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Do levels of airborne grass pollen influence asthma hospital admissions?

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Clinical and Experimental Allergy

Summary

Background The effects of environmental factors and ambient concentrations of grass pollen on allergic asthma are yet to be established.

Objective We sought to estimate the independent effects of grass pollen concentrations in the air over Melbourne on asthma hospital admissions for the 1992–1993 pollen season.

Methods Daily grass pollen concentrations were monitored over a 24-h period at three stations in Melbourne. The outcome variable was defined as all-age asthma hospital admissions with ICD9-493 codes. The ambient air pollutants were average daily measures of ozone, nitrogen dioxide and sulphur dioxide, and the airborne particle index representing fine particulate pollution. Semi-parametric Poisson regression models were used to estimate these effects, adjusted for air temperature, humidity, wind speed, rainfall, day-of-the-week effects and seasonal variation.

Results Grass pollen was a strong independent non-linear predictor of asthma hospital admissions in a multi-pollutant model ($P = 0.01$). Our data suggest that grass pollen had an increasing effect on asthma hospital admissions up to a threshold of 30 grains/m^3, and that the effect remains stable thereafter.

Conclusion Our findings suggest that grass pollen levels influence asthma hospital admissions. High grass pollen days, currently defined as more than 50 grains/m^3, are days when most sensitive individuals will experience allergic symptoms. However, some asthmatic patients may be at a significant risk even when airborne grass pollen levels are below this level. Patients with pollen allergies and asthma would be advised to take additional preventive medication at lower ambient concentrations.

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Introduction

The prevalence of asthma and allergic disorders continues to increase world-wide [1, 2]. In Australia alone, the prevalence of current asthma among adults ranges from 10% to 12%, and among children it ranges from 14% to 16%. These percentages are high by international standards, and have continued to increase since the early 1990s [3]. Asthma thus represents an increasing disease burden on global health care systems, as more resources are allocated to management programmes for effective day-to-day control. This notable increase, particularly in allergic asthma, may be explained in part by changes in environmental factors such as indoor and outdoor air pollution [4, 5]. Higher ambient concentrations of pollen [6, 7] and climatic factors may interact with these changes, such as higher temperatures, humidity and regular thunderstorms [8, 9]. Pollen grains can be inhaled, but are too large to penetrate the lower airways [10]. However, some pollen allergens found on small respirable particles, combined with other environmental and lifestyle factors, may lead to the exacerbation of asthma or may trigger symptoms in persons predisposed to allergic

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disorders [11, 12]. Numerous epidemiological studies have found associations between IgE specific to particular pollen types as demonstrated by skin tests, and symptoms of asthma and hayfever [13, 14]. Furthermore, evidence from epidemiological studies suggests associations between exposure to outdoor aeroallergens and the onset of symptoms, particularly in children [15–17].

Although the adverse effects of air pollutants on asthma are well established [18], the role played by pollen is poorly understood. At present, there is limited evidence on the effect of the interaction between air pollution and pollen (particularly grass pollen) on allergic asthma. Such evidence that does exist shows contradictory effects. For example, in a large time series study of air pollution and daily hospital admissions for asthma in London, UK, Anderson et al. [19] found no independent or combined effect of three different pollen types (grass and two trees, birch and oak) with air pollution on admissions. In contrast, two more recent studies report both linear and non-linear independent effects of three different types of allergenic pollen (grass and two weeds: *Urticaceae* and *Plantago*) on asthma emergency admissions in Madrid, Spain, with grass pollen registering the strongest association of the three [6, 7].

Melbourne, Australia, is one of the few cities in the world where studies of pollen allergens have consistently shown that exposure to different types of airborne pollen, such as rye grass and birch, can act as triggers for allergic asthma [15, 20, 12]. Although other time series studies in Melbourne have focused on air pollution and mortality [21], hospitalizations for cardiovascular disease [22] and childhood asthma admissions [23], none has investigated the joint associations between pollen and air pollution on asthma admissions. Therefore, there was the opportunity to conduct a time series study to evaluate the relationship between ambient concentrations of grass pollen in the air and asthma hospital admissions. In this study, we sought to estimate the effect of ambient concentrations of grass pollen in the air over Melbourne on asthma hospital admissions, for one grass pollen season.

Methods

Study design

This was an ecological study, which related routinely collected data on hospital admissions for asthma and atmospheric grass pollen data collected at three monitoring stations in Melbourne, Australia, for the grass pollen season of 1992–1993.

Data

Daily asthma hospital admissions (ICD9-493) for all ages were obtained from the Health Department Victoria for short-stay public hospitals in Melbourne, during the period from 27 September 1992 to 20 February 1993. Airborne grass pollen counts were obtained from the School of Botany, The University of Melbourne [24]. Measurements of average pollen concentrations in grains/m^3 for a 24-h period were made using Burkard volumetric traps as detailed elsewhere [25] located at the University of Melbourne, Footscray and Box Hill.

Air pollution data were obtained from the Environment Protection Authority, which operates nine routine monitoring stations located across Melbourne. The average daily concentrations (derived from hourly maximum values) of ozone (O_3), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2) in parts-per-hundred million (p.p.h.m.) were used in the study. We used the airborne particle index (API) from nephelometry to represent fine particulate pollution. API is derived from B$scat \times 10^{-4}$, detecting particulates between 0.1 and 1 μm in aerodynamic diameter. Daily average temperature, rainfall, wind speed and relative humidity were measured at four routine monitoring stations operated by the Bureau of Meteorology in the Melbourne metropolitan area.

Statistical Methods

To derive the pollen season for this study, we used daily airborne levels of grass pollen for the period from 27 September 1992 to 20 February 1993. A semi-parametric Poisson regression model was used to model the daily counts of asthma hospital admissions. Possible non-linear associations were fitted using a generalized additive approach.

We first began with a core model with grass pollen as the exposure variable, and each pollutant entered separately (single-pollutant models). We fitted non-parametric smoothing terms to all variables to assess their linearity. Any variables found to be linearly associated with asthma hospital admission were subsequently modelled as parametric terms in the multi-pollutant model. All models (single and multi-pollutant) were adjusted for day-of-the-week effects (entered as an indicator variable with Sunday as the reference day) and a smoothed function for the day of the study, daily air temperature, relative humidity and wind speed.

All smoothed terms are represented using penalized regression splines, with smoothing parameters selected by the Unbiased Risk Estimator (UBRE) procedure, using the GAM function in the mgcv package of R. The results are presented as smoothed terms in all single-pollutant models, where the degree of smoothness is represented by the degrees of freedom (df). If the corresponding df is 1, then parametric linear terms are fitted in the subsequent multi-pollutant model. Diagnostic plots were checked for model fit and the distribution of residuals. All statistical tests were two-sided, with the significance level chosen to

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Table 1. Summary statistics for grass pollen, air pollutants and meteorological variables

				Percentiles							
	Mean	SD	Minimum	5th	10th	25th	50th	75th	90th	95th	Maximum
Grass pollen/m^3	37.34	55.51	0.00	1.67	2.67	5.33	12.33	46.67	101.67	169.17	255.33
NO_2 p.p.h.m.	9.61	5.28	2.27	3.10	3.84	5.37	8.04	12.78	17.24	19.73	21.17
SO_2 p.p.h.m.	1.05	0.62	0.04	0.22	0.33	0.61	0.95	1.41	1.92	2.17	3.66
O_3 p.p.h.m.	17.05	5.75	7.58	10.28	11.63	13.04	15.75	19.44	24.80	27.68	40.55
Air particle index	0.73	0.34	0.36	0.40	0.43	0.47	0.61	0.91	1.26	1.48	1.97
Rainfall on wet days*	6.87	9.28	0.04	0.04	0.08	0.33	3.15	8.57	22.62	26.58	41.62
Air temperature	17.40	4.40	9.30	11.10	11.40	13.90	17.40	20.20	23.30	24.70	30.9
Wind speed km/hr	7.25	3.05	2.65	3.62	4.00	4.92	6.44	8.92	11.79	14.00	16.32
Relative humidity %	69.28	9.53	30.81	51.83	59.17	64.63	68.72	74.54	81.23	84.44	93.46

*There were 33 dry days (rainfall = 0) and 114 wet days (rainfall >0).
SO_2, sulphur dioxide; NO_2, nitrogen dioxide; O_3, ozone

Table 2. Single air pollutant models*

	Smooth term df	χ^2	*P*-value
Grass pollen/m^3	5.912	24.81	0.003
Grass pollen/m^3	4.5	15.72	0.01
NO_2 p.p.h.m.	1.0	3.47	0.06
Grass pollen/m^3	5.9	19.30	0.003
SO_2 p.p.h.m.	1.0	0.53	0.5
Grass pollen/m^3	5.9	19.07	0.004
O_3 p.p.h.m.	1.0	0.02	0.9
Grass pollen/m^3	6.0	18.71	0.005
Air particle index	1.74	4.89	0.07

*Every model is adjusted for days of the week, days, air temperature, humidity, and wind speed; the average grass pollen concentration was restricted to be <150. df, degrees of freedom; p.p.h.m., parts-per-hundred million; SO_2, sulphur dioxide; NO_2, nitrogen dioxide; O_3, ozone.

Table 3. Multi-pollutant model*

	Parametric coefficient	Standard error	*P*-value
NO_2 p.p.h.m.	0.02	0.007	0.002
SO_2 p.p.h.m.	−0.03	0.03	0.2
O_3 p.p.h.m.	0.006	0.004	0.2
	Smooth term df	χ^2	*P*-value
Grass pollen/m^3	5.1	13.49	0.01
Air particle index	1.8	9.05	0.009

*Adjusted for days of the week, days, air temperature, humidity, and wind speed; the average grass pollen was restricted to <150.
p.p.h.m., parts-per-hundred million; SO_2, sulphur dioxide; NO_2, nitrogen dioxide; O_3, ozone; df, degrees of freedom.

be 5%. All statistical analyses were performed using R version 2.3.1.

Results

A total of 147 daily asthma hospital admissions were recorded during the pollen season (mean 40.31 ± 18.01 SD). Summary measures of grass pollen levels, pollutant concentrations and meteorological variables are shown in Table 1. Previous studies have suggested that all sensitive individuals will experience allergic symptoms on days when the grass pollen count is 50 grains/m^3 or above [26]. By this standard, roughly 25% of the days in the study period would be considered high pollen count days (i.e. >50 grass pollen grains/m^3), and 10% would be classified as extremely high days (>100 grass pollen grains/m^3); see [24].

Grass pollen was an independent predictor of daily asthma hospital admissions in single-pollutant models (Table 2). Its effect was consistently non-linear and significant. The estimated df of smoothed grass pollen was 5.9 ($P = 0.003$) when fitted without any air pollutants; it decreased to 4.5 ($P = 0.01$) when fitted with NO_2, and remained around 5.9 when fitted with SO_2, O_3 and API ($P = 0.003$, 0.004 and 0.005, respectively). The levels of SO_2 and O_3 were not significantly associated with daily asthma hospital admissions during the pollen season ($P = 0.5$ and 0.9, respectively). Linear effects were observed for both NO_2 and the API, but both were only marginally significant ($P = 0.06$ and $P = 0.07$). The slight decrease of the smoothing term (df) and a decrease of the *P* value of grass pollen when fitted with NO_2 may suggest some confounding effect between the two predictors.

The results of fitting multi-pollutant models are displayed in Table 3. Grass pollen remained a significant non-linear predictor (df of the smooth term = 5.1) of daily asthma hospital admissions after the inclusion of all pollutants ($P = 0.01$).

The non-parametric spline fit of grass pollen [with 95% confidence interval (CI)] on asthma hospital admissions is

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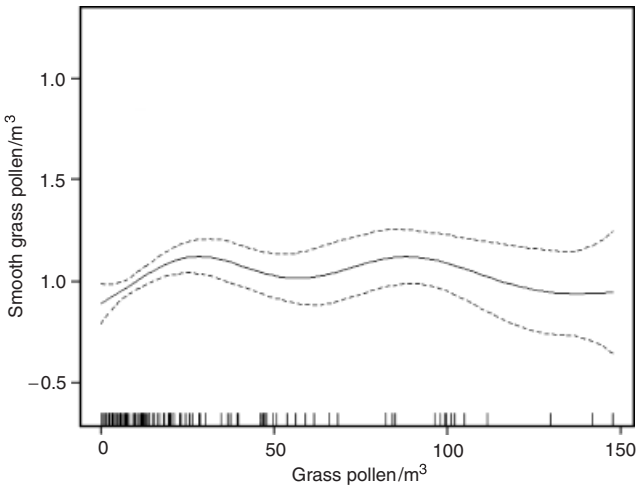

Fig. 1. Non-linear plot produced by GAM for the average grass pollen in the multi-pollutant model. Adjusted for days of the week, time trend, air temperature, humidity and wind speed; grass pollen was restricted to <150.

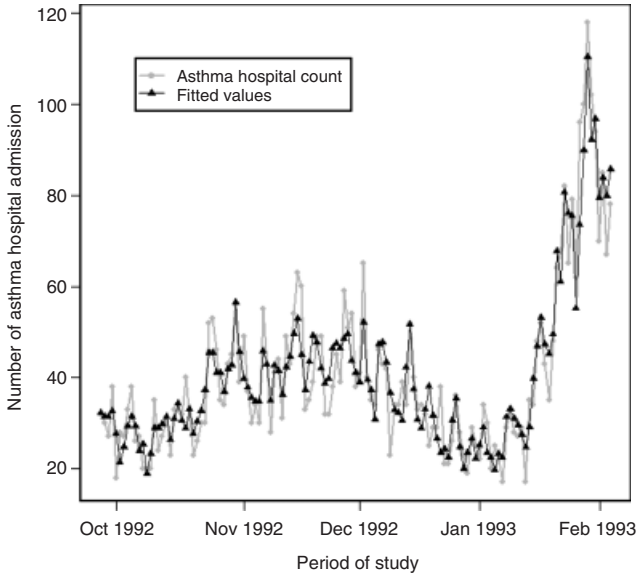

Fig. 2. Daily asthma hospital admissions during the Melbourne grass pollen season from 27 September 1992 to 20 February 1993 (grey line). The fitted data (black line) represent the additive multi-pollutant model.

displayed in Fig. 1. The observed pollen data are plotted as tick marks on the *X*-axis, and the *Y*-axis represents the estimated smooth fit of pollen on asthma admissions. The plot suggests that grass pollen has an increasing effect on asthma hospital admissions up to a threshold of about 30 grains/m^3. The increase over this range is 0.23 on the log scale, that is, 26% more admissions per day occur at 30 grains/m^3 than when there is no pollen. The width of the confidence intervals thereafter suggests that the effect of pollen on asthma is relatively constant for higher levels of grass pollen.

The time series of daily asthma admissions is shown in Fig. 2. There was a substantial increase in admissions in February, which may represent a 'back to school effect'. The black line shows the additive multipollutant model, which fitted the observed counts quite well.

The estimated effect of the API indicates some departure from linearity (df = 1.8, $P < 0.009$). As in the single-pollutant model, the Poisson regression model suggests that NO_2 was significantly predicting asthma hospital admissions, and that NO_2 was linearly associated with log-scaled daily asthma hospital admissions, with an estimated slope of 0.02 (95% CI = 0.006–0.034; $P = 0.002$) during the grass pollen season. No serial correlation was detected by the autocorrelation function (ACF) of the residuals.

Discussion

The aetiology of asthma is multifactorial, and is not yet fully understood [27]. The combination of genetic and lifestyle factors that may contribute to the development of asthma is under continuing investigation. On the other hand, the adverse effects of environmental factors such as air pollution and weather changes on asthma have long been established. Likewise, the adverse effects of aero-allergens such as pollen grains on respiratory disease, particularly in children, were implied as early as the 1970s [26].

In this study, we have found evidence of ambient levels of grass pollen having non-linear effects, independent of the effects of key pollutants such as NO_2, O_3 and air particulates, and allowing for the confounding effects of meteorological variables. These findings suggest that ambient concentrations of grass pollen have adverse effects on asthma hospital admissions. In particular, we have found that pollen reaches its maximum effect at 30 grains/m^3, with further increases in pollen levels appearing to have no further significant impact on asthma admissions. Thus, while the generally accepted classification of >50 grass pollen grains/m^3 being a high pollen day [24] remains a useful way of presenting pollen data to the general public, the notification of days when pollen levels are below this (>30 grass pollen grains/m^3) is potentially important for some groups in the community, such as asthmatics, as well as the medical services (e.g., hospital emergency wards) that are likely to receive these patients.

This is the first study in Australia to demonstrate an association between asthma morbidity and ambient concentrations of grass pollen using a semi-parametric time series regression approach. Pollen counts are already being broadcasted in television weather reports and printed in newspapers during the pollen season. More targeted messages could perhaps be directed by new media such as SMS to people with asthma for whom grass

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pollen is a known trigger. They could be encouraged to increase their preventive medication on days with >30 grass pollen grains/m^3. In order to develop such strategies, we must be able to forecast the next day's grass pollen count accurately. To this end, we have already shown that daily grass pollen counts during the pollen season can be predicted using meteorological variables as potential covariates and a semi-parametric regression-type approach [28].

Few studies have investigated the inter-relationship between air pollution and pollen on respiratory disease. Our findings are in agreement with two recent time series studies of air pollution and pollen on asthma morbidity [6, 7]. Both studies suggest adverse effects of ambient concentrations of pollen, independent of pollution effects and the confounding effects of temperature and relative humidity. Moreover, Tobias et al. [7] demonstrated consistent statistically significant non-linear effects of three different types of pollen, *Plantago* (the weed plantain), *Poaceae* (grass) and *Urticaceae* (a family that includes pellitory, a common weed), on asthma-related hospital emergency admissions. A similar earlier study of grass pollen and asthma morbidity in UK populations [29] found moderate associations between grass pollen and asthma morbidity, but stronger delayed effects after 1–3 days.

However, our findings on grass pollen differ from those reported by others. Using Spearman Rank correlations, Anderson et al. [30] found no significant positive association between grass pollen (*Poaceae*) and asthma admissions. Anderson et al. [19] actually found negative effects of grass pollen on daily asthma admissions for the 0–14-year-old age group. Although the statistical methods they used are widely accepted in the air pollution literature [31], they do not adequately capture the possible non-linear effects, similar to those observed in our study.

A number of limitations should be considered when interpreting the results of this study. The time period under investigation was short, covering only one pollen season. Hence, the power to detect an effect is very low, and any observed effect may be due to chance. With limited data available, we were unable to investigate potential confounders such as holiday (public and school) effects. Also for this reason, we were unable to evaluate any of the possible important lagged effects of pollen on asthma hospital admissions, as observed in other studies [19, 29, 7]. Nevertheless, the findings from this study demonstrate consistent effects of grass pollen, in both single- and multi-pollutant models, even after adequate adjustments for the confounding effects of trend, day of the week and meteorological variables. Moreover, our findings on NO_2 are consistent with other time series studies of air pollution in Melbourne, Australia [22, 32].

Interpretation of these data would be compromised if the grass pollen counts did not reflect the levels of airborne grass pollen across the city, due to inappropriate placement of the stations or other similar reasons. While grass pollen was only measured at three sites, these sites were well spread across the city, from Box Hill in the east to Footscray in the west, with one site on the main university campus in central Melbourne. The Box Hill and Footscray sites were separated by about 20 km, and all sites were positioned to conform to Australian Standard AS 2922–1987. Pollen counts at the three sites were also highly correlated, suggesting that grass pollen, which predominantly comes from pastures situated north of Melbourne [25], disperses more – or less evenly across the city.

There were also limitations in the hospital admissions data, which were collected for administrative and financial purposes rather than for research. Data capture was complete for public hospitals, but not all private hospitals contributed at that time. Furthermore, cases of asthma in emergency departments that did not result in hospital admission were not captured. The principal diagnosis was coded according to ICD9-CM, which distinguished between asthma (493) and COPD (496). Fortunately, the period of the study was before the introduction of Diagnosis Related Groups (DRGs), which provided financial incentives for hospitals to reclassify asthma admissions to other more remunerative DRGs. Furthermore, we had no information on patients' sensitization to pollen or individual exposure data. However, such lack of information would have misclassified the exposure status non-differentially, pushing the estimates towards null, which could not explain our positive findings.

In this study, we have estimated the association between levels of airborne grass pollen and asthma hospital admissions, after adjusting for other pollutants and meteorological variables. This association is likely to be stronger among those with a sensitization to pollen allergy. However, given that the association was found among asthmatics irrespective of their sensitization status, secondary prevention strategies such as warning the part of the population with asthma regarding high pollen days may be justified. The high-pollen day definition currently used is of hayfever (seasonal rhinitis) symptoms and is based on a single study by Davies and Smith [26] conducted in central London over 30 years ago. They reported that when the mean daily concentration of grass pollen was 50 or more per cubic metre, all people (in central London) who were clinically sensitive to grass pollen experienced symptoms (of seasonal rhinitis). Our study is investigating asthma hospital admissions, and there is no reason to expect the 100% symptom level for hayfever to be the same as that for asthma.

Our modelling suggests that pollen reaches a maximum effect at 30 grains/m^3, and beyond this appears to have no additional impact on asthma admissions. It is plausible that this effect is due to a small number of asthma patients who are highly responsive to grass pollen. That is, a

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proportion of this group will require hospitalization at low pollen counts and this proportion increases as grass pollen increases up to 30 grass pollen/m^3, thus removing the individual from the susceptible pool. Higher levels of exposure might sensitize additional individuals, but they could not mount an immune response to pollen for at least 2 weeks.

It is imperative that a routine surveillance programme that frequently monitors pollen levels be implemented, so that early warnings are set in place for populations susceptible to pollen allergens. Epidemiological exposure studies are required to examine this threshold hypothesis. Furthermore, larger studies are necessary to develop models to accurately forecast next day pollen counts, further quantify the role of outdoor aeroallergen levels on asthma, identify seasonal patterns and allow an adequate estimation of dose–response relationships. Moreover, these studies could also address the modifying effects of climate change on outdoor aeroallergen–asthma associations.

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