262 allergy than their Australian-born counterparts, but Asian children born in Australia have an
263 increased prevalence compared to non-Asian children (31, 32). With increasing globalization
264 and migration trends from the developing world to developed nations, our findings have

implications for the prevention, detection, and treatment of allergic diseases beyond epidemic thunderstorm asthma.

The risk profiles related to Asian ethnicity in this analysis did not extend to patients of Indian sub-continental ethnicity. It is possible that the two ethnic groups may have a different susceptibility to relevant gene-environment interactions, although there are currently no data to support this hypothesis. Different migration patterns may offer another explanation for our finding, as proportionally fewer patients of Indian ethnicity (16%) than Asian ethnicity (24%) were born in Australia.

An increased number of years spent in Australia, and increasing age, were also associated with a higher risk of hospitalization. The former may reflect increasing vulnerability with progressive environmental exposure among first-generation migrants. The latter suggests that thunderstorm asthma attacks may be treated more easily in children, and be more rapidly responsive to therapy.

A second major risk factor for hospital admission from the ER in this analysis was a known current asthma diagnosis. This suggests that although individuals without a prior asthma diagnosis (comprising 56% of our total cohort) are vulnerable to thunderstorm asthma, patients with known current asthma are at greater risk of a severe attack requiring admission. This may possibly relate to a greater degree of bronchial hyper-reactivity present in those with known current asthma. In addition, we found that patients with an overnight hospital admission in the previous 12 months were at even higher risk. This is consistent with our prior observation during the 2016 epidemic, that all 35 patients admitted to Victorian intensive care units had a known diagnosis of asthma (17). Taken together, it could be argued that the most effective approach in Australia to mitigate the danger of epidemic thunderstorm asthma lies in focusing on the known asthmatic population, and addressing existing gaps in

asthma care delivery (33,34). Such measures would also deliver the substantial benefit of reducing all asthma mortality, still approximating 400 Australian lives per year (35).

The finding that possessing an action plan increased the odds for admission among known asthmatics was not unexpected. It is likely to reflect higher possession of action plans among patients with poor asthma control or recent hospitalization. The finding of lower odds for admission among patients reporting food allergy may be due to unmeasured confounding factors.

Our study confirmed the presence of allergic rhinitis in the overwhelming majority of patients presenting to ERs, similar to previous Australian reports (6,12,36). In communities vulnerable to thunderstorm asthma, this highlights the need for allergic rhinitis management to be carefully optimised, with measures including intranasal corticosteroids and specific allergen immunotherapy for grass-pollen allergy (37,38).

We also found a low prevalence of smoking, again consistent with previous epidemics (12,39). Exposure to the outdoors did not appear to increase the risk of hospital admission, but this may reflect our questionnaire design; patients were asked where they spent most of the afternoon or evening, rather than whether they had any outdoors exposure or known direct exposure during the event.

The cohort comprised 40% of the estimated 3500 ER thunderstorm presentations across the state of Victoria, but 64% of all presentations to our eight metropolitan health services. Baseline demographic data from the Department of Health (4) of total ER presentations showing near identical mean age and gender distribution with our cohort give assurance that our data are representative of all cases affected by the 2016 Melbourne epidemic. We have interpreted the need for hospital admission from the ER to reflect a greater severity of acute

asthma (40). However, factors other than asthma acuity, including ethnicity, can also influence the clinical decision to admit from the ER (41). While we examined individual susceptibility for hospital admission due to thunderstorm asthma, we were unable to take into account variability in exposure due to local meteorological conditions, or the timing, intensity, and duration of exposure to airborne allergens.

We further acknowledge that the estimated confidence interval are wide for the interaction analysis of Asian born in Australia (aOR=5.42, 95% CI 1.56, 18.83) but note that the p value for the interaction term is highly significant p=0.008. As this is a discovery our findings should ideally be validated in another cohort that experienced epidemic thunderstorm asthma. But this is somewhat challenging because other thunderstorm asthma event studies may not have similar information about ethnicity. Our ethnicity strata sample size is small which raises the possibility of a power issue. Residual confounding may also be likely with small group stratification due to sampling error. We have as best possible, adjusted for possibly important confounding but acknowledge that it may not eliminate it due to unknown or un-included confounders (socioeconomic status, educational level or psychological comorbidity). Despite this possibility, we still found significant effects of an interaction term between ethnicity and born in Australia.

In conclusion, current asthma and previous hospitalization for asthma are compounding risk factors for hospital admission due to epidemic thunderstorm asthma. In addition, contrasting findings in first-generation Asian migrants and Australian-born Asian individuals suggest that gene-environment interactions may increase risk. These patient groups should be identified and targeted in public health education and preventative strategies.

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468 Table 1. Demographics of thunderstorm asthma patients by Emergency Room disposition

	All ER patients (n=1435)		Discharged home (n=1271)		Admitted to hospital (n=164)		p value
Age (Mean, SD)	31.9	18.5	31.6	17.8	35.1	23.5	0.06
Sex	n	%	n	%	n	%	0.34
Female	636	44	569	45	67	41	
Male	799	56	702	55	97	59	
Smoking	n	%	n	%	n	%	0.97
Never	934	65	828	65	106	65	0.99
Past	340	24	300	24	40	24	
Current	122	8	108	8	14	9	
Unknown	39	3	35	3	4	2	
Ethnicity	n	%	n	%	n	%	0.29
Caucasian	544	38	475	37	69	42	
Asian	320	22	293	23	27	16.5	
Indian	239	17	209	16	30	18	
Aboriginal or Torres Strait Islander	10	1	8	0.6	2	1.4	
Other	146	10	131	10	15	9	
Unknown	176	12	155	12	21	13	
Born in Australia	n	%	n	%	n	%	0.44
Yes	723	50	636	50	87	53	
No	698	49	623	49	75	46	
Unknown	14	1	12	0.9	2	1	
Exposure	n	%	n	%	n	%	0.71
Outdoors	518	36	463	36	55	33.5	
Indoors with open windows	464	32	410	32	54	33	
Indoors closed windows	431	30	378	30	53	32	
Unknown	22	2	20	1.6	2	1.2	

469 ER: Emergency Room. SD: standard deviation. Asian: Chinese, Vietnamese, East or South-East Asian. Indian: also includes Sri-Lankan, Pakistani and Bangladeshi

Table 2 Asthma and allergic disease status of thunderstorm asthma patients by Emergency Room disposition.

		All ER patients (n=1435)		Discharged home (n=1271)		Admitted to hospital (n=164)		p value
		n	%	n	%	n	%	
Asthma status								**0.003**
Known asthma	Current asthma (symptoms in prior 12 months)	395	28	337	27	58	35	
	Past asthma (symptoms > 12 months prior)	210	15	189	15	21	13	
No known asthma	Symptoms suggesting undiagnosed asthma	369	26	319	25	50	30	
	Never had asthma symptoms	424	30	394	31	30	18	
Unknown		10	0.7	9	0.7	1	0.6	
Allergic rhinitis	Any	1249	87	1112	87.5	137	84	0.94
	Seasonal & perennial	90	7	81	7	9	7	
	Seasonal only	995	80	887	80	108	80	
	Perennial only	149	12	132	12	17	13	
	None	172	12	146	11.5	26	16	
	Unknown	14	1	13	1	1	<1	
Allergic rhinitis severity	Mild (ARIA 0)	322	26	283	25	39	28	0.38
	Moderate (ARIA 1-3)	655	52	585	53	70	51	
	Severe (ARIA 4)	247	20	226	20	21	15	
	Unknown	25	2	18	2	7	5	
Self-reported allergy history	History of grass pollen allergy	727	51	651	51	76	46	0.24
	History of eczema	404	28	355	28	49	30	0.45
	History of food allergy	231	16	211	17	20	12	0.16

ER: Emergency Room. ARIA: Allergic Rhinitis and its Impact on Asthma (18).

Table 3. Asthma management, control, and health care utilization, among patients with known current asthma by Emergency Room disposition.

		All ER patients (n=395)		Discharged home (n=337)		Admitted to hospital (n=58)		p value
		n	%	n	%	n	%	
Uncontrolled Asthma	ACT<20	219	55	188	56	31	53	0.69
No regular preventer	No preventer use or preventer use <5 days per week	267	68	230	68	37	64	0.28
No action plan		228	58	205	61	23	40	**0.001**
Health care utilisation	Non urgent GP visit	175	44	147	44	28	48	0.35
	Urgent GP visit	91	23	78	23	13	22	0.91
	Hospital or ER visit	116	29	93	28	23	40	0.05
	One night in hospital	56	14	39	12	17	30	**<0.001**

GP: general practitioner, ER: Emergency Room, ACT: Asthma Control Test (19).

Table 4. Effect size of each variable on hospital admission for all patients.

Characteristic	Odds for hospital admission from Emergency room			
	Unadjusted		Adjusted for age & sex	
	OR (95% CI)	p	OR (95% CI)	p
Age (n=1 435)	**1.010 (1.002, 1.019)**	**0.02**	-	
Sex male (n=1 435)	1.17 (0.84, 1.63)	0.34	-	
Asian ethnicity (n=1 113)	**0.63 (0.40, 0.99)**	**0.04**	**0.59 (0.38, 0.94)**	**0.02**
Born in Australia (n=1 421)	1.14 (0.82, 1.58)	0.44	1.44 (0.99, 2.09)	0.05
Current asthma (n=1 052)	**1.92 (1.30, 2.85)**	**0.001**	**1.87 (1.26, 2.78)**	**0.002**
Exposure outdoors (n=1 413)	0.87 (0.62, 1.24)	0.44	0.88 (0.62, 1.24)	0.45
ARIA score (n=1 224)				
• 1	0.79 (0.46, 1.36)	0.39	0.79 (0.46. 1.37)	0.41
• 2	1.05 (0.62, 1.78)	0.86	1.03 (0.60, 1.75)	0.91
• 3	0.79 (0.45, 1.38)	0.39	0.78 (0.44, 1.36)	0.38
• 4	0.67 (0.39, 1.18)	0.16	0.66 (0.38, 1.16)	0.15
History of eczema (n=1 399)	1.15 (0.80, 1.64)	0.45	1.25 (0.86, 1.80)	0.24
History of food allergy (n=1 407)	0.71 (0.43, 1.16)	0.16	0.73 (0.45, 1.20)	0.22
History of grass pollen allergy (n=1 411)	0.82 (0.59, 1.14)	0.24	0.80 (0.57, 1.11)	0.17
Current smoking (n=1 396)	1.00 (0.56, 1.79)	0.99	0.96 (0.54, 1.73)	0.89
Years living in Australia (n=1 402)	**1.02 (1.01, 1.03)**	**0.001**	**1.22 (1.01, 1.03)[a]**	**0.001**

[a] Adjusted by sex. Asian: Chinese, Vietnamese, East or South-East Asian. ARIA: Allergic Rhinitis and its Impact on Asthma (18).

Table 5. Association between Asian ethnicity and hospital admission for the whole cohort, stratified by birth place.

Characteristic	Odds for hospital admission from Emergency			
	Born overseas (n=316)		Born in Australia (n=351)	
	OR (95% CI)	p	OR (95% CI)	p
p for interaction = 0.008[β]				
Age	1.02 (1.00, 1.05)	0.08	1.01 (0.99, 1.03)	0.38
Sex male	0.53 (0.22, 1.26)	0.14	1.29 (0.62, 2.66)	0.49
Asian ethnicity	**0.33 (0.13, 0.85)**	**0.02**	1.76 (0.73, 4.22)	0.20
Current asthma	**2.58 (1.04, 6.41)**	**0.04**	1.35 (0.68, 2.69)	0.39
Exposure outdoors	2.07 (0.88, 4.86)	0.09	0.52 (0.25, 1.12)	0.09
ARIA score				
• 1	0.64 (0.15, 2.82)	0.55	0.95 (0.37, 2.42)	0.91
• 2	0.55 (0.13, 2.22)	0.39	2.18 (0.84, 5.66)	0.10
• 3	0.80 (0.20, 3.25)	0.75	1.11 (0.38, 3.24)	0.84
• 4	1.63 (0.50, 5.35)	0.41	0.84 (0.27, 2.59)	0.75
History of eczema	1.25 (0.46, 3.40)	0.66	1.70 (0.81, 3.43)	0.16
History of food allergy	1.41 (0.50, 3.99)	0.51	0.72 (0.29, 1.75)	0.46
History of grass pollen allergy	0.49 (0.20, 1.19)	0.11	0.72 (0.36, 1.44)	0.35
Current smoking	0.70 (0.08, 5.96)	0.74	1.25 (0.34, 4.69)	0.73

[β] OR for interaction between Asian ethnicity and born in Australia = 5.42 (95% CI: 1.56, 18.83)

Asian: Chinese, Vietnamese, East or South-East Asian. ARIA: Allergic Rhinitis and its Impact on Asthma (18).

Table 6. Effect size of each variable on hospital admission, for patients with known current asthma.

Characteristic	Odds for hospital admission from Emergency			
	Unadjusted		Adjusted with age & sex	
	OR (95% CI)	p	OR (95% CI)	p
Age (n=395)	1.00 (0.98, 1.01)	0.80	-	
Sex male (n=395)	1.56 (0.87, 2.79)	0.13	-	
Asian ethnicity (n=328)	0.65 (0.31, 1.36)	0.25	0.61 (0.29, 1.31)	0.20
Born in Australia (n=394)	1.00 (0.57, 1.76)	0.99	0.94 (0.49, 1.80)	0.85
Exposure outdoors (n=393)	0.86 (0.47, 1.57)	0.62	0.84 (0.46, 1.54)	0.58
ARIA score (n=334)				
• 1	0.69 (0.22, 2.15)	0.52	0.66 (0.21, 2.05)	0.47
• 2	2.03 (0.79, 5.20)	0.14	2.11 (0.82, 5.43)	0.12
• 3	0.9 (0.31, 2.64)	0.84	0.89 (0.30, 2.62)	0.83
• 4	0.99 (0.40, 2.47)	0.98	1.01 (0.40, 2.53)	0.98
History of eczema (n=385)	1.47 (0.83, 2.62)	0.19	1.47 (0.81, 2.68)	0.20
History of food allergy (n=386)	**0.32 (0.13, 0.76)**	**0.01**	**0.31 (0.13, 0.76)**	**0.01**
History of grass pollen allergy (n=387)	0.76 (0.43, 1.35)	0.35	0.77 (0.44, 1.37)	0.38
Current smoking (n=387)	1.35 (0.49, 3.71)	0.56	1.26 (0.45, 3.50)	0.65
Have asthma action plan (n=387)	**2.52 (1.42, 4.45)**	**0.002**	**2.75 (1.51, 5.00)**	**0.001**
ACT controlled (n=386)	1.12 (0.64, 1.97)	0.69	1.07 (0.60, 1.91)	0.81
*Non-urgent visit to GP (n=380)	1.31 (0.74, 2.33)	0.35	1.31 (0.73, 2.33)	0.36
*Urgent visit to GP (n=383)	0.97 (0.49, 1.89)	0.91	0.99 (0.50, 1.93)	0.96
*Visit to hospital (n=384)	1.76 (0.98, 3.16)	0.05	1.77 (0.98, 3.20)	0.06
*Admitted to hospital for at least one night (n=379)	**3.17 (1.64, 6.15)**	**0.001**	**3.16 (1.63, 6.12)**	**0.001**
Years living in Australia (n=389)	1.01 (0.99, 1.03)	0.24	1.01 (0.99, 1.03)	0.17[a]

[a] Adjusted by sex. * In prior 12 months.
ARIA: Allergic Rhinitis and its Impact on Asthma (18), ACT: asthma control test (19).
Asian: Chinese, Vietnamese, East or South-East Asian.