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Meteorological Mechanisms Explaining Thunderstorm-Related Asthma

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Abstract

Associations between thunderstorms and asthma morbidity have been identified in multiple locations around the world. The most prominent hypotheses for thunderstorm-related asthma are linked with bioaerosols, and involve the roles of rainwater in promoting the release of respirable allergens from pollen and fungi and downdraft winds spreading the allergens near the surface. Other hypotheses that are independent of bioaerosols involve the production of irritant gases and charged particles by lightning as well as rapid cooling of air temperatures, each of which may promote an asthmatic response. A major limitation of existing studies is the use of meteorological data with poor spatial resolution and a lack of appropriate air quality data to explicitly support and/or refute any of the specific meteorological hypotheses. Increased interdisciplinary collaboration between health scientists and geographers, climatologists, atmospheric and exposure scientists will aid in discerning the factors linking thunderstorms and asthma.

Introduction

Thunderstorm asthma or thunderstorm-related asthma are broadly defined terms used to identify the observed association between thunderstorm activity and increases in asthma exacerbation that has been noted in a variety of locations around the world (Table 1). In some studies, up to 10-fold increases in the numbers of people seeking medical attention for their asthma have been observed following thunderstorms, stressing the resources of local emergency departments and other healthcare providers. These events and the resulting impacts on public health have sparked debates among the research community as to the physical and biological mechanisms involved.

Existing studies, published almost exclusively in health science and medical journals, have been adept at identifying the epidemiological relationships between thunderstorm activity and asthma morbidity, and in proposing various hypotheses to explain the phenomenon. For example, a key hypothesis that many of these articles cite is that thunderstorms

Table 1. Thunderstorm-related asthma studies.

Article type	Date	Location	# TS days	Asthma outcome[a]	Reference
Epidemic	July 6–7, 1983	Birmingham, UK	1	ED visits to 1 hospital	Letter (Packe et al. 1983); comment (Brown and Jackson 1983); comment (Brown and Jackson 1985a)
				ED visits and HAs to 8 hospitals	Article (Packe and Ayres 1985); comment (Brown and Jackson 1985b)
				18 asthma cases versus 18 asthma controls	Letter (Packe and Ayres 1986b); comment (Brown 1986); reply (Packe and Ayres 1986a)
Epidemic	June 20, 1984	Nottingham, UK	1	ED visits to 1 hospital	Letter (Alderman et al. 1986)
Epidemic	November 11, 1984	Melbourne, Australia	1	ED visits and HAs to 1 hospital	Letter (Egan 1985)
Epidemic	November 8–9, 1987; November 29–30, 1989	Melbourne, Australia	2	ED visits and HAs to all major hospitals; 12 asthma cases vs. 16 asthma controls	Article (Bellomo et al. 1992); comment (McEvoy 1992); reply (Bellomo 1992)
Epidemic	June 24–25, 1994	Northeast London, UK	1	ED visits to 8 hospitals	Letter (Murray et al. 1994)
		Luton, UK	1	ED visits to 1 hospital; calls to 1 DSO	Letter (Higham 1994)
		Peterborough & Cambridge, UK	1	ED visits and HAs to 2 hospitals	Letter (Campbell-Hewson et al. 1994)
		Nottingham, UK	4[b]	ED visits and HAs to 1 hospital	Letter (Sutherland and Hall 1994)
		London, UK	1	ED visits and HAs to 12 hospitals	Article (Davidson et al. 1996); editorial (Bauman 1996)
		West central London, UK	1	ED visits to 1 hospital	Article (Celenza et al. 1996); editorial (Bauman 1996)
		England, Scotland, Wales	1	Calls to 29 DSOs	Article (Higham et al. 1997)
		London, UK	1	Visits to 45–47 GP practices[c]	Article (Hajat et al. 1997)
		England	1	ED visits and calls to DSOs	Article (Venables et al. 1997)
Epidemic	October 30–31, 1997	Wagga Wagga, Australia	1	183 asthma cases versus 121 asthma controls	Article (Girgis et al. 2000)

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Table 1. ***Continued.***

Article type	Date	Location	# TS days	Asthma outcome[a]	Reference
Epidemic	July 31–August 1, 2000	Calgary, Canada	1	ED and urgent care visits to 4 hospitals	Article (Wardman et al. 2002)
Epidemic	July 29, 2002	Cambridge, UK	1	26 asthma cases versus 31 asthma controls	Article (Pulimood et al. 2007); editorial (Marks and Bush 2007); comment (D'Amato et al. 2008); reply (Marks and Bush 2008)
Case–control[d]	1985–1992	Athens, Greece	74	ED visits to 1 hospital	Article (Ilias et al. 1998)
Case–control[e]	1987–1994	England	n/a	HAs to 14 RHAs	Article (Newson et al. 1998)
Time-series	1990–1994	England	81[f]	HAs to 14 RHAs	Article (Newson et al. 1997); editorial (Anto and Sunyer 1997)
Case–control[e]	1992–1995	Atlanta, USA	30	ED visits to 1 hospital	Thesis (Fedeyko and White 1997)
Time-series	1993–1996	Derbyshire, UK	40	ED visits and HAs to 2 hospitals	Article (Lewis et al. 2000)
Time-series	1990–1996	Cardiff and Newport, UK	32	HAs to 1 hospital	Article (Anderson et al. 2001)
Time-series	1993–1997	Ottawa, Canada	151	ED visits to 1 hospital	Article (Dales et al. 2003)
Case–control[e]	1995–1998	Southeastern Australia	31	ED visits to hospitals in 6 towns	Article (Marks et al. 2001)
Time-series	1992–2000	Ottawa, Canada	175	ED visits to 1 hospital	Article (Villeneuve et al. 2005)
Case–control[d]	2002	Shanghai, China	3	Mortality at 4 hospitals	Article (Deng et al. 2003)
Time-series	1993–2004	Atlanta, USA	564	ED visits to 41 hospitals	Letter (Grundstein et al. 2008)

[a]HAs, hospital admissions; EDs, emergency department visits; DSO, deputising service office for out of hours calls; RHA, regional health authority; SHA, strategic health authority; asthma cases, patients seen in the ED during an epidemic; asthma controls, patients with no emergency treatment during the epidemic.
[b]Sutherland and Hall (1994) examined data from the entire summer of 1994, including the epidemic.
[c]Hajat et al. (1997) examined data for the 1992–1994 time period, including the epidemic.
[d]Case–control type of study approach where asthma visits are compared between thunderstorm and non-thunderstorm days
[e]Case–control type of study approach where meteorological and other air quality variables are compared between asthma epidemic days and non-epidemic control days.
[f]Count of thunderstorm days based on number of high sferic density days (≥ 1 sferic/100 km^2/day); note that each of the 14 RHAs also experienced a number of low sferic density days, which are not reflected in this count.

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increase bioaerosol levels, particularly allergen-carrying respirable particles, which in turn impact asthma exacerbation. A major gap in the research, however, is in the atmospheric and exposure assessment portion of the studies where there has been little empirical work to support or refute the various hypotheses. This offers a potentially rich area of research for scientists with expertise in geography, climatology, atmospheric and exposure science. Several previous review articles have detailed the phenomenon of thunderstorm asthma (D'Amato et al. 2007; Knox 1993; Suphioglu 1998; Taylor and Jonsson 2004; Wilson and Pyatt 1996). The object of the present article is to build on the existing review literature, focusing on the current understanding of the various meteorological explanations for thunderstorm-related asthma.

A better understanding of the causes and public health implications of thunderstorm asthma is essential for directing public health policy, which may be particularly relevant in the context of climate change. A shift in climate may affect both the frequency of thunderstorms and the concentration of bioaerosols in some areas. A modeling study by Trapp et al. (2007), for instance, indicates that in response to greenhouse warming, thunderstorm activity may increase across much of the United States. In particular, certain areas in the eastern and southern United States are expected to experience increases in severe thunderstorm activity approaching 100% by the end of the twenty-first century. In addition, changes such as higher temperatures and earlier spring onset, have been associated with increased production of ragweed (*Ambrosia artemisiifolia*; Rogers et al. 2006) and greater concentrations of *Alternaria* fungal spores (Corden and Millington 2001). Further increases in bioaerosols are expected in some areas, such as the northeastern United States, in an enhanced greenhouse climate (Ziska et al. 2008).

The review will begin with a brief overview of asthma and its environmental triggers. This will be followed by a more detailed discussion of the meteorological mechanisms that may link thunderstorms with asthma morbidity.

Asthma and Environmental Triggers

Asthma is a chronic disease, characterized by inflammation, reversible airway obstruction, and increased airway responsiveness to various stimuli. Clinical expressions of this disease include wheezing, coughing, and shortness of breath. This disease places a tremendous burden on affected individuals and their families as well as the entire healthcare system. In its most recent surveillance summary for asthma, the US Centers for Disease Control report that the estimated average annual number of persons with self-reported asthma in the United States. is greater than 20 million, with over 11.5 million people reporting an asthma attack in the previous year (for the years 2001–2004) (Moorman et al. 2007).

The known causes of asthma exacerbation are multifold, including several environmental factors, such as outdoor allergens and air pollution. The largest sources of outdoor allergens are bioaerosols, including pollen grains and fungal spores. Pollen grains range in size between 10 and 100 μm and arise from trees, weeds, and grasses. Approximately 10% of the >250,000 pollen-producing species are wind-dispersed and of allergological significance (Behrendt and Beckert 2001; Solomon 2002). Fungi are organisms that grow wherever there is moisture and a source of carbohydrate. Airborne fungal spores are smaller in size than pollen grains, ranging between 2 and 50 μm in diameter, with most spores falling into the 2–10 μm range (Burge and Rogers 2000).

In a review of population-based studies examining asthma and atopy (allergic hypersensitivity), Pearce et al. (1999) determined that on average 58% of children and 54% of adults with asthma were atopic. In the United States, the National Health and Nutrition Examination Survey found the overall allergic sensitivity of the population (for the years 1976–1980) to outdoor pollen and fungal allergens to be 23.6% (Gergen and Turkeltaub 1992). Allergic sensitivity can vary by region and will depend on allergen type. For example, a study in middle-school children with bronchial hyperreactivity showed that 61% in New Mexico and 19% in Virginia were allergic to fungi (Perzanowski et al. 1998). Overall, these findings suggest that pollen and fungal allergies are not uncommon and that their prevalence is geographically diverse. Moreover, exposure to pollen grains and fungal spores can trigger atopic disease exacerbations, including allergic asthma. Numerous epidemiological studies have been conducted on asthmatic populations in various regions, observing associations between pollen and/or spore counts and clinical and subclinical symptoms, emergency department visits, or hospital admissions (Dales et al. 2000, 2004; Delfino et al. 2002; Hiltermann et al. 1997; Stieb et al. 2000; Tobias et al. 2003, 2004).

Air pollutants such as particulate matter (PM), nitrogen dioxide (NO_2), and ozone (O_3) have also been associated with acute asthma morbidity (Goldsmith and Kobzik 1999; Sarnat and Holguin 2007). These associations have been observed in a variety of locations around the world and across a variety of study designs, including time-series studies examining healthcare utilization for asthma, such as emergency department visits (Fauroux et al. 2000; Jaffe et al. 2003; Peel et al. 2005), as well as studies examining asthma symptoms and other markers of health in small cohorts of patients (Gent et al. 2003; Koenig et al. 2003; Mar et al. 2004). Air pollutants are thought to exacerbate asthma by causing inflammation in the airways (Peden 2001). In support of this hypothesis, for example, Koenig et al. (2003) found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ (particulate matter with aerodynamic diameter of less than 2.5 μm) concentrations was significantly associated with a 3.8 ppb increase in exhaled nitric oxide, a marker of pulmonary inflammation, in a study of asthmatic children in Seattle, Washington.

Asthma and Thunderstorms

Associations between thunderstorms and asthma have been identified in a number of studies. Several 'thunderstorm asthma' epidemics have been reported in the literature where single thunderstorm events have been linked to 5- to 10-fold increases in emergency department visits for asthma in locations such as the United Kingdom (Alderman et al. 1986; Packe and Ayers 1985; Pulimood et al. 2007; Venables et al. 1997), Australia (Bellomo et al. 1992; Girgis et al. 2000; Marks et al. 2001), and Canada (Wardman et al. 2002). A number of these asthma epidemics have received much attention in the literature, initiating multiple research letters, editorials, and full length research articles (Table 1). These events have important public health implications, particularly concerning the ability of healthcare systems to respond in short timeframes. For example, emergency departments during an epidemic on June 24–25, 1994 in London reported running out of medication, nebulisers, and mouthpieces for peak flow meters (Murray et al. 1994; Venables et al. 1997). This particular epidemic was observed not only at emergency departments but also among general care practitioners (Hajat et al. 1997) and on-call physicians (Higham et al. 1997). An estimated 1500–1600 additional calls were made by asthma sufferers to their general care practitioners in southeast England that night (Higham et al. 1997; Venables et al. 1997).

While early articles on thunderstorm asthma described asthma epidemics surrounding specific thunderstorm events, more recent studies have also considered the impacts on asthma morbidity from multiple thunderstorm events (Table 1). These analyses have found thunderstorms associated with non-epidemic increases in asthma in locations such as the United Kingdom (Anderson et al. 2001; Lewis et al. 2000; Newson et al. 1997), Australia (Marks et al. 2001), Canada (Dales et al. 2003; Villeneuve et al. 2005), and the United States (Fedeyko and White 1997; Grundstein et al. 2008). Dales et al. (2003), for instance, found that emergency department visits for asthma in children increased by 15% on days with thunderstorms compared to days without thunderstorms. Similarly, Grundstein et al. (2008) observed a 3% increase in emergency department visits for asthma on days following thunderstorms compared to days without thunderstorms.

Studies of asthma and thunderstorms that examine morbidity data (e.g. clinic visit, emergency department visit or hospital admissions data) for large populations generally lack details on the health status of each patient. As a result, it is difficult to decipher the types of patients that are impacted by thunderstorms. A number of studies have thus attempted to better characterize asthma patients by administering questionnaires and medical exams (e.g. skin prick tests to determine allergic sensitization to various allergens of interest) to those patients seen at a medical facility during the time of an epidemic (cases) and to other asthma patients not seen during

the epidemic (controls), with the aim of understanding why certain patients are affected by thunderstorms. Using this approach, for example, Packe and Ayres (1986b) found that the number of positive skin prick tests for various allergens did not differ between cases and controls and ruled out the involvement of fungal spores in a 1983 Birmingham epidemic. In contrast, Pulimood et al. (2007) found that thunderstorm-related asthma toward the end of the grass pollen season in a 2002 Cambridge epidemic was related to fungal spore sensitivity. Thus, these results from the UK have been somewhat inconsistent with regards to fungal spore sensitivity. However, some consistent findings have been observed in Australia, where several studies have found grass pollen allergy to be a strong predictor for severe asthma exacerbation following late spring thunderstorms (Bellomo et al. 1992; Girgis et al. 2000; Wark et al. 2002). Moreover, both studies in Australia and the United Kingdom have observed that a previous history of hay fever, and not necessarily a previous history of asthma, was characteristic of those seeking medical attention after thunderstorms (Bellomo et al. 1992; Campbell-Hewson et al. 1994; Davidson et al. 1996; Girgis et al. 2000; Venables et al. 1997; Wark et al. 2002). Baumann (1996) suggests 'there [may] be a group of atopic individuals who are sensitive to grass pollen and who react acutely to this seasonal but infrequent interaction of biological and meteorological conditions.' Indeed, through detailed analyses of patients' blood samples, Wark et al. (2002) found that thunderstorm asthma was characterized by airway inflammation consistent with allergen exposure as the cause of asthma exacerbation. Some studies have also found that individuals who were outdoors during or after the thunderstorm and/or were not taking regular medication to control their asthma were more likely to experience asthma symptoms or seek medical attention (Girgis et al. 2000; Wardman et al. 2002).

Meteorological Mechanisms of Thunderstorm-Related Asthma

While numerous studies have documented an association between thunderstorm activity and increases in asthma morbidity, a definitive explanation for the particular mechanism that links the two remains elusive. The most widely cited explanation is that thunderstorm activity leads to increases in asthma morbidity through increases in bioaerosol levels, particularly in the warm season months. Indeed, elevated levels of grass pollen (Bellomo et al. 1992; Celenza et al. 1996; Davidson et al. 1996; Lewis et al. 2000; Newson et al. 1998) and/or fungal spores (Atkinson et al. 2006; Dales et al. 2003; Packe and Ayers 1985; Pulimood et al. 2007) have been observed during periods of thunderstorm asthma. Lewis et al. (2000) emphasized the importance of bioaerosols on asthma by noting that the thunderstorm effect was reduced outside of the grass pollen season. Several meteorological-based hypotheses of thunderstorm asthma that encompass bioaerosols consider the influence of downdraft

winds and rainfall on asthma exacerbation. Meteorological mechanisms that are independent of bioaerosols have also been proposed and involve the production of irritant gases and charged particles by lightning as well as rapid cooling of air temperatures, each of which may promote an asthmatic response. The discussion below, therefore, will be divided into a section that considers meteorological mechanisms that operate in conjunction with bioaerosols and those that operate independently.

METEOROLOGICAL MECHANISMS ASSOCIATED WITH BIOAEROSOLS

Rainfall, winds, and lightning from thunderstorms have all been hypothesized to work in conjunction with bioaerosols to exacerbate asthma. The degree of bioaerosol concentrations in the atmosphere is a function of production and emission. Pollen and mold spore production are influenced by factors such as season as well as precursor meteorological conditions like rainfall and temperature (Weber 2003). Thus, the effects of thunderstorms on bioaerosol exposure and asthma incidence could be mitigated if production is limited by season or other factors. The focus of this section, however, will be on the potential impacts of thunderstorms on the emission of bioaerosols and their fracture into respirable-sized particles.

Emissions of pollen grains from the anthers of plants are largely triggered by wind speed, whereas fungal emissions are triggered by various meteorological factors depending on the fungal species, including wind, rainfall, relative humidity, rainfall, and/or temperature (Weber 2003). Laboratory experiments show that wind speeds of ≥ 2.5 m/s are required for pollen entrainment and that flower morphology may assist in the distribution of pollen (Taylor et al. 2007). Grass anthers, for instance, are suspended on long filaments that can rotate under wind speeds exceeding 3 m/s and increase pollen emission (Taylor et al. 2007). It is believed that the strong, gusty winds from thunderstorm downdrafts may be highly effective at entraining and transporting bioaerosols (Burch and Levetin 2002; Davidson et al. 1996; Marks et al. 2001; Packe and Ayers 1985; Villeneuve et al. 2005; Wardman et al. 2002). Burch and Levetin (2002) observed a rapid increase in dry air fungal spore concentrations (i.e. a spore plume) in response to strong winds triggered by a severe thunderstorm. However, over their entire 4-day study period, they found only a weak correlation between wind speed and fungal spore concentrations. The weak correlation may be explained by the use of horizontal winds rather than vertical air motions which may be more directly related to the dispersal of bioaerosols (Burch and Levetin 2002). Marks et al. (2001) explicitly examined the role of thunderstorm downdrafts in conjunction with asthma emergency department visits. The authors used abrupt changes in meteorological variables such as wind speed and temperature as indicators of downdrafts on thunderstorm days. They found a strong association between thunderstorm outflows and asthma epidemics (days

where hospital visits for asthma were greater than four standard deviations from the mean), particularly in the summer months. Thunderstorm outflows occurred on nearly half of the epidemic days compared to only 10% on control days. For one specific well-characterized event, they found a close temporal association between the arrival of the thunderstorm outflow, a rise in concentration of intact and ruptured pollen grains, and an increase in asthma emergency department visits.

Strong downdraft winds may also entrain particulate matter (Weber 2003), which can include bioaerosols as well as other particle types. Particulate matter, especially particles less than 10 μm (PM_{10}) and 2.5 μm ($PM_{2.5}$) in aerodynamic diameter, has been associated with asthma incidence and exacerbation in numerous studies (Islam et al. 2007; Ma et al. 2008; Peel et al. 2005; Sarnat and Holguin 2007). It is possible that co-deposition of these particles along with bioaerosols in the airways may lead to an increase in the immune response and have a synergistic effect on exacerbating asthma (Saxon and Diaz-Sanchez 2005; Taylor et al. 2007). Yet, investigations of thunderstorm asthma have not generally considered particles other than bioaerosols.

In addition to emission, the fragmentation of pollen grains into smaller particles (≤5 μm) is believed to be especially important for exacerbating asthma because these particles are sufficiently small to enter into the lower airways (Knox 1993; Suphioglu 1998; Taylor et al. 2007). In the presence of moisture, pollen grains may rupture by osmotic shock, releasing large quantities of respirable-sized granules (Grote et al. 2003; Knox et al. 1993; Schappi et al. 1999; Suphioglu et al. 1992; Taylor et al. 2002, 2004). While rainfall may cleanse the air of larger particles like intact pollen grains and fungal spores (McDonald 1962), the rupture of pollen grains in rainwater may lead to an increase in respirable-sized particles (Schappi et al. 1999). Suphioglu et al. (1992), for instance, observed 50-fold increases in respirable-sized particles following rainfall events. The fragmentation of pollen grains may occur not only when in contact with rain droplets but also possibly within clouds (Taylor and Jonsson 2004) and on the plant (Taylor et al. 2007). Moisture may also activate certain fungal species to release respirable-sized particles, including spores (e.g. upon exposure to rain, ascospores and basidiospores can release small spores of respirable size that become airborne via hydrostatic pressure; C. Rogers, personal communication; Weber 2003), liquid, and cytoplasmic debris (Elbert et al. 2006).

These findings may explain the results of several epidemiological studies showing that days with thunderstorms and rain were associated with increased visits to emergency departments for asthma compared to days without rain (Grundstein et al. 2008; Lewis et al. 2000; Newson et al. 1997, 1998). In a study of asthma hospital admissions and outpatient visits, Lewis et al. (2000) found positive associations between asthma visits and thunderstorms, grass pollen, and *Cladosporium* levels. Interestingly, the

authors found that the asthma-grass pollen association was dependent on rainfall levels such that asthma visits were unrelated to grass pollen counts on dry days, but were positively associated with grass pollen counts on days with light rain. Newson et al. (1997, 1998) found a similar relationship where asthma attacks increased significantly on days with high pollen counts and thunderstorms but not on days with high pollen levels and no thunderstorm activity. Both the Lewis et al. (2000) and Newson et al. (1997) studies examined relatively low numbers of thunderstorm events (40 and 61 thunderstorm days, respectively), thus possibly affecting the ability to observe differences in the thunderstorm asthma relationship by rainfall levels. Examining 564 thunderstorm days, Grundstein et al. (2008) stratified thunderstorms by rainfall amount and found associations between asthma emergency department visits and thunderstorms with recorded rainfall, but not for thunderstorms with no recorded rainfall.

Electrical activity during a thunderstorm may additionally impact pollen fragmentation and/or the ability of bioaerosols to cause a health response. Laboratory experiments show that electrical fields of similar magnitude to those found in thunderstorms enhanced pollen rupture within water and primed the pollen grains for release of respirable-sized particles (Taylor et al. 2007; Vaidyanathan et al. 2006). In addition, positive ions from the earth's surface, attracted to the negative charge at the base of the cloud, rise into the air and attach themselves to various particles including bioaerosols (Taylor and Jonsson 2004). Willoughby (1994) proposed that the resulting charge on bioaerosols may facilitate binding to cells of the immune system, for example, resulting in an increased immune response. Simulations with a lung model support this hypothesis, demonstrating that increasing the charge of respirable-sized particles promotes greater deposition in lung tissue where allergic responses can occur (O'Leary et al. 2005).

The strong association between thunderstorms and asthma may be explained by the unique characteristics of thunderstorms (e.g. updrafts and downdrafts, and electrical activity) compared with other rain producing weather systems. Taylor and Jonsson (2004) present a comprehensive model of how these characteristics may produce and distribute respirable-sized particles, and ultimately contribute to asthma exacerbation (Figure 1). The authors suggest that grass pollen grains are drawn into a cloud by the thunderstorm updraft, where they rupture in the high-humidity environment of the cloud releasing smaller granules, which are then transported to ground level through the thunderstorm downdraft. The release of pollen granules may be enhanced when rain droplets that contain pollen evaporate. In addition, electrical activity in the storms may enhance rupture of pollen grains and lead to charges on the bioaerosol fragments that increase deposition in the lungs. Pulimood et al. (2007) suggest that similar processes may explain how fungal spores (e.g. *Alternaria*) are released into the atmosphere during thunderstorms.

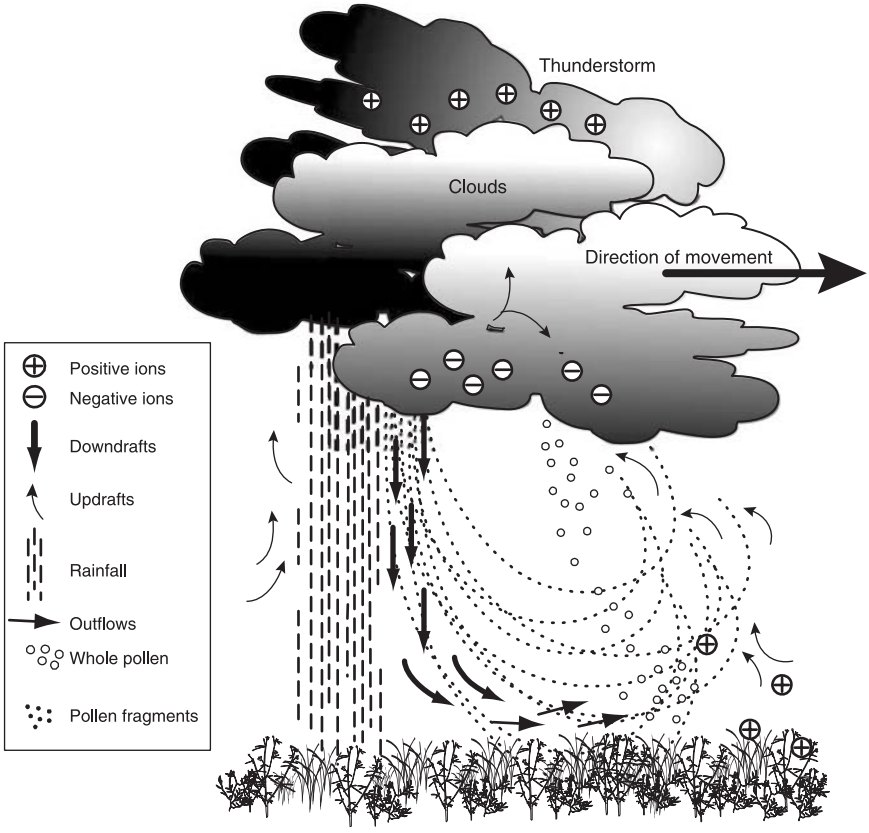

Fig. 1. A proposed mechanism of respirable allergen aerosol formation and distribution, with regard to pollen, during episodes of thunderstorm asthma, adapted from Taylor and Jonsson (2004).

Marks and Bush (2007) list four conditions that appear necessary, in combination, for an asthma epidemic to occur in conjunction with a thunderstorm: (i) high concentrations of bioaerosols (grass pollen and fungi), (ii) a thunderstorm outflow that suspends the bioaersols and concentrates them near population centers, (iii) formation of respirable-sized particles (<10 μm) through the rupture of pollen grains or otherwise associated with fungal spore emission, and (iv) exposure of individuals who are sensitive to the particular allergen and have a propensity for asthma.

While the hypothesis that thunderstorm asthma is caused by increased bioaerosol and respirable allergen levels is compelling, it is not supported by all studies (Anderson et al. 2001; Packe and Ayres 1986a,b). For example, following several detailed analyses of the July 6–7, 1983 thunderstorm asthma epidemic in Birmingham, UK, Packe and Ayres (1986a,b) determine that neither increases in pollen grains nor increases in fungal spores accounted for the extreme rise in asthma exacerbation. Furthermore, in a

time-series analysis of 32 thunderstorm events, Anderson et al. (2001) found an association between thunderstorms and emergency hospital admissions for asthma, but this association was not in conjunction with increased allergen loads. Such studies leave open the possibility that other factors linked with thunderstorms may additionally contribute to asthma morbidity.

METEOROLOGICAL MECHANISMS INDEPENDENT OF BIOAEROSOLS

Downdraft winds and lightning have been proposed as direct mechanisms by which thunderstorms exacerbate asthma. Some studies speculate that an asthmatic response may be caused by the rapid drop in temperature associated with thunderstorm downdraft winds (Packe and Ayers 1985; Weber 2003). Clinical studies of asthmatics during exercise indicate that rapid cooling of the airways may lead to airway narrowing and thus aggravate asthma (Packe and Ayers 1985; Strauss et al. 1977, 1978).

It has also been proposed that increases in photochemically generated irritant gases during thunderstorms may contribute to increased asthma cases. Lightning has been documented to increase nitrogen oxide concentrations which in turn may influence ozone production (Biazar and McNider 1995). In a study of summertime lightning across the United States, Biazar and McNider (1995) found that the production of nitrogen oxides from lighting were comparable or even exceeded those produced by anthropogenic emissions. This natural production of nitrogen oxides may therefore play an important role in influencing tropospheric ozone concentrations in some parts of the country. Indeed, a climatological examination of periods with elevated ozone levels in the southeastern United States indicated that they often occurred in conjunction with air mass thunderstorms (McNider et al. 1995).

While there is a documented relationship between exposure to photochemically generated irritant gases, like ozone, and asthma incidence and exacerbation (Fauroux et al. 2000; Gent et al. 2003; Hiltermann et al. 1999; McConnell et al. 2002; Sunyer et al. 2002), the relationship between asthma morbidity during thunderstorms and irritant gases remains unclear. Campbell-Hewson et al. (1994) found elevated levels of ozone during a 1994 thunderstorm asthma epidemic. A positive association between ozone concentrations and hospital admissions for asthma was found by Anderson et al. (2001) but the effect was found to be independent of thunderstorm activity. Studies by Celenza et al. (1996), Bellomo et al. (1992), and Packe and Ayers (1985) did not find unusually high air pollution levels during thunderstorm asthma events and thus discount the role of air pollutants as trigger mechanisms. It is possible, however, that pollution may act to enhance the immune response (i.e. act as an immunoadjuvant) to allergen exposure (Taylor and Jonsson 2004). Thus, thunderstorm asthma may be indirectly exacerbated in regions with generally high air pollution levels.

Another hypothesized mechanism that involves lightning considers the role of ionized gases. Clinical studies have documented that positive ions have a deleterious effect on bronchial function and may provoke an asthmatic response (Lipin et al. 1984; Podleski 1980). Negative ions, in contrast, have been found to protect against asthma (Ben-Dov et al. 1983; Podleski 1980). Thus, some authors (Anderson et al. 2001; Packe and Ayers 1985) have suggested that the increase of positive ions during thunderstorm events may be associated with an increased risk for asthma. However, it is unclear how long positive ions remain in the atmosphere after lightning strikes and the resulting extent of human exposure. Furthermore, Campbell-Hewson et al. (1994) argues against the role of lightning in thunderstorm asthma as they observed widely varying numbers of lightning strikes between two sights but similar hospital attendances for asthma. A more extensive investigation with a large number of thunderstorm events is needed to verify this hypothesis.

Conclusions

Despite the established body of literature linking thunderstorm activity with asthma, the specific meteorological mechanisms, particularly those that are independent of bioaerosol involvement, have yet to be fully examined. This is an area where geographers, climatologists, atmospheric and exposure scientists may make an important contribution. Ultimately closer interdisciplinary collaboration will aid in discerning the factors impacting thunderstorm asthma.

Methodologically, there are a number of limitations to existing studies. First, the existing epidemiologic research has largely been conducted in regions where thunderstorms are not frequent and, therefore, has been limited by relatively small sample sizes of thunderstorm days. Second, the meteorological portions of the studies to date have relied on single observing stations for obtaining data on meteorological parameters such as rainfall and wind speed. Because thunderstorms are small-scale phenomena, these stations may only represent the thunderstorm events that are in close proximity and may miss important meteorological influences on populated areas. Third, the lack of detailed concentration data on specific respirable-sized bioaerosols is a major technical limitation to ascribing causation (Taylor et al. 2007). Obtaining respirable allergen data, for example, is far more complicated than obtaining pollen and spore counts, requiring extensive laboratory analysis and knowledge of the specific allergen(s) of interest (Miguel et al. 2006; Taylor et al. 2007). However, these particles are likely more relevant to asthma exacerbation than whole pollen grains and spores, due to their ability to penetrate the lower airways more readily. Obtaining a better understanding of how respirable-sized bioaerosols arise in various species, their airborne nature, and resulting human exposures is a research area that deserves attention.

Improvements in the understanding of the meteorological aspect of thunderstorm asthma may be accomplished in a variety of ways. First, longer-term studies in regions known for high frequencies of thunderstorms may provide larger samples of thunderstorm days that would allow for epidemiological relationships to be characterized as well as the meteorological factors that contribute to this phenomenon. The southeastern United States, for instance, has frequent summer thunderstorms, yet the association between asthma and thunderstorm activity has not been widely examined (Fedeyko and White 1997; Grundstein et al. 2008). Furthermore, it will be particularly relevant to consider the impacts of thunderstorm-related health effects in regions anticipated to be most affected by climate change. Across the United States, for instance, thunderstorm activity is forecast to increase in cities such as Atlanta, Dallas, and New York due to anthropogenically forced climate change (Trapp et al. 2007). In particular, Trapp et al. (2007) indicates that in Atlanta, there may be a 100% increase in days suitable for severe thunderstorms by the end of the twenty-first century. Second, it is recommended that meteorological data with greater spatial resolution should be utilized in these studies to better capture the spatial characteristics of thunderstorms. For instance, lightning detection networks and radar data may provide useful means for identifying characteristics of thunderstorm activity and intensity over space.

Although thunderstorm asthma has been identified in a variety of locations around the world, the recognition of its health implications differs by country. In the United States, for instance, it is a rather obscure phenomenon where few people are aware of the links between asthma and thunderstorm activity. In contrast, there has been a greater appreciation of the health implications of thunderstorm asthma in areas like the UK where high profile epidemics have occurred (Table 1). Indeed, the UK Meteorological Office has been working with asthma and pollen experts to help predict thunderstorm asthma events, so that they can more effectively warn hospitals and medical professionals (UK Met Office 2006). In the absence of a better understanding of exactly how thunderstorms contribute to asthma, however, Newson et al. (1998) concede that an early warning system may lead to an unacceptably high rate of false alarms. Thus, furthering our understanding of the meteorological components of this phenomenon will enable better prediction of relevant weather events, leading to enhanced prevention abilities and ultimate improvements in public health. The ability to do so may be especially critical in the context of climate change and the predicted increased incidence of thunderstorms in some areas.

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References

Alderman, P. M., Sloan, J. P., and Basran, G. S. (1986). Asthma and thunderstorms. *Archives of Emergency Medicine* 3, pp. 260–262.

Anderson, W., et al. (2001). Asthma admissions and thunderstorms: a study of pollen, fungal spores, rainfall, and ozone. *Quarterly Journal of Medicine* 94, pp. 429–433.

Anto, J. M., and Sunyer, J. (1997). Thunderstorms: a risk factor for asthma attacks. *Thorax* 52, pp. 669–670.

Atkinson, R. W., et al. (2006). Temporal associations between daily counts of fungal spores and asthma exacerbations. *Occupational and Environmental Medicine* 63, pp. 580–590.

Bauman, A. (1996). Asthma associated with thunderstorms: grass pollen and the fall in temperature seem to be to blame. *British Medical Journal* 312, pp. 590–591.

Behrendt, H., and Beckert, W. M. (2001). Localization, release and bioavailability of pollen allergens: the influence of environmental factors. *Current Opinion in Immunology* 13, pp. 709–715.

Bellomo, R. (1992). Thunderstorm associated epidemics of asthma – reply. *Medical Journal of Australia* 157, pp. 352–353.

Bellomo, R., et al. (1992). Two consecutive thunderstorm associated epidemics of asthma in the city of Melbourne: the possible role of rye grass pollen. *Medical Journal of Australia* 156, pp. 834–837

Ben-Dov, I., et al. (1983). Effect of negative ionisation of inspired air on the response of asthmatic children to exercise and inhaled histamine. *Thorax* 38, pp. 584–588

Biazar, A. P., and McNider, R. T. (1995). Regional estimates of lightning production of nitrogen oxides. *Journal of Geophysical Research* 100 (D11), pp. 22861–22874

Brown, H. M. (1986). Skin sensitivity to aeroallergens. *Lancet* 1, pp. 980–980.

Brown, H. M., and Jackson, F. A (1983). Asthma and the weather. *Lancet* 2, pp. 630–630.

——. (1985a). Didymella, asthma, and the weather. *Lancet* 1, pp. 1326–1327.

——. (1985b). Asthma outbreak during a thunderstorm. *Lancet* 2, pp. 562–562.

Burch, M., and Levetin, E. (2002). Effects of meteorological conditions on spore plumes. *International Journal of Biometeorology* 46, pp. 107–117.

Burge, H. A., and Rogers, C. A. (2000). Outdoor allergens. *Environmental Health Perspectives* 108 (Suppl. 4), pp. 653–659.

Campbell-Hewson, G., et al. (1994). Epidemic of asthma possibly associated with electrical storms. *British Medical Journal* 309, pp. 1087–1087.

Celenza, A., et al. (1996). Thunderstorms associated asthma: a detailed analysis of environmental factors. *British Medical Journal* 312, pp. 604–607.

Corden, J. M., and Millington, W. M. (2001). The long term trends and seasonal variation in the aeroallergen Alternaria in Drby, UK. *Aerobiologia* 17, pp. 127–136.

D'Amato, G., Cecchi, L., and Liccardi, G. (2008). Thunderstorm-related asthma: not only grass pollen and spores. *Journal of Allergy and Clinical Immunology* 121, pp. 537–538.

D'Amato, G., Liccardi, G., and Frenguelli, G. (2007). Thunderstorm-asthma and pollen allergy. *Allergy* 62, pp. 11–16.

Dales, R. E., et al. (2000). Influence of ambient fungal spores on emergency visits for asthma to a regional children's hospital. *American Journal of Respiratory and Critical Care Medicine* 162, pp. 2087–2090.

——. (2003). The role of fungal spores in thunderstorm asthma *Chest* 123 (3), pp. 745–750.
——. (2004). Influence of outdoor aeroallergens on hospitalization for asthma in Canada. *Journal of Allergy and Clinical Immunology* 113, pp. 303–306.
Davidson, A. C., et al. (1996). A major outbreak of asthma associated with a thunderstorm: experience of accident and emergency departments and patients' characteristics. *British Medical Journal* 312, pp. 601–604.
Delfino, R. J., et al. (2002). Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. *Environmental Health Perspectives* 110, pp. 607–617.
Deng, Q. F., et al. (2003). A preliminary study on the relation between thunderstorms and mortality of in-patients. *Journal of Public Health Medicine* 25(3), pp. 246–248.
Egan, P. (1985). Weather or not. *Medical Journal of Australia* 142, pp. 330–330.
Elbert, W., et al. (2006). Contribution of fungi to primary aerosols in the atmosphere: active discharge of spores, carbohydrates, and inorganic ions by Asco- and Basidiomycota. *Atmospheric Chemistry and Physics Discussions* 6, pp. 11317–11355.
Fauroux, B., et al. (2000). Ozone: a trigger for hospital pediatric asthma emergency room visits. *Pediatric Pulmonology* 30, pp. 41–46.
Fedeyko, H. J., and White, M. C. (1997). *Thunderstorms and pollen as risk factors for elevated daily counts of emergency room visits for pediatric asthma in Atlanta, Georgia [MPH thesis]*. Atlanta, GA: Emory University.
Gent, J. F., et al. (2003). Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *Journal of the American Medical Association* 290, pp. 1859–1867.
Gergen, P. J., and Turkeltaub, P. C. (1992). The association of individual allergen reactivity with respiratory disease in a national sample: data from the second National Health and Nutrition Examination Survey, 1976–80 (NHANES II). *Journal of Allergy and Clinical Immunology* 90, pp. 579–588.
Girgis, S. T., et al. (2000). Thunderstorm-associated asthma in an inland town in south-eastern Australia. Who is at risk? *European Respiratory Journal* 16, pp. 3–8.
Goldsmith, C. A., and Kobzik, L. (1999). Particulate air pollution and asthma: a review of epidemiological and biological studies. *Reviews on Environmental Health* 14, pp. 121–134.
Grote, M., Valenta, R., and Reichelt, R. (2003). Abortive pollen germination: a mechanism of allergen release in birch, alder, and hazel revealed by immunogold electron microscopy. *Journal of Allergy and Clinical Immunology* 111 (5), pp. 1017–1023
Grundstein, A., et al. (2008). Thunderstorm-associated asthma in Atlanta, Georgia. *Thorax* 63, pp. 659–660.
Hajat, S., Goubet, S. A., and Haines, A. (1997). Thunderstorm-associated asthma: the effect on GP consultations. *British Journal of General Practice* 47, pp. 639–641.
Higham, J. H. (1994). Asthma trends – thunderstorm peak in Luton. *British Medical Journal* 309, pp. 604–604.
Higham, J. H., et al. (1997). Asthma and thunderstorms: description of an epidemic in general practice in Britain using data from a doctors' deputizing service in the UK. *Journal of Epidemiology and Community Health* 51, pp. 233–238.
Hiltermann, T. J. N., et al. (1997). Effects of photochemical air pollution and allergen exposure on upper respiratory tract inflammation in asthmatics. *American Journal of Respiratory and Critical Care Medicine* 156, pp. 1765–1772.
——. (1999). Ozone-induced inflammation assessed in sputum and bronchial, lavage fluid from asthmatics: a new noninvasive tool in epidemiologic studies on air pollution and asthma. *Free Radical Biology and Medicine* 27, pp. 1448–1454.
Ilias, I., Ilias, D., and Filippou, N. (1998). Eight year retrospective study of thunderstorm-associated asthma attacks in Athens, Greece. *International Journal of Environmental Health Research* 8, pp. 335–337.
Islam, T., et al. (2007). Relationship between air pollution, lung function and asthma in adolescents. *Thorax* 62, pp. 957–963.
Jaffe, D. H., Singer, M. E., and Rimm, A. A. (2003). Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991–1996. *Environmental Research* 91, pp. 21–28.

Knox, R. B. (1993). Grass pollen, thunderstorms and asthma. *Clinical Experimental Allergy* 23, pp. 354–359.

Koenig, J. Q., et al. (2003). Measurement of offline exhaled nitric oxide in a study of community exposure to air pollution. *Environmental Health Perspectives* 111, pp. 1625–1629.

Lewis, S. A., et al. (2000). Combined effects of aerobiological pollutants, chemical pollutants and meteorological conditions on asthma admissions and A & E attendances in Derbyshire UK, 1993–1996. *Clinical and Experimental Allergy* 30, pp. 1724–1732.

Lipin, I., et al. (1984). Effect of positive ionisation of inspired air of asthmatic children to exercise. *Thorax* 39, pp. 594–596.

Ma, L., et al. (2008). Effects of airborne particulate matter on respiratory morbidity in asthmatic children. *Journal of Epidemiology* 18, pp. 97–110.

Mar, T. F., et al. (2004). An analysis of the association between respiratory symptoms in subjects with asthma and daily air pollution in Spokane, Washington. *Inhalation Toxicology* 16, pp. 809–815.

Marks, G. B., and Bush, R. K. (2007). It's blowing in the wind: new insights into thunderstorm-related asthma. *Journal of Allergy and Clinical Immunology* 120, pp. 530–532.

——. (2008). Thunderstorm-related asthma: not only grass pollen and spores – reply. *Journal of Allergy and Clinical Immunology* 121, pp. 538–538.

Marks, G. B., et al. (2001). Thunderstorm outflows preceding epidemics of asthma during spring and summer. *Thorax* 56, pp. 468–471.

McConnell, R, et al. (2002). Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359, pp. 386–391.

McDonald, J. E. (1962). Collection and washout of airborne pollens and spores by raindrops. *Science* 135, pp. 435–437.

McEvoy, R. J. (1992). Thunderstorm associated epidemics of asthma. *Medical Journal of Australia* 157, pp. 352–352

McNider, R. T., Norris, W. B., and Song, J. A. (1995). *Regional meteorological characteristics during ozone episodes in the southeastern United States transactions, regional photochemical measurement and modeling studies*. Air and Waste Management Association International Specialty Conference, San Diego, CA, November 8–12, 1993.

Miguel, A. G., et al. (2006). Meteorological influences on respirable fragment release from Chinese elm pollen. *Aerosol Science and Technology* 40, pp. 690–696.

Moorman, J. E., et al. (2007). National surveillance for asthma – United States, 1980–2004. *Morbidity and Mortality Weekly Report* 56, pp. 1–14, 18–54.

Murray, V., et al. (1994). Epidemic of asthma possibly related to thunderstorms. *British Medical Journal* 309 (6947), pp. 131–132.

Newson, R., et al. (1997). Effect of thunderstorms and airborne grass pollen on the incidence of acute asthma in England, 1990–1994. *Thorax* 52, pp. 669–670.

——. (1998). Acute asthma epidemics, weather and pollen in England, 1987–1994. *European Respiratory Journal* 11 (3), pp. 694–701.

O'Leary, M., Koolpiruck, D., and Balachandran, W. (2005). The role of electrostatic charge accumulated by respirable sized allergens with regard to thunderstorm asthma Industry Applications Conference, Fourtieth IAS Annual Meeting. *Conference Record* 2, pp. 779–783.

Packe, G. E., and Ayers, J. G. (1985). Asthma outbreak during a thunderstorm. *Lancet* 2, pp. 199–204.

——. (1986a). Aerollargens in an asthma outbreak. *Lancet* 1, pp. 1333–1334.

——. (1986b). Aeroallergen skin sensitivity in patients with severe asthma during a thunderstorm. *Lancet* 1, pp. 850–851.

Packe, G. E., Archer, P. S., and Ayres, J. G. (1983). Asthma and the weather. *Lancet* 2, pp. 281–281

Pearce, N., Pekkanen, J., and Beasley, R. (1999). How much asthma is really attributable to atopy? *Thorax* 54, pp. 268–272.

Peden, D. B. (2001). Air pollution in asthma: effect of pollutants on airway inflammation. *Annals of Allergy, Asthma & Immunology* 87, pp. 12–17.

Peel, J. L., et al. (2005). Ambient air pollution and respiratory emergency department visits. *Epidemiology* 16, pp. 164–174.

Perzanowski, M. S., et al. (1998). Association of sensitization to Alternaria allergens with asthma among school-age children. *Journal of Allergy and Clinical Immunology* 101, pp. 626–632.

Podleski, W. K. (1980). Asthma and ionisation. *Lancet* 8 November, p. 1035.

Pulimood, T. B., et al. (2007). Epidemic asthma and the role of the fungal mold *Alternaria alternata*. *Journal of Allergy and Clinical Immunology* 12, pp. 10–617.

Rogers, C. A., et al. (2006). Interaction of the onset of spring and elevated atmospheric CO_2 on ragweed (*Ambrosia artemisiifolia* L.) pollen production. *Environmental Health Perspectives* 114 (6), pp. 865–869; doi: 10.1289/ehp.8549.

Sarnat, J. A., and Holguin, F. (2007). Asthma and air quality. *Current Opinion in Pulmonary Medicine* 13, pp. 63–66.

Saxon, A., and Diaz-Sanchez, D. (2005). Air pollution and allergy: you are what you breathe. *Nature Immunology* 6, p. 223.

Schappi, G. F., et al. (1999). Concentrations of major grass group 5 allergens in pollen grains and astmospheric particles: implications for hay fever and allergic asthma sufrferers sensitized to grass pollen allergens. *Clinical and Experimental Allergy* 29, pp. 633–641.

Solomon, WR (2002). Airborne pollen: a brief life. *Journal of Allergy and Clinical Immunology* 109, pp. 895–900.

Stieb, D. M., et al. (2000). Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. *Journal of Exposure Analysis and Environmental Epidemiology* 10, pp. 461–477.

Strauss, R. H., et al. (1977). Enhancement of exercise-induced asthma by cold air. *New England Journal Medicine* 297, pp. 743–747.

——. (1978). Influence of heat and humidity on the airway obstruction induced by exercise in asthma. *Journal of Clinical Investigation* 61, pp. 433–440.

Sunyer, J., et al. (2002). Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthma. *Thorax* 57, pp. 687–693.

Suphioglu, C. (1998). Thunderstorm asthma due to grass pollen. *International Archives of Allergy and Immunology* 116, pp. 253–260.

Suphioglu, C., et al. (1992). Mechanism of grass-pollen-induced asthma. *Lancet* 339, pp. 569–572.

Sutherland, A., and Hall, I. P. (1994). Thunderstorms and asthma admissions. *Lancet* 344, pp. 1503–1504.

Taylor, P. E., and Jonsson, H. (2004). Thunderstorm asthma. *Current Allergy and Asthma Reports* 4, pp. 409–413.

Taylor, P. E., et al. (2004). Birch pollen rupture and the release of aerosols of respirable allergens. *Clinical and Experimental Allergy* 34 (10), pp. 1591–1596.

——. (2002). Release of allergens as respirable aerosols: a link between grass pollen and asthma. *Journal of Allergy and Clinical Immunology* 109 (1), pp. 51–56.

——. (2007). Links between pollen, atopy and the asthma epidemic. *International Archives of Allergy and Immunology* 144, pp. 162–170.

Tobias, A., et al. (2003). Short term effects of airborne pollen concentrations on asthma epidemic. *Thorax* 58, pp. 708–710.

Tobias, A., Galan, I., and Banegas, J. R. (2004). Non-linear short-term effects of airborne pollen levels with allergenic capacity on asthma emergency room admissions in Madrid, Spain. *Clinical and Experimental Allergy* 34, pp. 871–878.

Trapp, R. J., et al. (2007). Changes in severe thunderstorm environment frequency during the 21st century caused by anthropogenically enhanced global radiative forcing. *Proceedings of the National Academy of Sciences* 104 (50), pp. 19719–19723.

UK Met Office (2006). Health factsheet – thunderstorm-related asthma. *UK Met Office* p. 2.

Vaidyanathan, V., et al. (2006). Effects of electric fields on pollen rupture. *Journal of Allergy and Clinical Immunology* 117 (2), pp. S157.

Venables, K. M., et al. (1997). Thunderstorm-related asthma – the epidemic of 24/25 June 1994. *Clinical and Experimental Allergy* 27, pp. 725–736.

Villeneuve, P. J., Leech, J., and Bourque, D. (2005). Frequency of emergency room visits for childhood asthma in Ottawa, Canada: the role of weather. *International Journal of Biometeorology* 50, pp. 48–56.

Wardman, A. E. D., Stefani, D., and MacDonald, J. C. (2002). Thunderstorm-associated asthma or shortness of breath epidemic: a Canadian case report. *Journal of the Canadian Thoracic Society* 9 (3), pp. 267–270.

Wark, P. A. B., et al. (2002). Airway inflammation in thunderstorm asthma. *Clinical and Experimental Allergy* 32, pp. 1750–1756.

Weber, R. W. (2003). Meteorologic variables in aerobiology. *Immunology and Allergy Clinics of North America* 23, pp. 411–422.

Willoughby, D. A. (1994). Asthma increase, Summer 1994. *Lancet* 344, p. 413.

Wilson, J., and Pyatt, B. (1996). Thunderstorms and asthma – a review of current research progress. *Journal of Meteorology* 21, pp. 121–125.

Ziska, L. H., Epstein, P. R., and Rogers, C. A. (2008). Climate change, aerobiology, and public health in the northeast United States. *Mitigation and Adaptation Strategies for Global Change* 13 (5–6), pp. 607–613; doi: 10.1007/s11027-007-9134-1.