The Melbourne epidemic thunderstorm asthma event 2016: an investigation of environmental triggers, effect on health services, and patient risk factors

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Summary

Background A multidisciplinary collaboration investigated the world's largest, most catastrophic epidemic thunderstorm asthma event that took place in Melbourne, Australia, on Nov 21, 2016, to inform mechanisms and preventive strategies.

Methods Meteorological and airborne pollen data, satellite-derived vegetation index, ambulance callouts, emergency department presentations, and data on hospital admissions for Nov 21, 2016, as well as leading up to and following the event were collected between Nov 21, 2016, and March 31, 2017, and analysed. We contacted patients who presented during the epidemic thunderstorm asthma event at eight metropolitan health services (each including up to three hospitals) via telephone questionnaire to determine patient characteristics, and investigated outcomes of intensive care unit (ICU) admissions.

Findings Grass pollen concentrations on Nov 21, 2016, were extremely high (>100 grains/m³). At 1800 AEDT, a gust front crossed Melbourne, plunging temperatures 10°C, raising humidity above 70%, and concentrating particulate matter. Within 30 h, there were 3365 (672%) excess respiratory-related presentations to emergency departments, and 476 (992%) excess asthma-related admissions to hospital, especially individuals of Indian or Sri Lankan birth (10% *vs* 1%, p<0·0001) and south-east Asian birth (8% *vs* 1%, p<0·0001) compared with previous 3 years. Questionnaire data from 1435 (64%) of 2248 emergency department presentations showed a mean age of 32·0 years (SD 18·6), 56% of whom were male. Only 28% had current doctor-diagnosed asthma. 39% of the presentations were of Asian or Indian ethnicity (25% of the Melbourne population were of this ethnicity according to the 2016 census, relative risk [RR] 1·93, 95% CI 1·74–2·15, p <0·0001). Of ten individuals who died, six were Asian or Indian (RR 4·54, 95% CI 1·28–16·09; p=0·01). 35 individuals were admitted to an intensive care unit, all had asthma, 12 took inhaled preventers, and five died.

Interpretation Convergent environmental factors triggered a thunderstorm asthma epidemic of unprecedented magnitude, tempo, and geographical range and severity on Nov 21, 2016, creating a new benchmark for emergency and health service escalation. Asian or Indian ethnicity and current doctor-diagnosed asthma portended life-threatening exacerbations such as those requiring admission to an ICU. Overall, the findings provide important public health lessons applicable to future event forecasting, health care response coordination, protection of at-risk populations, and medical management of epidemic thunderstorm asthma.

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See Comment page e236

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Introduction

Thunderstorm asthma is the triggering of an asthma attack by environmental conditions caused by a local thunderstorm. On Nov 21, 2016, the world's largest and most catastrophic epidemic of thunderstorm asthma occurred in Melbourne, Australia, precipitating several thousand acute respiratory presentations to emergency departments and associated with ten deaths. Since first described more than 30 years ago,[1,2] research suggests thunderstorm asthma events result from a complex interaction of environmental and individual susceptibility factors.[3,4] Postulated environmental factors include high concentrations of an aeroallergen (predominantly ryegrass [*Lolium perenne*] pollen in

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See Online for appendix

Research in context

Evidence before this study

Scientific literature was comprehensively reviewed for studies examining thunderstorm asthma, searching the databases CINAHL (via EBSCOhost), PUBMED, Embase (including Medline), Web of Science, and SCOPUS for articles published between 2007–18 with the specific terms "thunderstorm" and "asthma". Synthesis and qualitative analysis of the literature indicated that three combined factors appear to be necessary for thunderstorm asthma. These factors are high-level aeroallergen exposure, frequently grass pollen, particular meteorological conditions such as some thunderstorms with outflows, and individual susceptibility factors including presence of allergic rhinitis with grass pollen sensitisation. The Melbourne thunderstorm asthma event was outstanding in size and severity by orders of magnitude compared with previous events.

Added value of this study

Our unique comprehensive multidisciplinary approach analysed the continuum from environmental and health service factors to emergency responses, in-patient care and intensive care units (ICU), and surveyed patients directly within 1 month of the event. By integrating datasets from many sites we were able to examine the largest cohort ever described, with a gradient of severity of those affected from emergency department presentations to ICU admissions and death and include unique data on ethnicity. Our findings suggested that convergent environmental factors triggered a thunderstorm asthma epidemic of unprecedented magnitude and severity, with current doctor-diagnosed asthma, and Asian or Indian ethnicity conferring particularly increased risk.

Implications of all the available evidence

Our findings provide vital public health lessons applicable to management of epidemic thunderstorm asthma and highlight the importance of predicting future events, coordinating health care responses and protection of at-risk asthma populations.

Australia);[5] rain and moisture to rupture ryegrass pollen grains, releasing fine allergen-bearing starch-granules (<2·5 µm) respirable to the lower airways;[6] and thunderstorm outflows which bring respirable allergen particles down to ground level.[7] Individual susceptibility factors include: previous sensitisation to seasonal aeroallergens;[8] a history of seasonal allergic rhinitis;[9] and low rates of inhaled corticosteroid use in patients with diagnosed asthma.[10] Although our understanding of thunderstorm asthma has improved, many questions remain pertaining to environmental and meteorological trigger factors, population risk and susceptibility, effects on health services, and mitigation strategies. We undertook a multidisciplinary investigation to detail the dynamics of the event, describe its effect on health services and patients, and inform public health strategies to prevent and mitigate future thunderstorm asthma epidemics.

Methods

Study design and environmental data

We obtained climate and weather data from the Australian Bureau of Meteorology including rainfall in Victoria over the preceding (2016) spring season, and radar imagery for the greater Melbourne and Geelong region and 1-min wind speed and direction, temperature, relative humidity, and rainfall for a representative meteorological station (Laverton) on Nov 21, 2016. Air pollutant data (including carbon monoxide, ozone, nitrogen dioxide, sulphur dioxide, and fine particles as PM_{10} or $PM_{2·5}$) were obtained from the Victorian Environment Protection Authority for air quality monitoring stations across greater Melbourne and Geelong on Nov 21, 2016. To examine regional pastureland growth, remotely-sensed monthly Normalized Difference Vegetation Index data were obtained from the Australian Bureau of Meteorology. Aerobiological data, including atmospheric pollen and fungal spore concentrations, were obtained from the Parkville and Burwood monitoring sites in Melbourne, from The University of Melbourne and Deakin University, respectively.

Data on use of health services

Data on acute medical impacts were sourced from Ambulance Victoria and the Victorian Department of Health and Human Services' Victorian Emergency Minimum Dataset and Victorian Admitted Episodes Dataset and analysed (appendix).

All presentations to emergency departments in eight Melbourne health services (each including up to three hospitals) with respiratory symptoms from 6pm on Nov 21 for up to 48 h were reviewed to confirm consistency with acute asthma and exclude alternative diagnoses. A structured phone questionnaire (appendix) was developed to follow up patients who presented with epidemic thunderstorm asthma 3–4 weeks after the thunderstorm event. Specific questions regarding self-identified ethnicity and birthplace in Australia were included. Self-identified Asian or Indian ethnicity were combined and compared with a non-Asian or Indian category. The ethnic identities of any deceased patients were obtained from clinical documentation (the thunderstorm asthma deaths are currently under investigation by the Victorian coroner and we did not apply for access to coronial data. Although the enquiries are ongoing and no findings have been made by the investigating Coroner, it was understood that among

the matters to be considered was the role, if any, that ethnicity might have played in the deaths).

Census data (from August, 2016) from the Australian Bureau of Statistics were interrogated (appendix) for the 23 local government areas (comprising a population of 3·2 million) serviced by the eight metropolitan health services. Responses to the census question on ancestry were pooled for Asian and Indian or subcontinent origins to infer Asian or Indian ethnicity of the resident population. The relative risk (RR) and corresponding 95% CI for thunderstorm asthma presentations or deaths for Asians or Indians versus non-Asians or Indians were calculated compared with the resident population.

We obtained data from the Australian and New Zealand Intensive Care Society Adult Patient Database, as well as individual intensive care units (ICUs) in 15 Victorian hospitals. Inclusion criteria were patients admitted to an ICU with a diagnosis of asthma (appendix), or if, in the opinion of the ICU specialist at each site, admission was due to acute thunderstorm-related bronchospasm. Demographic characteristics, previous respiratory diagnoses, and chronic disease status as defined by the Australian and New Zealand Intensive Care Society Adult Patient Database were recorded. Previous respiratory medication use and country of birth were also recorded. We determined patient characteristics, and investigated outcomes of ICU admissions from epidemic thunderstorm asthma. Data were summarised using mean (SD), median (IQR), or number (%) as appropriate, and compared using either unpaired two-tailed *t* tests, rank sum tests, χ^2 test, or Fisher's exact test where applicable. A p value of less than 0·05 was considered statistically significant.

Role of the funding source

There was no funding source for this study. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Spring, 2016, in Australia was Victoria's 10th wettest on record, but rainfall over this season was highly variable

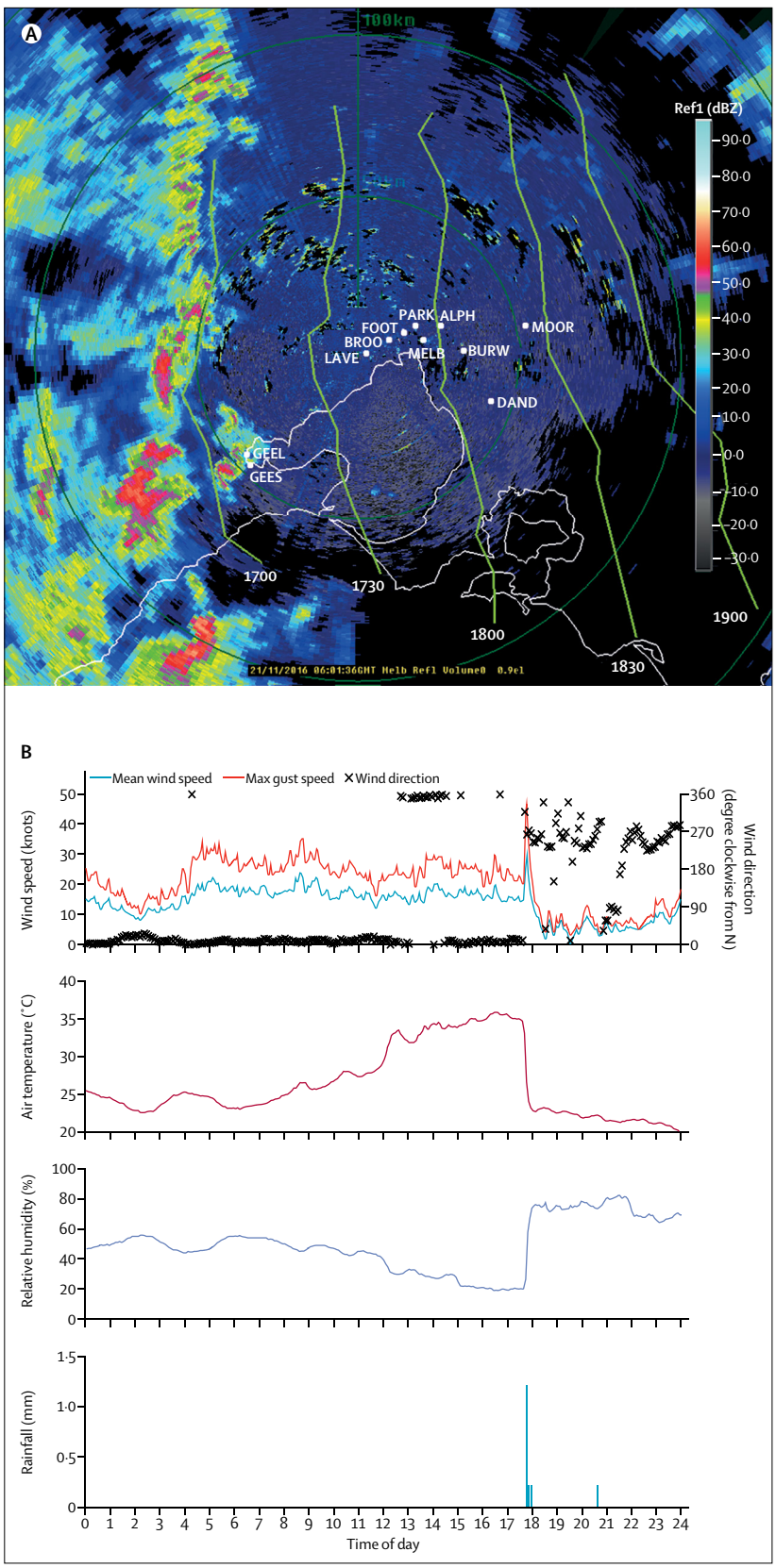

Figure 1: **Meteorological conditions in Melbourne, Australia, on Nov 21, 2016**

Radar image of the greater Melbourne region on the day of the epidemic thunderstorm asthma event, showing the thunderstorm (red areas) at 1701 AEDT, with the associated 1700–1900 AEDT gust front positions indicated by the green lines (panel A). The colour scale bar indicates reflectance (in the unit: decibel relative to Z [dBZ]) of the radar signal from rain and hail and is proportional to precipitation intensity. Also shown are locations of Geelong (GEEL) and Melbourne CBD (MELB) as well as pollen and fungal spore monitoring sites (PARK=Parkville, BURW=Burwood), the representative meteorological site (LAVE=Laverton), and the six air quality monitoring stations (ALPH=Alphington, BROO=Brooklyn, DAND=Dandenong, FOOT=Footscray, GEES=Geelong South, and MOOR=Mooroolbark). 5-min average wind speed, wind gust, wind direction, temperature, relative humidity, and rainfall at Laverton over 24 h (midnight to midnight; panel B). Source: Australian Bureau of Meteorology.

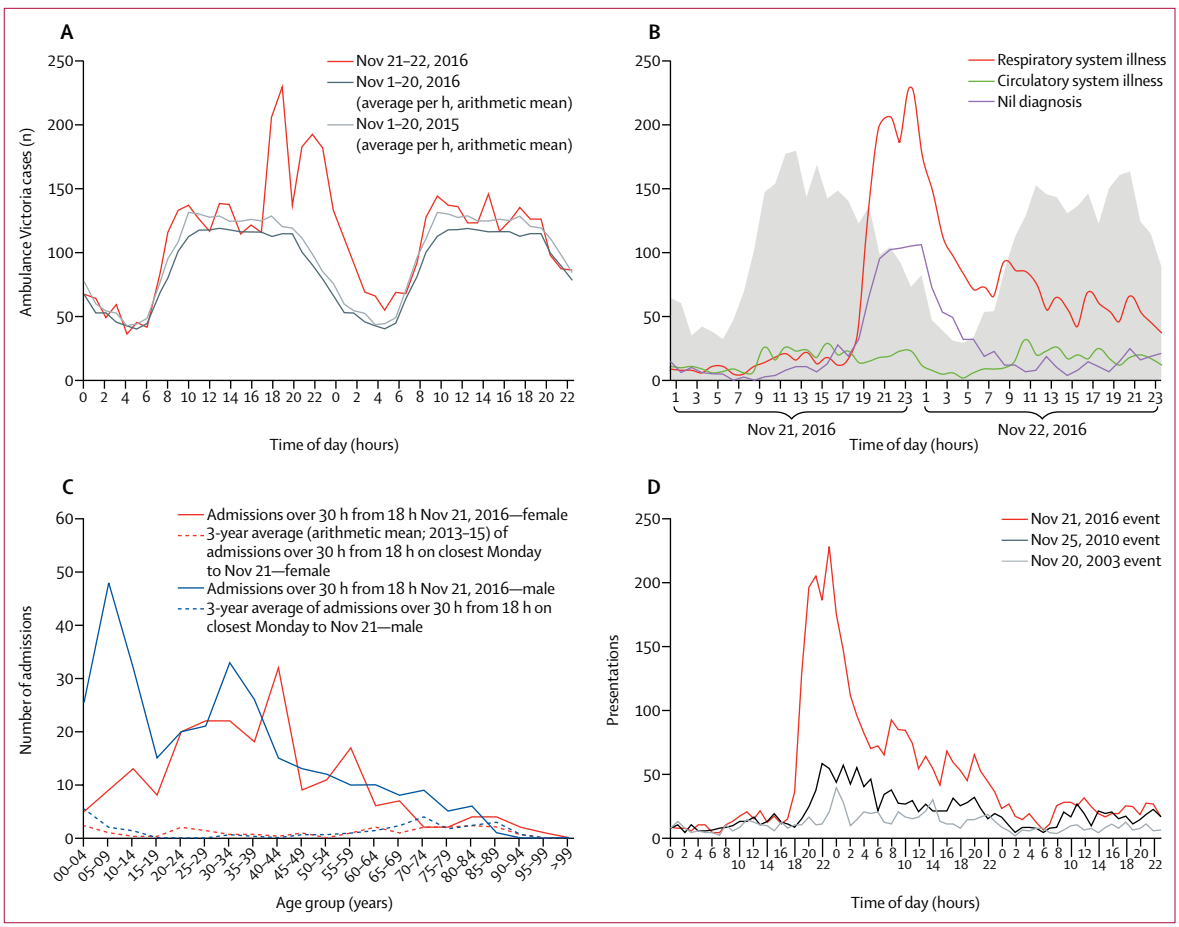

Figure 2: **Effect on health services of Nov 21, 2016 thunderstorm asthma event in Melbourne, VIC, Australia**
Total number of Ambulance Victoria cases by hour for 21–22 Nov, 2016 (panel A). Also shown are the average number of Ambulance Victoria cases on Nov 1– Nov 20, 2015 and 2016 averages for the corresponding times of the day. Source: Ambulance Victoria. Hourly presentations to Melbourne and Geelong public hospital emergency departments on 21–22 Nov, 2016 (panel B). Age and sex distribution of asthma-related hospital admissions to Melbourne and Geelong public hospitals for 30 h from 6pm on 21 Nov, 2016, compared with the same period averaged over the previous 3 years (2013–15; panel C). Hourly respiratory presentations (including nil diagnosis) to Victoria (Australia) public hospital emergency departments during the November 2016 thunderstorm asthma event and two other epidemic thunderstorm asthma events over 3 days (panel D).

with record highs in September and a generally drier than average November (appendix). Remotely sensed Normalized Difference Vegetation Index revealed anomalously high pastureland growth in October and November, especially north and west of Melbourne (appendix). On Nov 4, 7, and 12, there were extremely high concentrations of airborne grass pollen (>100 grains per m³ of air). On Nov 21, Victoria had its first very hot day since the previous summer as a gusty northerly wind flow developed, with temperatures peaking around 40–44°C, and peaks around 35°C in greater Melbourne itself. Airborne grass pollen concentrations in Melbourne on this day were in the extreme range, 102 and 171 grains per m³ of air at Parkville and Burwood respectively (appendix). There was also a recorded rise in fungal spores, tree and weed pollen, and ruptured pollen grains on Nov 21. A north–south line of thunderstorms developed to the west of Melbourne and Geelong, which resulted in a gust front that progressed eastwards through the greater Melbourne area between around 1700 AEDT and 1830 AEDT (figure 1). This gust front was associated with a rapid decrease in temperature of about 10°C, an increase in relative humidity to 70–80% (figure 1), and particulate matter (PM_{10}) concentrations exceeding the associated air quality guideline and constituting a raised dust event; eg, Geelong South peaked at 590·3 μg/m³, Footscray peaked at 698·6 μg/m³, and Brooklyn peaked at 942·5 μg/m³ (appendix).

From 1800 AEDT on Nov 21, 2016, Victorian ambulance services had a substantial increase in demand with 814 ambulance cases generated in the first 6 hours (figure 2), 643 being categorised as code 1 (the most urgent). This was associated with substantially increased out-of-hospital cardiac arrests and prehospital deaths as previously published.[11]

The sheer magnitude of the patient demand on emergency departments resulted in many incomplete medical records in favour of providing urgent clinical care. All major emergency department presentation diagnostic blocks were within normal variance on Nov 21 and 22, with the exception of the respiratory block and cases coded nil diagnosis (which includes incomplete records). Both rose rapidly from 1800 AEDT, peaked near midnight and then declined through the morning hours, leaving a high residual case load of respiratory presentations the following day (figure 2). Patients coded as nil diagnosis over this specific period were regarded as presentations of epidemic thunderstorm asthma and included in the analysis.

During the 30 h after 1800 AEDT an estimated 3365 excess respiratory-related cases presented to Melbourne and Geelong public hospital emergency departments (a 672% increase above the 3-year average of 501). Emergency department triage categories 3 and 4 (assessment and treatment required within 30 or 60 min, respectively) accounted for 83% of presenting patients, with a smaller but clinically important early rise in category 2 (imminently life-threatening or important time-critical treatment required with assessment and treatment within 10 min). 57% of presenting cases were male.

Over this period there were an estimated 476 excess asthma-related admissions to public hospitals in Melbourne and Geelong, a 992% increase, of which 59% were male. Those aged 20–59 years comprised 57% of the asthma admissions compared with 21% average for the same 30 h period, in the previous 3 years (p<0·0001; figure 2). By contrast, the proportion of total asthma admissions made up by those aged less than 20 years remained similar in 2016 (30%) to the average for the same 30 h period in the previous 3 years (25%, p=0·58). There was a smaller increase in the number of admissions involving those aged 60 years and older. This relatively small increase caused a large decrease in the proportion of older adults admitted from a 54% average in the previous 3 years, to 13% during the 2016 event (p<0·00001; figure 2).

Analysis of the country of birth of patients admitted for asthma showed they were more likely to have been born overseas (40% *vs* 26%, p<0·0001); in particular, there was an increased proportion of patients born in India and Sri Lanka (10% *vs* 1%, p<0·0001) and south-east Asia (8% *vs* 1%, p<0·0001) compared with the previous 3 years.

From Nov 21 until the end of November, there were 1121 admissions for asthma in Melbourne and Geelong compared with a 3-year average of 351. This was an excess of 770 cases (of which 62% occurred in the 30 h from 1800 AEDT on Nov 21). Comparison with two previously reported recent Melbourne epidemic thunderstorm asthma events (Nov 20, 2003 and Nov 25, 2010)[12,13] shows them both to have become completely dwarfed by the unprecedented magnitude of the Nov 21, 2016, event (figure 2).

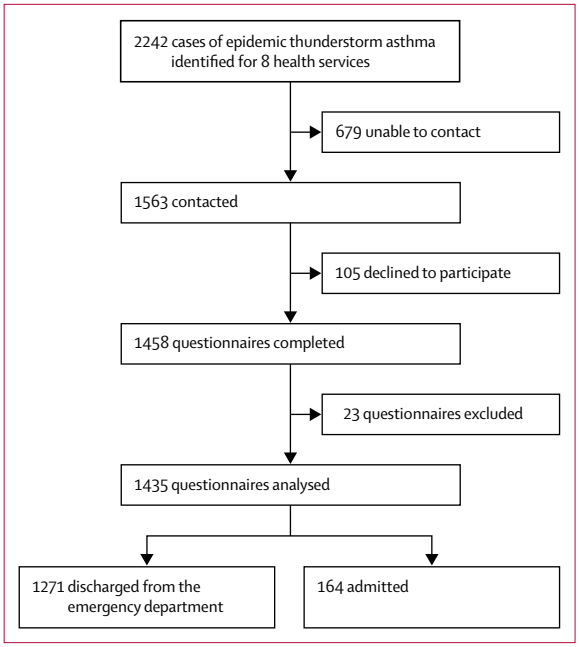

Figure 3: **Study profile**
Follow-up of patients from Melbourne hospital emergency departments after the Melbourne Nov 21, 2016, epidemic thunderstorm asthma event.

From the emergency department records of eight Melbourne metropolitan health services, 2242 cases of emergency thunderstorm asthma were identified with a final response of 1435 (64%) completed questionnaires (figure 3). The mean age of respondents was 32·0 years (SD 18·6) and 56% were male (table). 43% had doctor-diagnosed asthma with 64% (28% overall) reporting symptoms within the past 12 months. Of those who had never been diagnosed with asthma (56%, n=809), 46% reported having experienced symptoms suggestive of underlying asthma. Ethnicity was 39% Asian or Indian (*vs* 25% from 2016 census-derived data, relative risk [RR] 1·93, 95% CI 1·74–2·15, p <0·0001), 79% of whom were born overseas. Of ten deaths (mean age 38·5 years [SD 13·1], range 18–57, seven of whom were male), six were Asian or Indian (RR 4·54, 95% CI 1·28–16·09, p=0·01).

35 patients were admitted to a Victorian ICU with asthma or bronchospasm on Nov 21 and 22 2016, of which 28 (80%) were admitted over an 8h period from 2000–0400 AEDT (figure 4). Of these 35 patients, 22 (63%) were male. Two children aged 9 and 12 years were admitted; median age of adult patients was 42 years (IQR 32–53, range 21–69), with all patients known to have doctor-diagnosed asthma. Only one in three patients (12 [34%] of 35) were taking inhaled corticosteroid medication at the time of the event, although all but one were using inhaled bronchodilator (reliever) medication. 19 (54%) of the patients admitted to ICU were born outside Australia, including ten (29%) born in Asian or subcontinental countries.

	Cases of thunderstorm asthma (n=1435)
Age (years)	32·0 (18·6)
Women	636 (44%)
Men	799 (56%)
Ever been diagnosed with asthma	616 (43%)
Current asthma (symptoms within 12 months)	395 (64%)
Past asthma (symptoms >12 months ago)	210 (34%)
Unknown	11 (2%)
Never been diagnosed with asthma	809 (56%)
Symptoms suggestive of undiagnosed asthma	369 (46%)
Never had asthma symptoms	424 (52%)
Unknown asthma symptoms	16 (2%)
Missing data	10 (1%)
Ethnicity	
Asian or Indian	559 (39%)
Non-Asian, non-Indian, or unknown	876 (61%)
Born in Australia	
Yes	723 (50%)
No	698 (49%)
Unknown	14 (1%)
Epidemic thunderstorm asthma deaths	10 (<1%)
Age (years)	38·5 (13·1)
Women	3 (30%)
Men	7 (70%)
Asian or Indian ethnicity	6 (60%)

Values are emergency department follow-up data of patients and deaths (mean [SD] or n [%]).

Table: **Demographic characteristics of patients presenting with thunderstorm asthma during Melbourne Nov 21, 2016, epidemic thunderstorm asthma event**

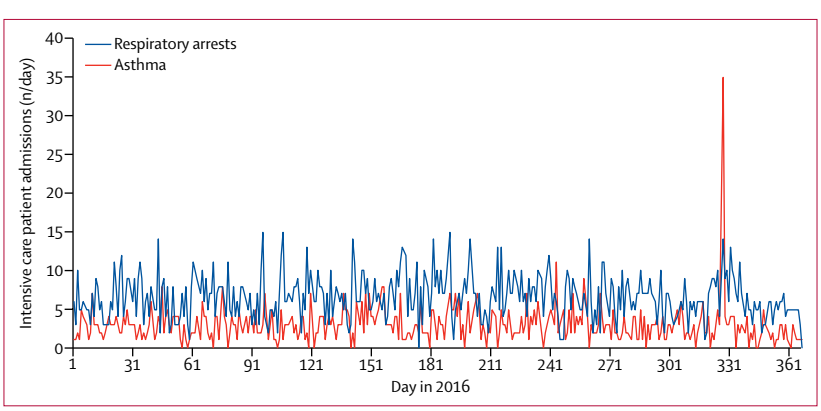

Figure 4: **Daily ICU admissions in Australia in 2016**
Data show admissions to intensive care units (ICU) for asthma (red) and respiratory arrests (blue).

Five patients who were admitted to ICU died (14%), all due to neurological complications associated with cardiac arrest. A further four patients sustained cardiac arrest but survived and were discharged home without neurological impairment. The other five non-ICU deaths were out-of-hospital deaths either while awaiting emergency transport, or who could not be resuscitated by ambulance emergency services. 26 patients required endotracheal intubation (74%), of whom 13 (50%) had a respiratory arrest. Median ICU and hospital length of stay was short at 1·6 (IQR 0·8–3·8) and 2·7 (1·9–5·0) days, respectively. Asian or subcontinental patients were over-represented among those who had a cardiac arrest (five [50%] of ten compared with four [16%] of 25, RR 3·13, 95% CI 1·05–9·31).

Discussion

The convergence of a series of environmental factors resulted in an unprecedented thunderstorm asthma epidemic in Melbourne on Nov 21, 2016. Interaction between a line of thunderstorms, the resulting gust front, and extreme airborne grass pollen concentrations were the probable key environmental factors that culminated in the exposure of a large urban population during their evening homeward commute. Extreme airborne grass pollen concentrations are reasonably rare in Melbourne, and are variable from year to year, with some years experiencing no such days, others just a few days, and yet others as many as ten or more.[14] Several of the environmental features of the event are similar to previous events in Melbourne, such as the occurrence in late spring (November), the involvement of grass pollen, and the association with a thunderstorm-induced gust front or outflow at ground level.[7,8,13] Further, the findings from our study again raise the potential for the allergenic contents of ruptured ryegrass pollen grains to have played a substantial role in this epidemic.[6–8]

Although cumulative daily grass pollen concentrations before Nov 21 generally remained close to average,[15] the days of extreme pollen counts earlier in the month might have contributed to the epidemic by priming grass pollen-sensitised patients.[16] Serological characterisation done at two of our own centres suggests a prevalence for ryegrass pollen sensitisation of between 87–100% among individuals affected by the November, 2016, event, with very large mean skin prick wheals (10–11 mm) and very high mean serum specific IgE (55kIU/L).[17,18] We thus conclude that extreme exposure to rye grass allergen is critically relevant to highly-sensitised pollen-allergic individuals, for whom protective measures can and should be pursued.[19,20]

Although several of the environmental factors leading up to and during this event were extreme, we cannot be certain that these and the other factors we describe fully account for the magnitude and severity of the epidemic, and as yet unidentified factors might have been involved. From the environmental perspective, the role of climate change is worthy of consideration;[21] eg, increasing atmospheric carbon dioxide concentrations and temperatures affecting aeroallergen concentrations.[22,23] There is also suggestion that climate change might extend pollen seasons worldwide, with an increasing likelihood of extreme weather events such as

thunderstorms, the confluence of which might increase the risk of more frequent epidemic thunderstorm asthma events in the future.[24] Other major cities elsewhere around the world that encounter similar combinations of environmental conditions might be similarly unprepared and vulnerable to such an epidemic.

The age distribution of asthma admissions from the 2016 epidemic thunderstorm asthma event saw a departure from a typical U-shaped pattern because of the large increase in patients aged 20–59 years, followed by those younger than 20 years with a preponderance of males. This increase in patients in their middle years rather than at the extremes of life corresponds with the age distribution of allergic rhinitis,[25] a subset of whom will have seasonal rhinitis related to rye grass pollen. The pattern of admissions across age and sex, is likely to also reflect a complex mix of many other potential factors including differing degrees of environmental exposure; differing medical management, treatment adherence, health-seeking behaviours (especially those naive to asthma); differing admission tendencies; and differences in pathophysiology.

The health data presented was limited to the tertiary-care setting; however, increased health-service demand was reported across primary care involving the community telephone health advice service, community pharmacies, and general practice.[15] The patient impact of the Melbourne epidemic thunderstorm asthma event of November, 2016, was by far the largest and most severe to have been documented.[15] The next largest event in Australia was on Nov 29, 1989, in Melbourne, with 277 emergency department attendances, 47 hospital admissions, and 3 ICU admissions.[8] Internationally, an event on June 24, 1994, in the UK was perhaps the next largest, with 104 hospital admissions, five ICU admissions, and one death.[26] The Melbourne 2016 event also far exceeds many other well documented acute allergic asthma epidemics, such as those caused by inhalation of allergenic soybean dust released during the unloading of soybeans into a silo in Barcelona, Spain, in the 1980s.[27,28]

Most patients presenting to emergency departments did not have diagnosed or current asthma, but asthma was present in all ICU admissions and deaths. This suggests that although most patients affected by the epidemic thunderstorm asthma event were asthma-diagnosis naive or were symptom-free for more than a year, the presence of current doctor-diagnosed asthma is a risk factor for severe bronchospasm requiring ICU care and increased mortality. This study more than doubled all combined previous reports of ICU-admitted patients with epidemic thunderstorm asthma (14 patients previously reported),[9] and for the first time describes their demographic characteristics, risk factors, and outcomes. All critically ill epidemic thunderstorm asthma affected patients were young, and by contrast with previous reports,[29] all had known doctor-diagnosed asthma. Although there were five deaths, all other patients (including eight who had a respiratory arrest) survived to discharge without neurological impairment. This result is in keeping with the overall favourable and improving survival seen in Australian ICUs from acute asthma.[30] Duration of ICU and hospital stay was short, supportive of the pathophysiological explanation of an acute allergic precipitant for bronchospasm in epidemic thunderstorm asthma.

Paralleling the documented medical consequences was a commensurate effect on emergency and health service systems. The Emergency Services Telecommunications Authority, Ambulance Victoria, and hospital emergency departments were operating in an environment characterised by rapidly escalating demands and substantial uncertainty.[31] With the cause of the event not known at the time, the pre-hospital and hospital system response was commendable, as was that of the broader community, and this prevented what would otherwise have been an even bigger tragedy. However, important lessons were learnt, and a range of specific and system-wide emergency management recommendations are being implemented. These include the development of epidemic thunderstorm asthma forecasts and technological and other systems for improved situational awareness, and health system responsiveness and stewardship.[31]

All our datasets revealed a strikingly consistent predominance of Asian or Indian ethnicity, with increased risks for emergency department presentations (based on self-identified ethnicity), hospital or ICU admissions (based on country of birth data), and case fatality (based on clinical documentation). This was an unexpected finding that raises important questions but there are important caveats. Confounding factors might have played a role such as poor education and knowledge about asthma in migrant populations, varying cultural beliefs about asthma, unequal access to medical care and variable adherence with preventer medications. Conversely, ethnicity in admitted and critically ill patients was not explored beyond country of birth and it is possible that the effect of ethnicity in the hospital admission or ICU cohort was underestimated in Australian-born patients of Asian or Indian background. Self-identified ethnicity on our questionnaire might also be subtly different to the ancestry question in the national census. However, they have been used interchangeably in genetic research[32] and we believe this interpretation of census data is reasonable, especially in relation to Asian or Indian ethnicity or ancestry, supported by the strength and consistency of our findings across datasets.

Atopy and asthma result from the effects of environmental factors on genetically susceptible individuals. Lifestyle and environmental factors in high-income, industrialised countries facilitate atopy and asthma in migrants, who are more susceptible to the development of allergies than the local population with a time-dependent effect, affected by age at the time of

immigration.[33,34] Possible mechanisms for this loss of immune tolerance in migrants include: the hygiene hypothesis associated with an altered microbiome; impaired immunoregulatory effects of relative vitamin D deficiency mediated via the role of skin pigmentation and sun exposure; and novel allergen exposure in genetically susceptible populations. The increased susceptibility of non-white populations to the adverse effects of so-called westernisation has substantial global implications with progressive urbanisation and migration from the developing to the developed world.[35,36] Asian immigrants to Australia appear particularly susceptible to this migration effect of increased allergy and asthma risk. Studies have documented a significantly higher prevalence of seasonal allergic rhinitis in Asians living in Australia (whether immigrant or Australian-born) than in Australian-born non-Asians.[37] The prevalence of hay fever and to a lesser degree, asthma in Asian immigrants, increases significantly with the length of stay in Australia, independent of age at arrival, sex, and atopic status.[38] After 10 years in Australia, up to 60% of south-east Asian immigrants developed hay fever and 15% had symptoms of asthma.[39] Our study findings suggested that the thunderstorm asthma event substantially exposed this migration allergy risk with a massive small airways allergen challenge resulting in tragic consequences. This has important public health implications for risk mitigation in a target population, and is of particular relevance to other locations prone to epidemic thunderstorm asthma events with coexisting large numbers of Asian immigrants, such as the UK. The largest previous epidemic thunderstorm asthma event occurred in the Greater London area of UK, which has among the highest proportion of British Asian migrants in the UK.

In conclusion, convergence of environmental and patient factors triggered a thunderstorm asthma epidemic of unprecedented severity on Nov 21, 2016, in Melbourne, Australia. The event has created a new benchmark for emergency and health service escalation. Our findings provide vital public health lessons applicable to management of epidemic thunderstorm asthma and highlight the importance of predicting future events, coordinating health care responses, protection of at-risk asthma populations, and optimal medical management of epidemic thunderstorm asthma.

Contributors

FT, JMD, PJB, DC, JDa, and MH planned the study collaboration. PJB was the environmental section lead. DC was the public health section lead. JDa was the intensive care unit section lead. MH and FT did the emergency department follow-up. All authors helped to recruit patients and obtain or analyse data. FT, PJB, DC, JDa, MH, JMD, PGB, and CG analysed and interpreted data and drafted the manuscript. All authors approved the final version of the manuscript.

Declaration of interests

We declare no competing interests.

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