Allergies in the workplace

AIR POLLUTION, POLLENS AND CHILDHOOD ASTHMA – IS THERE A LINK?

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INTRODUCTION

Childhood asthma is the most common chronic disease in children globally and ranks among the top 20 contributors to global disability-adjusted life years (DALY) in all children; it also ranks among the top ten causes of DALY in 5–14-year-olds.1 The growing increase in the prevalence of childhood asthma symptoms has been correlated with the global increase in outdoor air pollution.2 Ambient air pollution is a major environmental health concern globally that affects populations in highly industrialised and developing countries. As recently documented by the World Health Organisation (WHO), 98% of cities in low- and middle-income (LMI) countries with more than 100 000 inhabitants do not meet WHO air-quality guidelines, whereas in high-income countries the percentage decreases to 56%.3 According to recent global burden of disease indicators, the estimated ambient air pollution (especially particulate matter) was responsible for 5.5 million deaths per year in 2013 and 141.5 million DALY (14% of global total DALY); in terms of DALY, it is the fifth leading global risk factor.4 The WHO has reported 3.7 million premature deaths in both urban and rural areas caused by air pollution, which is due mainly to small particulate matter of 10 microns or less in diameter (PM_{10}). LMI countries, in which air-pollution emissions from power plants, traffic, open waste burning and other combustion sources are very common, account for approximately 88% of these deaths.5

While the relationship between ambient air pollutants and their role in the development of asthma continues to remain a subject of detailed study, with our increasing understanding of the implications of climate change and global warming, a link has been postulated between ambient air pollutants and airborne pollens in the increasing prevalence of allergic airway diseases, including the exacerbation of asthma and the role of air pollutants in modifying the effect of pollen exposures. Pollen allergy has been used more recently to study the interrelationship between ambient air pollutants and respiratory allergy, including asthma in sensitised individuals.6

The aim of this article is to identify the mechanisms involved in the interrelationship between air pollutants...
and pollen allergens with asthma and to summarise the evidence for the independent and/or co-effect of environmental exposures (ambient air pollutants and pollens) on childhood asthma.

**AMBIENT AIR POLLUTANTS**

Ambient air pollutants commonly arise from primary and secondary sources. The primary pollutants are emitted directly from sources that include gaseous pollutants – for example, sulphur dioxide ($\text{SO}_2$) and mono-nitrogen oxides ($\text{NO}_x$) as well as particulate matter (PM) (e.g. soot). The secondary pollutants arise from primary pollutants in the atmosphere that combine with UV-rays from sunlight and/or moisture (e.g. ozone ($\text{O}_3$), secondary particles such as sulphates). The air pollutants referred to in this article are the four key primary sources – PM, $\text{SO}_2$, nitrogen dioxide ($\text{NO}_2$) and $\text{O}_3$ – identified by the WHO 2005 Air Quality Guidelines (AQG), reviewed previously.1

**ALLERGENIC POLLEN AND POLLEN ALLERGY**

Although airborne pollens comprise a small proportion of the atmosphere, they are important causative agents of allergic respiratory disease in sensitised individuals. Allergic respiratory disease has become a public health problem due to the increased prevalence of airborne allergenic pollen causing clinical disease and resulting in escalating costs to patients and society at large.7 Globally, the common non-animal-related allergens that are common sensitisers are pollens from trees, weeds, grass and fungal spores. Important grass-pollen allergens are Cynodon dactylon (Bermuda grass) and Lolium perenne (Rye grass). These tropical grasses are commonly found along latitudes between 30°N and 31.4° + 7.5°S and have their optimal growth at temperatures between 24°C and 37°C. Bermuda grass has been linked to allergic conjunctivitis, asthma, seasonal allergic rhinitis exacerbation and asthma.8 Grass-pollen allergy is also a common respiratory allergy (median prevalence of 16.9%) in most European countries and also in South Africa.8 Approximately 80% of South Africans react to Eragrostis (lovegrass) and Buffalo grass pollens.10 Besides grass, birch (mostly in east-central Europe) and ragweed (predominantly in northern countries) are among the major pollens responsible for causing rhinitis and asthma. Cypress and Parietaria (wall pellitory) also play a major role in southern countries.

**INTERACTION BETWEEN AIR POLLUTANTS AND POLLEN IN ASTHMA AETIOLOGY**

Exposure to single pollutants has been the focus of many studies on respiratory and allergic risk factors. However, multiple exposures to several pollutants occur in ‘real life’. There are always complex mixtures of pollutants from various sources in the environment, which often contribute jointly to synergistic or additive toxic effects.11,12 Owing to the intrinsic electrostatic properties and porous surfaces of ambient respirable PM, they readily adhere to airborne allergens released from pollens. PM interacts with aero-allergens via modulation of the allergenicity of airborne allergens, in this way promoting airway sensitisation.13,14 Phleum pratense pollen releases more allergen-containing granules when treated with various concentrations of $\text{O}_3$ and $\text{NO}_2$ under experimental conditions compared to exposure to air only (see Figure 3).15 The bio-availability of airborne pollen allergens has also been identified to increase owing to the effect of traffic-related pollutants.15 Air pollutants may increase acute responses to allergens in various ways:

- increasing epithelial permeability;
- ‘priming’ allergen-induced responses caused by airway inflammation, in this way enhancing the recruitment and activation of inflammatory cells;
- increasing airway oxidative stress, and
- increasing the release of neuropeptide.7

The access of inhaled allergens to the immune system may be facilitated by impaired mucociliary clearance and airway mucosal damage, which enhances atopic sensitisation risk and symptoms exacerbation in sensitised individuals.16,17

**MECHANISMS BY WHICH AIR POLLUTANTS CAUSE ASTHMA**

The development of asthma has been better understood owing to the availability of techniques for investigating gene polymorphisms and variants associated with the increased risk of asthma. There are four groups of genes attributed to conferring susceptibility to the development of
asthma. They are those controlling:
- airway repair and remodelling, including airway development;
- immune system responses;
- bronchial hyperresponsiveness, and
- the endogenous production of anti-oxidants in the airways (see Figure 4).

Various mechanisms enable either different gene types on their own or those interacting with environmental exposures (pollutants and allergens) through gene-environment interactions to cause asthma. These mechanisms include oxidative stress and damage, airway wall remodelling, immunological effects and inflammatory pathways, and the enhancement of respiratory sensitisation to allergens. It is postulated that air pollution increases the likelihood of sensitisation to allergens by:
- acting as a carrier for allergens to the lower respiratory tract and parts of the lungs that would otherwise be difficult for allergens to reach;
- increasing the permeability of the epithelium and thereby increasing the surface area of immunological cells responding to allergens;
- interacting with antigenic protein and in this way increasing its potency, and
- air pollutants (especially PM), acting as adjuvants, serving as a depot and in this way preventing antigens from dispersing.

During this process, PM stimulates the division of Type-2-helper T cells or activates antigen-presenting cells.

EXPOSURE-RESPONSE STUDIES BETWEEN AMBIENT AIR POLLUTANTS AND EXACERBATION, PREVALENCE AND INCIDENCE OF CHILDHOOD ASTHMA

In the literature, the evidence for the association between ambient air pollution exposure and asthma prevalence or asthma induction in children is inconclusive. A systematic review by Anderson et al of 21 studies on the prevalence of wheeze symptoms or asthma diagnosis and levels of air pollution in five or more communities demonstrated that only 11% of studies showed a positive, statistically significant association between the pollution-outcome estimates. Epidemiological evidence from a pooled analysis of five European birth-cohort studies – GINI and LISA (both Germany, divided into north and south areas), MAAS (England, United Kingdom), PIAMA (the Netherlands), and BAMSE (Sweden) – showed no association with an increased prevalence of childhood asthma, while some suggested effects on only a sub-population of age-groups and girls. The results of a systematic review and a meta-analysis by Bowatte et al of pooled five birth-cohort studies (PIAMA, Oslo, BAMSE, Vancouver and BCO) showed a modest association between the incidence of asthma and longitudinal childhood exposure to NO₂: OR 1.09; 95% CI 0.96 to 1.23 per 10 µg/m³ increase in pollutant. However, the association was highly variable between all the studies pooled together, with a heterogeneity of 75.5%. A recent study evaluating the association with traffic-related pollution found no association between incidence of asthma with the measured pollutants at baseline and follow-up. However, there is evidence from epidemiological studies that exposure to ambient air pollutants in early childhood plays an important role in the exacerbation of asthma and other respiratory symptoms, with a greater effect being observed in asthmatic children.

EXPOSURE-RESPONSE STUDIES BETWEEN AIR POLLUTANTS AND AIRWAY INFLAMMATION AND LUNG FUNCTION DEFICITS IN CHILDHOOD ASTHMA

AIRWAY INFLAMMATION

There is growing evidence of an association between ambient air pollution and increased exhaled nitric-oxide (FeNO) levels, a subclinical marker of airway inflammation in children. In the Southern California Children’s Health Study (SCCHS), higher FeNO levels were associated with both background and traffic-related pollutants. Among children aged seven to 11 years, cumulative lagged averages of daily O₃ (over 1–23 days), PM₁₀ (over 1–7 days), and PM₂.₅ (over 1–8 days) were associated with 14.3% (p<0.01), 9.3% (p<0.05), and 17.4% (p<0.001) higher FeNO levels across an IQR of 15.42 ppb of O₃, 12.97 µg/m³ of PM₁₀ and 7.5 µg/m³ of PM₂.₅. Similarly, longitudinal analysis of chronic exposures to PM₂.₅ and NO₂ (scaled to the IQR of 2.4 µg/m³ and 1.8 ppb, respectively) showed that PM₂.₅ and NO₂ exposures were associated with 4.94 ppb (p = 0.005) and 2.29 ppb (p = 0.02) increase in FeNO, respectively.

LUNG FUNCTION DEFICITS

There is more consistent evidence for the role of ambient air pollution exposure, particularly traffic-related pollutants, in reducing lung function, especially in young children. A large proportion of epidemiological studies found a consistent association between ambient air pollution exposure and reduced lung function in children, with three studies showing mixed results. Some studies demonstrated significant associations with only traffic-related pollutants such as NO₂, PM₂.₅ and PM₁₀ in younger children and non-asthmatics and boys. Findings from the SCCHS were consistent in demonstrating a significant positive association with lung-function deficits.
For example, exposures to NO₂, PM₁₀, elemental carbon (EC), and acid vapour were significantly associated with reduced FEV₁ in children followed from age ten to 18 years. Similarly, the proportion of low FEV₁ (defined as the percent FEV₁ below 80%) was four times greater in children from communities with the highest level of PM₂.₅ compared to those with the lowest level (7.9% vs 1.6%, p = 0.002).⁴⁶

**EXPOSURE-RESPONSE STUDIES BETWEEN POLLEN EXPOSURE AND THE EXACERBATION, PREVALENCE AND INCIDENCE OF CHILDHOOD ASTHMA**

The association between pollens and the onset of allergic disease and exacerbations has also been reported in various studies (Table I).⁴⁷,⁴⁸ however, inconsistencies
were evident in the reported associations in these studies. Positive correlations between cedar-pollen counts and asthma in six- to seven-year-old children have been reported in the Japanese ISAAC study.\(^{47}\) In contrast, no association between increased prevalence of asthma and airborne pollen was found in a Hungarian study.\(^{48}\) In a large international epidemiological study,\(^{50}\) grass-pollen allergy was associated with seasonal asthma exacerbations, with elevated risks for sensitised participants in early summer in southern Europe (OR March/April = 2.60, 95% CI: 1.70–3.97; OR May/June = 4.43, 95% CI: 2.34–8.39). In northern Europe, a similar association was observed for birch-sensitised individuals (OR May/June = 2.94, 95% CI: 1.92–4.50; OR July/August = 2.01, 95% CI: 1.38–2.94). A significant association was also observed between grass pollens and hospital visits for asthma attacks in a French study,\(^{51}\) with an interquartile range increase of 17.6 grains/m\(^2\) of the Poaceae grass family for a 54% increased risk of asthma attacks. However, three of the reviewed studies showed no association between pollen exposure and asthma exacerbation in children (see Table I).\(^{52–54}\)

With regard to asthma incidence, a nine-year German longitudinal study\(^{49}\) found a significant association between new onset of asthma with previous sensitisation to grass pollen (RR 1.79, 95% CI: 1.01–3.19). In an Australian birth cohort study,\(^{55}\) cumulative exposure to pollen between four and six months was associated with asthma among 620 children aged six to seven years (OR 1.4 95% CI: 1.1–1.7). Similarly, in a birth-cohort study of 514 children followed from birth to 24 months of age, children exposed to pollen and spores in the first three months of life were reported to be at increased risk of early wheezing, independent of other seasonal factors, lower respiratory infections and ambient air pollutants.\(^{56}\)

**EXPOSURE-RESPONSE STUDIES BETWEEN CO-EXPOSURE TO AIR POLLUTANTS AND POLLEN AND ASTHMA**

At the population level, little is known about the possible synergistic effect between allergens and air pollutants since this association is difficult to analyse in uncontrolled settings. The current knowledge of the pathogenesis of allergies and asthma due to combined exposure to biological agents (e.g. pollen, mites) and air pollutants has been based primarily on animal or in vitro laboratory studies.\(^{2}\) Several clinical laboratory studies have demonstrated that, following prior exposure to ambient air pollutants, there was a greater effect of allergen challenge on asthma.\(^{57,58}\) In 2015, Brandt et al demonstrated a significantly higher house-dust mite (HDM)-specific memory T cells in the lungs of mice exposed to both HDM and diesel exhaust particles (DEPs) compared to mice exposed to only saline, DEPs or HDM alone.\(^{57}\) The authors’ hypothesised that early-life exposures to DEP may increase the risk of allergic asthma development in children, should exposure to DEPs potentiate recall responses to allergen. The same authors further tested the hypothesis by assessing the effect of early-life exposure to DEP in children sensitised and non-sensitised to HDM. Their result showed a two-fold higher prevalence of asthma at seven years in sensitised children exposed to DEP at birth, compared to non-sensitised children.\(^{57}\) However, the clinical relevance of such modification in the wider population is unclear, because inconsistent results were found in other epidemiological studies.

A number of the reviewed articles demonstrated a positive association between increased pollen exposures and asthma exacerbation (see Table I).\(^{33,59,60–62}\) However, the effects persisted after controlling for several air pollutants and meteorological factors, indicative of non-modifiable effects of air pollution and seasonality. Similarly, in a Spanish study of 3 939 participants, an increase of 10 µg/m\(^3\) in SO\(_2\) and NO\(_2\) was associated with a higher risk of asthma emergency room visits (RR 5.2 95% CI: 0.5–10.1 for SO\(_2\) and RR 2.6 95% CI: 0.3–5.0 for NO\(_2\)), while the risk level remained the same with the combined effect of plant species such as Urticaceae and Poaceae (RR 5.7 95% CI: 0.9–10.6 for SO\(_2\) and RR 2.7 95% CI: 0.4–5.1 for NO\(_2\)).\(^{63}\)

In conclusion, although the synergistic effect of the combined exposure to air pollutants and allergens has been demonstrated in experimental studies, studies of this association at the population level remain inconclusive, indicating the need for more research to explore this further.

**DECLARATION OF CONFLICT OF INTEREST**

The authors declare no conflict of interest.

This article has been peer reviewed.

**REFERENCES**

### TABLE I: STUDIES OF THE ASSOCIATION BETWEEN AMBIENT POLLEN AND/OR AIR POLLUTANTS AND CHILDHOOD ASTHMA

<p>| AUTHOR(S)            | AIM                                                                 | STUDY DESIGN  | STUDY POPULATION                                                                 | EXPOSURE MEASUREMENTS                                                                                                                                                                                                                                                                                                                                                                                                                                                                 | OUTCOME MEASUREMENTS                                                                                                                                                                                                 | RESULTS                                                                                                                                                                                                                     | CONCLUSION                                                                                                                                                                                                                   |
|----------------------|----------------------------------------------------------------------|----------------|-----------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Gleason et al, 2014* | Studied transient impact of ozone, PM$<em>{2.5}$, and pollen on emergency department visit for childhood asthma | Time-stratified case-crossover design | Children aged 3–17 years residing in New Jersey, United States between 2004 and 2007 | Daily concentrations of pollen. Daily averages of PM$</em>{2.5}$ and daily eight-hour average of O$<em>3$ from fixed-monitoring station, and meteorological data from New Jersey State Climatologist                                                                                                                                                                                                                                                                                                                                                                      | Daily number of emergency visits for childhood asthma                                                                                                                                                                                                                                   | Multipollutant model showed positive associations between daily emergency department visits and same-day O$<em>3$ (RR$</em>{O_3}$ = 1.08, 95% CI 1.06–1.10), three-day tree pollen average (RR$</em>{TPA}$ = 1.19, 95% CI 1.17–1.20), three-day grass pollen average (RR$_{GP}$ = 1.08, 95% CI 1.09–1.11) respectively | Ozone, grass and weed pollen are independent risk factors for asthma exacerbation                                                                                                                                               |
| Tosca et al, 2014**  | Investigated relationship between asthma exacerbation, pollen, air pollution and meteorological factors | Time-series ecological study design | Children residing in Genoa, Italy between 2002 and 2011                           | Daily concentrations of pollen (Betulaceae, Urticaceae, Gramineae, Oleaceae), outdoor air pollutants, and meteorological variables                                                                                                                                                                                                                                                                                                                                                                                                              | Number of emergency calls for asthma exacerbation                                                                                                                                                                                                                           | Number of emergency calls associated with pollens in spring ($r = 0.498$), SO$_2$ ($r = 0.622$), NO$<em>2$ = 0.58, NO ($r = 0.699$), rainfall ($r = 0.0.318$), and wind speed ($r = 0.727$) | Environmental factors may induce exacerbation of asthma                                                                                                                                                                      |
| Yoshida et al, 2013*** | Evaluated association between pollen exposure and prevalence of allergic diseases | Ecological analysis | Children aged between six and seven years and those aged between 13 and 14 years residing in Japan | Daily pollen data on Japanese cedar and Japanese cypress                                                                                                                                                                                                                                                                                                                                                                                                                  | Allergic diseases assessed using International Study of Asthma and Allergy in childhood (ISAAC) questionnaire                                                                                                                                                                              | Cedar but not cypress pollen counts positively associated with prevalence of asthma in six- to seven-year-old children ($r = 0.49$, $p = 0.003$) but not in other age group. | Evidence of ecological associations between pollen counts and prevalence of asthma                                                                                                                                               |
| DellaValle et al, 2012* | Investigated relationship between ambient pollen concentration and severity of asthma symptoms among asthmatic children | Cross-sectional study | 430 asthmatic children aged 4–12 years in Connecticut, MA and New York             | Mixed-effect models used to estimate daily exposures to tree, grass, weed, and total pollen                                                                                                                                                                                                                                                                                                                                                          | Daily symptoms of rescue medication use, night symptoms, wheeze, persistent cough, chest tightness, shortness of breath, and night symptoms                                                                                                                                                                                      | 23% increased likelihood of any respiratory symptoms (95% CI: 1.01–1.50) and 11% increased likelihood in rescue medication use (95% CI: 1.02–1.21), comparing the highest quintile of weed pollen to the lowest, while adjusting-for C$<em>O$, PM$</em>{2.5}$, maximum temperature, antibiotic use, and season | Daily asthmatic symptoms are associated with pollen exposure                                                                                                                                                                      |
| Harley et al, 2009*   | Assessed risk of early childhood wheezing following fungal and pollen exposure in first months of life | Birth-cohort study | 514 children enrolled before birth and followed for 24 months                    | Ambient aeroallergens measured throughout study period                                                                                                                                                                                                                                                                                                                                                                                                               | Early wheezing obtained from medical records                                                                                                                                                                                                                                   | Positive association between seasonal patterns (spore season) and odds of early wheezing (adjusted OR = 3.1, 95% CI: 1.3–7.4) controlling for PM$</em>{2.5}$ and lower respiratory infections | Early wheezing in children exposed to pollen and spores in first three months of life is independent of air pollutant exposure and lower respiratory infections                                                                 |</p>
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<th>AUTHOR(S)</th>
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<tr>
<td>Heguy et al, 2008&lt;sup&gt;60&lt;/sup&gt;</td>
<td>Explored short-term effects of grass and weed pollen exposure on childhood asthma (re) admission to emergency department</td>
<td>Time-series analysis</td>
<td>Children aged 0–9 years living in Montreal, Canada between 1994 and 2004</td>
<td>Daily concentrations of pollen. Bi-hourly measurements of CO, SO&lt;sub&gt;2&lt;/sub&gt;, NO&lt;sub&gt;2&lt;/sub&gt;, and O&lt;sub&gt;3&lt;/sub&gt; from fixed-monitoring stations, and meteorological data from Meteorological Service of Canada</td>
<td>Daily number of emergency visits for childhood asthma</td>
<td>Positive association between emergency department visit and grass pollen concentration three days following exposure (mean percent change = 1.73%; 95% CI: 0.24–3.25%) after adjusting for air pollutants and meteorological variables</td>
<td>Increasing risk of emergency department visit with increasing grass pollen concentrations</td>
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<td>Endre et al, 2007&lt;sup&gt;69&lt;/sup&gt;</td>
<td>Measured prevalence of childhood asthma in relation to air pollution and total pollen count</td>
<td>Time-series ecological study design</td>
<td>Children under care of paediatricians between 1995 and 2003 in Budapest, Hungary</td>
<td>Online measurements of CO, NO&lt;sub&gt;2&lt;/sub&gt;, and SO&lt;sub&gt;2&lt;/sub&gt; at eight points in Budapest, O&lt;sub&gt;3&lt;/sub&gt; measurements at two stations, while pollen and fungal counts were calculated in Budapest</td>
<td>Doctors reported childhood asthma from response to survey questionnaire</td>
<td>Significant increase in prevalence of reported asthma from 1.88% in 1995 to 2.68% in 2003 (p &lt; 0.0001). No increase in levels of air pollutants and pollens across studied years</td>
<td>Increase of 50% in prevalence of asthma in study period, independent of air pollution and pollen exposures</td>
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<td>Atkinson et al, 2006&lt;sup&gt;61&lt;/sup&gt;</td>
<td>Investigated short-term effects of daily counts of fungal spores on asthma exacerbation</td>
<td>Time-series ecological study design</td>
<td>Children aged 0–14 years residing in London between 1992 and 1993</td>
<td>Daily concentrations of fungal spores, pollen, outdoor air pollutants, and meteorological variables</td>
<td>Daily counts of visit for childhood asthma to family doctors, emergency department, and hospital admissions</td>
<td>Changes in fungal spore concentrations from lower to upper quartiles showed increase risk of emergency department visit (RR 1.06 95% CI: 0.94–1.18) and hospital admissions (RR 1.07 95% CI: 0.97–1.19). Observed effect was independent of air pollutants</td>
<td>Exacerbation of asthma may be induced by increased fungal spore concentrations independent of other environmental factors such as air pollution</td>
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<td>Dales et al, 2009&lt;sup&gt;32&lt;/sup&gt;</td>
<td>Explored association between emergency department visit for asthma and daily concentrations of both pollen and fungal spores</td>
<td>Time-series ecological study design</td>
<td>Children reporting to children’s hospital between 1993 and 1997</td>
<td>Daily concentrations of fungal spores, pollen, outdoor air pollutants (SO&lt;sub&gt;2&lt;/sub&gt;, NO&lt;sub&gt;2&lt;/sub&gt;, O&lt;sub&gt;3&lt;/sub&gt;), and meteorological variables (barometric pressure, relative humidity, temperature)</td>
<td>Emergency department visit for asthma reporting at the children’s hospital</td>
<td>Fungal spores, but not pollen, associated with percentage increase in number of asthma visits to emergency department (8.8%, p &lt; 0.05)</td>
<td>Fungal spores implicated in exacerbation of asthma in children</td>
</tr>
<tr>
<td>Newson et al, 2009&lt;sup&gt;33&lt;/sup&gt;</td>
<td>Explored association between fungal spore counts and asthma admission</td>
<td>Time-series ecological study design</td>
<td>Children and adults residing in Trent, England between 1987 and 1994</td>
<td>Daily counts of 25 spore taxa measured in Derby</td>
<td>Hospital admission for asthma including spore counts on six days of asthma epidemics</td>
<td>No association found between total spore count and hospital admission, except for total spore count above the 90th percentile showing associations with four of the six epidemic days (OR = 9.92, 95% CI: 1.41–109.84)</td>
<td>Although no specific taxon was implicated, there is some evidence that high rates of asthma admissions tend to occur on days with high total mould spore counts</td>
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<td>Garty et al., 1998</td>
<td>Examined association between emergency department asthma visit and air pollution, weather, and airborne allergens</td>
<td>Prospective cohort study</td>
<td>Asthmatic children aged 1–18 years reporting at Children’s ER between January and December, 1993</td>
<td>Daily concentrations of airborne allergens, air pollutants, and meteorological variables</td>
<td>Number of emergency room visits for asthma</td>
<td>Positive correlation between number of emergency room visits and NO₂ and O₃, high barometric pressure and negative associations with O₃ and temperature, but no association with airborne pollens and spores</td>
<td>High levels of NOₓ and SOₓ associated with emergency room visits for asthma but not airborne pollen</td>
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<td>Rosas et al., 1998*</td>
<td>Assessed the relationship between environmental factors such as allergens, air pollution and weather and emergency department admissions for asthma</td>
<td>Time-series ecological study design</td>
<td>Children under 15 years of age residing in Mexico City, Mexico</td>
<td>Daily concentrations of airborne allergens, air pollutants, and meteorological variables</td>
<td>Number of emergency room admissions for asthma</td>
<td>Both grass and fungal spores associated with asthma admissions in both wet and dry seasons, adjusting for air pollution</td>
<td>Positive association between pollen and asthma exacerbation in children</td>
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17. D’Amato G. Environmental urban factors (air pollution and allergens) and the rising trends in allergic respiratory diseases. Allergy 2002;57 Suppl:30–33.  


