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EUROPEAN RESPIRATORY UPDATE

A trans-disciplinary overview of case reports of thunderstorm-related asthma outbreaks and relapse

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There is evidence that, during the pollen season, thunderstorms can be associated with allergic asthma outbreaks in patients suffering from pollen allergy [1], and there are observations in favour of the possibility that thunderstorms disturb ground-level pollen grains, which may release allergenic particles of respirable size into the atmosphere after rupture by osmotic shock [2, 3].

During the first 20–30 min of a thunderstorm, patients suffering from pollen allergy may inhale a high concentration of the allergenic material that is dispersed into the atmosphere, which in turn can induce (severe) asthmatic reactions in some cases [3–6]. Even though thunderstorms can induce severe asthma attacks or exacerbations, they are neither frequent nor responsible for a high amount of disease exacerbation. However, physicians and pollen allergy patients should know the mechanisms involved in the release of allergens from airborne pollen grains during thunderstorms and the associated risk in view of prevention. Information about the risk of an asthma attack is also relevant in subjects affected only by seasonal allergic rhinitis who can inhale lower airways pollen aerosol. In addition, there is a potential risk of thunderstorm-related relapse of asthma attacks in some patients. This constitutes a major concern nowadays as the possibility of thunderstorm-associated asthma outbreaks have become of dramatic actuality due to the ''highly likely'' increase in frequency of heavy precipitation events, including thunderstorms, projected by the climate change scenarios for the future decades [7].

The purpose of this article is to gather existing trans-disciplinary data on thunderstorm-related asthma attacks and potential relapse in the same patient.

ALLERGENIC POLLEN IN THE ATMOSPHERE AND POLLEN ALLERGY

Pollen grain, which is a causative agent of allergic respiratory responses, is among the commonest allergens and pollen allergy due to its elevated prevalence and associated costs is now a public health problem [8]. Worldwide, up to 40% of the general population are reported to suffer from hay fever [8, 9]. In the European Union countries, between 8% and 35% of young adults show immunoglobulin (Ig)E serum antibodies to the most commonly encountered grass pollen allergens [10] and the cost of pollen allergy in terms of impaired work fitness, sick leave, consulting physicians and drugs is very high [11].

The concentration of allergenic pollen influences the degree of symptoms, but the relationship between allergen exposure, inflammation of airways and clinical symptoms is complex, and factors other than allergens are involved [12]. Pollen grains penetrate into the upper respiratory tract but, because of their size, which is always >10 μm diameter, they rarely reach the bronchial regions. However, bronchial asthma and its equivalents, such as irritative cough, are not infrequent in people affected by pollen-induced allergy [8].

During natural pollination, mature pollen grains are dehydrated when they are released by anthers at the dispersal time. Once the pollen grains come into contact with a wet surface they absorb water undergoing rapid metabolic changes together with ultrastructural modifications. The pollinic allergens that are located in the pollen walls and/or in the cytoplasm are then rapidly released. This happens when the pollen grains come into contact with the oral, nasal or conjunctival mucosa that are humid, thereby inducing the appearance of pollinosis symptoms in sensitised patients [13]. Cytoplasmic allergens are also released into the atmosphere when the pollen bursts under osmotic shock and can create a respirable allergenic aerosol.

THUNDERSTORMS AND ALLERGIC ASTHMA EPIDEMICS IN POLLEN ALLERGY SUBJECTS DURING POLLEN SEASON

A PubMed search with MeSH terms ''THUNDERSTORM'' and ''ASTHMA'' resulted in 38 papers, 11 of which reported on cases of thunderstorm-related asthma that were considered to be of quality and included in the overview. The studies evaluated outbreaks of asthma during thunderstorms (table 1) and allowed the identification of associated characteristics (table 2).

Thunderstorm-related asthma outbreaks have been described in various geographical zones. One of the first observations regarding thunderstorms and asthma outbreaks was provided by PACKE and AYRES [3] at the East Birmingham Hospital

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TABLE 1 Thunderstorm asthma studies

Country	Study year	Event	Comments
UK [3]	1983	26 sudden cases of asthma attacks	Increased risk of asthma in relation to thunderstorms
Australia [14]	1992	Hospital attendances and admissions because of asthma exacerbation	Late spring thunderstorms in Melbourne can trigger epidemics of asthma attacks (five to 10-fold rise) The seasonal nature of the phenomenon and the pattern of allergic responses found in affected patients suggests a possible aetiological role for rye grass pollen
UK [15]	1997	Asthma or other airways disease hospital visits	640 cases who attended during a 30-h period from 18:00 h on June 24, 1994, nearly 10 times the expected number
Canada [16]	1992–2000	18970 hospital ED asthma visits among children 2–15 yrs of age	Summer thunderstorm activity was associated with an OR of 1.35 (95% CI 1.02–1.77) relative to summer periods with no activity
USA [17]	1993–2004	215832 asthma ED visits; 24350 of these visits occurred on days following thunderstorms	Significant association between daily counts of asthma ED visits and thunderstorm occurrence Asthma visits were 3% higher on days following thunderstorms
Australia [18]	2000	Asthma visits during thunderstorms	History of hay fever and allergy to rye grass are strong predictors for asthma exacerbation during thunderstorms in spring
Australia [19]	2001	Hospital attendances for asthma	The incidence of excess hospital attendances for asthma during late spring and summer was strongly linked to the occurrence of thunderstorm outflows
UK [20]	2002	A case–control study of 26 patients presenting to Cambridge University Hospital with asthma after the thunderstorm	*Alternaria alternata* sensitivity is a compelling predictor of epidemic asthma in patients with seasonal asthma and grass pollen allergy and is likely to be the important factor in thunderstorm-related asthma
Italy [21]	2004	ED asthma	Six cases of thunderstorm-related asthma because of pollen (*Paretaria*)
Italy [22]	2010	ED asthma	20 cases of thunderstorm-related asthma because of pollen (olive tree)
Australia [23]	2010	Epidemics of ''thunderstorm asthma'' that occurred in Melbourne during spring 2010	The approach of spring, together with high winter rainfall in and around Melbourne that heralds another severe pollen season, raises the risk of allergic rhinitis and asthma in pollen-sensitive individuals

ED: emergency department; OR: odds ratio.

(Birmingham, UK) on July 6 and 7, 1983. These authors described a remarkable increase in the number of asthma emergency department admissions during the hours of a thunderstorm. In a 36-h period, 26 asthma cases were treated in the emergency department, compared with a daily average of two or three cases in the days preceding the outbreak. Another asthma outbreak occurred in London, UK, coinciding with a heavy thunderstorm on June 24, 1994, when a large increase in the number of visits for asthma at the emergency departments of London and the southwest of England was observed. Several patients who were examined, who were not known to be asthmatics or were affected only by seasonal rhinitis, experienced an asthma attack [15]. During a 30-h period from 18:00 h on June 24, 1994, 640 patients with asthma or other airways disease (283 of whom were not known to be asthmatic and 403 were affected only by seasonal rhinitis) attended several emergency departments, nearly 10 times the expected number of 66 patients. In total, 104 patients were admitted (including five to an intensive care unit) (574 patients whose asthma was attributable to the thunderstorm). Not all affected patients attended a hospital and this epidemic was the largest outbreak ever recorded.

Other asthma outbreaks during thunderstorms have been described in Australia. In Melbourne, two large asthma outbreaks (rapid increase in hospital or general practitioner visits for asthma) coincided with thunderstorms [14]. In Wagga Wagga, 215 asthmatic subjects attended the local emergency department, 41 of whom required admission to hospital [18]. In south eastern Australia, MARKS *et al*. [19] observed that the incidence of excess hospital attendances for asthma during late spring and summer was strongly linked to the occurrence of thunderstorm outflows and demonstrated that the arrival of a thunderstorm outflow was accompanied by a large increase in the concentration of ruptured pollen grains in ambient air.

Thunderstorm-related asthma was observed in Naples, Italy, on June 3 and 4, 2004, when six adults and one child received treatment in emergency departments. One patient was admitted to an intensive care unit for very severe bronchial obstruction and acute respiratory insufficiency following a sudden thunderstorm. All individuals were outdoors when the thunderstorm struck. In one severe case, a female sensitised only to *Parietaria* pollen allergen, soon began to show symptoms of intense dyspnoea, which gradually worsened. She was taken to hospital where she was intubated and given high intravenous doses of corticosteroids. She was discharged a few days later. This patient had previously suffered from seasonal asthma but had been asthma-free for the past few years and did not need continuous therapy. None of the other six subjects regularly took anti-allergic and/or anti-asthma drugs. All seven patients were sensitised with allergic

TABLE 2 Characteristics of documented epidemics of thunderstorm-associated asthma

There is a link between asthma epidemics and thunderstorms
The epidemics related to thunderstorms are limited to seasons when there are high atmospheric concentrations of airborne allergenic pollens
There is a close temporal association between the start of the thunderstorm and the onset of epidemics
There are not high levels of gaseous and particulate components of air pollution during thunderstorm-related asthma outbreaks
Subjects with pollen allergy who stay indoors with the window closed during thunderstorms are not involved
There is a major risk for subjects who are not receiving anti-asthma treatment but subjects with allergic rhinitis and without previous asthma can experience severe bronchoconstriction
There are no observations on the involvement of asthma in nonallergic subjects

respiratory symptoms upon exposure to *Parietaria* pollen (fig. 1) but were not sensitised to grasses [21]. *Parietaria* is an Urticacea that is widespread in the Naples area of Italy with a spring and summer pollen season that is, in part, coexistent with that of grasses. During the thunderstorm, the concentration of airborne *Parietaria* pollen grains was particularly high, with a peak of 144 grains·m^{-3} being recorded on June 3, 2004 [24]. Air pollution levels for both gaseous and particulate components based on the hourly concentrations of nitric dioxide, ozone and respirable particulate matter were not particularly high in Naples on June 3 and 4, 2004. Subjects with sensitisation to *Parietaria* who were indoors in Naples with the windows closed during the night between June 3 and 4, 2004, did not experience asthma attacks. No moulds or viruses were involved in the Naples epidemics. Recently, LOSAPPIO *et al*. [22] observed 20 patients with allergic sensitisation to *Olea* pollen who presented to an emergency department in Barletta, Italy, for sudden and severe asthmatic symptoms in May 2010 following a thunderstorm. A similar phenomenon has been suggested for moulds [25] after the observation of a possible key role of sensitisation to *Alternaria* species in thunderstorm-related asthma [21]. To date, among pollens, only grass, *Parietaria* and olive pollen have been suggested as possible key factors in thunderstorm-related asthma [26]. Of note, most of the aforementioned asthma epidemics were observed while levels of chemical air pollution were below or similar to levels in a control period. Although the value of these studies can be criticised because of the small sample size, two studies based on larger samples sizes in two North American countries support the existence of thunderstorm-related asthma cases. In Ottawa, Canada, hospital emergency department visits for asthma between 1992 and 2000 were identified and their relationships to fog, thunderstorms, snow, and liquid and freezing forms of precipitation were investigated. In total, there were 18,970 asthma visits among children aged 2–15 yrs and their occurrence was significantly associated with summer thunderstorm activity relative to summer periods with no activity (OR 1.35, 95% CI 1.02–1.77) [16]. In Atlanta, GA, USA, 24,350 emergency department asthma visits occurred on days following thunderstorms (out of 215,832 emergency department asthma visits during the study period) [17]. A statistically significant association was observed between daily counts of

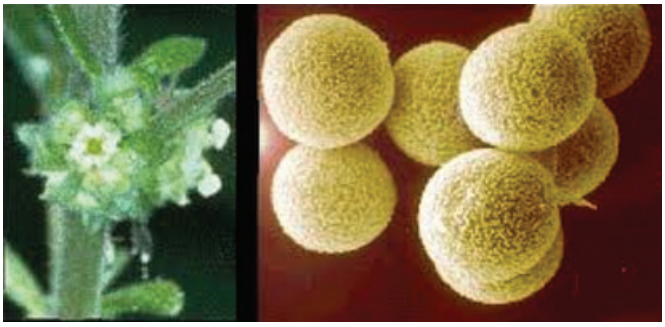

FIGURE 1. *Parietaria* pollen.

asthma emergency department visits and thunderstorm occurrence ($p<0.001$). Overall, the number of emergency department asthma visits was 3% higher on days following thunderstorms. Rainfall and gusts of wind played a role, with higher risks of asthma associated with rainfall and strong gusts.

Possible mechanisms for thunderstorm-related asthma

Although much remains to be discovered about the relationship between an increase in the number of asthma attacks and thunderstorms, reasonable evidence exists in favour of a causal link between them in patients suffering from pollen allergy. Although rainfall is usually known to remove pollen from the air, that is not always the case. During a thunderstorm, dry updrafts entrain whole pollens into the high humidity at the cloud base where pollens may rupture and cold downdrafts carry pollen fragments (pollen grains are too large to penetrate the deeper airways) to ground level where outflows distribute them. As a consequence, there is a high respirable allergen load in the air. TAYLOR *et al*. [27] hypothesised that the turbulent front of the advancing outflow releases more pollen from flowering grasses. Due to strong electric fields that develop during thunderstorms, positive ions are released from the ground and could attach to particles and/or electric charge may enhance pollen rupture, thus, enhancing bronchial hyperresponsiveness. Moreover, grass pollen may release large amounts of paucimicronic allergenic particles, *i.e.* cytoplasmatic starch granules containing grass allergens (allergen-bearing starch granules), after rupture by osmotic shock during thunderstorms. In 1998, SUPHIOGLU *et al*. [28] showed that rye grass pollen grains contain a large amount of starch granules coated with allergens. After being ruptured in rainwater by osmotic shock, each grain can release ~700 starch granules, which are small enough to penetrate the airways and trigger asthma attacks in previously sensitised subjects. Allergen-bearing starch granules obtained upon contact of pollen with water have been shown to create an inhalable allergenic aerosol capable of triggering an early asthmatic response in an experimental *in vitro* study. Starch granules were shown to be recognised by pollen sensitised in rat serum and to trigger lymph node cell proliferation in these rats [29]. These data provide new arguments supporting the implication of grass pollen starch granules in thunderstorm-related asthma. Other pollens, such as those of *Parietaria*, which do have not starch granules in the cytoplasm, can release other paucimicronic cytoplasmic component carrying allergens, as recently confirmed by JATO *et al*. [30]. Due to their very small size (<5 μm), these microparticles can penetrate the lower airways inducing the occurrence of bronchial

allergic symptoms. In rats, it has been shown that pollen cytoplasmic granules (PCGs) containing allergens are able to deeply penetrate the respiratory tract and induce local and strong allergic and inflammatory responses more closely linked with asthma- than rhinitis-related allergic symptoms [31]. The proliferative responses of lymph node cells were similar in PCG- and pollen-sensitised rats. IgE and IgG_1 levels were higher in pollen- than PCG-sensitised rats. However, eosinophils, lymphocytes and pro-allergy cytokines in bronchoalveolar lavage fluid were higher in PCG- than pollen-sensitised rats. Overall, significantly increased amounts of pollen and mould spore counts have been found during thunderstorms, although published data are scant [19, 21, 24]. However, to date, no experimental study reproducing thunderstorm conditions has been conducted to test the thunderstorm hypothesis in more detail. It is likely that there is no role for cold or thunderbolts as only patients with allergic sensitisation to pollen and not patients suffering from intrinsic asthma have been involved. In the case of fungal spores, such as *Alternaria*, the prerequisites for asthma epidemics associated with thunderstorms have been described as follows [20, 25]: 1) a sensitised, atopic, asthmatic individual; 2) a sudden, large allergen exposure; 3) a large-scale thunderstorm with cold outflow occurring at a time and location during an allergen season in which large numbers of asthmatics are outdoors; and 4) sudden release of large amounts of respirable allergenic fragments, particularly fungal spores such as *Alternaria*.

IS THERE A RISK OF RELAPSE OF THUNDERSTORM-RELATED ASTHMA?

Whereas there is increasing evidence of a possible link between thunderstorms and asthma, the fact that relapses of thunderstorm-related asthma are also possible, as shown by the following case report, is less well known.

A 35-yr-old female who experienced near fatal asthma in concomitance with a thunderstorm in June 2004 and who was treated in the emergency department of Cardarelli hospital in Naples was admitted to the emergency department of the same hospital 7 yrs later, on May 24, 2011, despite appropriate treatment with salmeterol 50 μg and fluticasone 250 μg *b.i.d.* She was immediately treated with a high concentration of oxygen and 2 g *i.v.* methylprednisolone on admission to the emergency department. Given the severity of respiratory failure, arterial blood gas analysis was performed under oxygen therapy, giving 70 mmHg. Since the first episode, the patient had avoided being outdoors when a thunderstorm was approaching. The second time, an unexpected thunderstorm occurred while she was driving her motorbike and she experienced increasing dyspnoea that had to be treated in the emergency department some hours later. Clinical parameters were similar to those during the first episode. On those days, the *Parietaria* pollen count was higher than in the past 10 yrs in Naples in the same period of the year (260, 113 and 79.4 pollen·m^{-3} air in the previous 3 days, respectively), but no

TABLE 3 Hill's criteria [32] applied to thunderstorm-related asthma

Hill's criteria	Application to the thunderstorm-related asthma
Temporal relationship Exposure always precedes the outcome	Thunderstorms always precede asthma attacks
Strength The stronger the association, the more likely it is that the relationship of the factor to the health outcome is causal	Increased risk of asthma attacks in relation to thunderstorms
Dose–response relationship An increasing amount of exposure increases the probability of the health outcome Of note, the absence of a dose–response relationship does not rule out a causal relationship	Increased amount of pollen and mould spores at the beginning of the thunderstorm associated with increased probability of asthma attacks in pollen patients and other allergic patients
Consistency The association is consistent when results in different studies and among different populations	Association between thunderstorm and asthma found in different studies and different populations
Plausibility The association agrees with currently accepted understanding of pathological processes	Evidence of biological plausibility at the basis of thunderstorm-related asthma through pollen exposure (allergens and starch granules in the cytoplasm or other paucimicronic cytoplasmic-components carrying allergens) Evidence to be established in the case of mould spores
Consideration of alternate explanations It is necessary to consider multiple hypotheses before making conclusions about the causal relationship between any two items under investigation	Alternate hypothesis involving chemical air pollution less explanatory than thunderstorm-related asthma
Experimental The condition can be altered (prevented or ameliorated) by an appropriate experimental regimen	Evidence indicating that prevention is possible by avoiding exposure to thunderstorm (at its beginning) in pollen patients
Specificity This is established when a single putative cause produces a specific effect	Poorly shown by experimental data (also sparse and heterogeneous)
Coherence The association should be compatible with existing theory and knowledge	Existing theory and knowledge support the existence of thunderstorm-related asthma

air pollutants were observed. Her symptoms became stable within a couple of weeks following a short course of treatment with oral corticosteroids. She was offered specific immunotherapy treatment with *Parietaria* pollen extract but refused because of a possible pregnancy. Because this is a unique report, further investigations are needed to confirm thunderstorm-related asthma relapses.

CRITERIA APPLIED TO THUNDERSTORM-RELATED ASTHMA

To better understand the existing evidence on thunderstorm-related asthma, we explored the nature of the relationship between thunderstorm and asthma attacks by applying the criteria of HILL [32], usually used in epidemiological research to identify causation between a risk factor and a health event. Most of the criteria of HILL [32], although not all, support the hypothesis of a causal link of thunderstorms to asthma attacks through pollen exposure (table 3). The main reason for uncertainty is that data are still scant, in particular experimental data, which challenges the specificity criterion according to which a single putative cause produces a specific effect. MARKS *et al*. [19] demonstrated that the arrival of a thunderstorm outflow was accompanied by a large increase in the concentration of ruptured pollen grains in ambient air. PULIMOOD *et al*. [20] reported that in the thunderstorm in which they studied asthma, epidemics were associated with increased levels of *Alternaria*, *Cladosporium* and *Didymella* spp. but this has not been reproduced in any experiment.

Public health dimension

Depending on the size of the population at risk, thunderstorm-related asthma outbreaks may threaten the operative capacity of health services, as was the case in London [15]. Therefore, it is of interest to establish which risk factors may predict the occurrence of asthma outbreaks in such a way that early warning systems can be developed. With this approach, and using the same data set as above, NEWSON *et al*. [33] identified 56 asthma epidemics defined as periods of exceptionally high asthma admission counts compared with predictions of a log-linear autoregressive model. Of note, the authors measured pollen counts and, for the first time, thunderstorms using densities of sferics (lightning flashes). However, the data from NEWSON *et al*. [33] do not support the possibility of predicting asthma outbreaks by using meteorological data and pollen counts. In their study, thunderstorms and high grass pollen levels preceded asthma epidemics more often than expected by chance. However, most thunderstorms, even following high grass pollen levels, do not precede epidemics and most epidemics are not associated with thunderstorms or unusual weather conditions but to other factors, such as respiratory infections. An early warning system based on the indicators examined here would, therefore, detect few epidemics and generate an unacceptably high rate of false alarms. In addition, real-time data on both weather forecasting and pollination are not available in many countries at present. Finally, despite the increasing amount of data about allergen content of ambient air published in recent years [34, 35], to date, pollens or mould allergens have never been quantified during a thunderstorm. Hopefully, availability of pollen allergen count on a daily basis, as is planned in the frame of the European HIALINE project (www.hialine.eu), could provide conclusive data on the actual changes in the allergenic load of the air during such an extreme precipitative event. Preliminary data have shown that although pollen count and allergen in ambient air follow the same temporal trends, a 10-fold difference can exist in allergen potency of birch pollen, so that symptoms might be difficult to correlate with pollen counts, but perhaps better with allergen exposure [34].

CONCLUSIONS

Thunderstorm-related asthma is a dramatic example of the allergenic potential of pollen antigens. Pollen allergic patients who encounter the allergenic cloud of pollen are at higher risk of having an asthma attack. Subjects allergic to pollen who are in the path of the thunderstorm outflow are likely to inhale airborne pollen allergens and experience an airway asthmatic response. Relapse is also possible. A similar phenomenon has been hypothesised for mould spores.

Thunderstorms are sudden and poorly predictable in nature and a specifically designed study is therefore a challenge for researchers. However, it is mandatory to analyse available and forthcoming data with a multidisciplinary approach in order to address the full spectrum of this issue.

In conclusion, patients suffering from pollen allergy should be alerted of the danger of being outdoors during a thunderstorm in the pollen season. Patients who experienced an episode of severe thunderstorm-related asthma could be at risk of a relapse during a heavy precipitation event, even if receiving treatment.

STATEMENT OF INTEREST

None declared.

REFERENCES

1 D'Amato G, Liccardi G, Frenguelli G. Thunderstorm-asthma and pollen allergy. *Allergy* 2007; 62: 11–16.
2 Knox RB. Grass pollen, thunderstorms and asthma. *Clin Exp Allergy* 1993; 23: 354–356.
3 Packe GE, Ayres JG. Asthma outbreak during a thunderstorm. *Lancet* 1985; 2: 199–204.
4 Wallis DN, Davidson AC, Weilch J, *et al.* Clinical and immunological characteristics of patients with thunderstorm asthma. *Eur Respir J* 1995; 8: Suppl. 19, 500s.
5 Antò JM, Sunyer J. Thunderstorms: a risk factor for asthma attacks. *Thorax* 1997; 52: 669–670.
6 Newson R, Strachan D, Archibald E, *et al.* Acute asthma epidemics, weather and pollen in England, 1987–1994. *Eur Respir J* 1998; 11: 694–701.
7 Intergovernmental Panel on Climate Change. Climate Change 2007: The Physical Science Basis. Cambridge, Cambridge University Press, 2007.
8 D'Amato G, Cecchi L, Bonini S, *et al.* Allergenic pollen and pollen allergy in Europe. *Allergy* 2007; 62: 976–990.
9 Bousquet J, Khaltaev N, Cruz AA, *et al.* Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA(2)LEN and AllerGen). *Allergy* 2008; 63: 8–160.
10 Burney PGJ, Malmberg E, Chinn S, *et al.* The distribution of total and specific serum IgE in the European community respiratory health survey. *J Allergy Clin Immuno* 1997; 99: 314–322.
11 Pawankar R, Canonica GW, Holgate ST, *et al*. WAO White Book on Allergy. Milwaukee, World Allergy Organization, 2011. Available

from: www.worldallergy.org/UserFiles/file/WAO-White-Book-on-Allergy_web.pdf
12 Traidl-Hoffmann C, Jakob T, Behrendt H. Determinants of allergenicity. *J Allergy Clin Immunol* 2009; 123: 558–566.
13 Behrendt H, Becker WM. Localization, release and bioavailability of pollen allergens: the influence of environmental factors. *Curr Opin Immunol* 2001; 13: 709–715.
14 Bellomo R, Gigliotti P, Treloar A, *et al.* Two consecutive thunderstorm associated epidemic of asthma in Melbourne. *Med J Aust* 1992; 156: 834–837.
15 Venables KM, Allitt U, Collier CG, *et al.* Thunderstorm-related asthma – epidemic 24/25 June 1994. *Clin Exp Allergy* 1997; 27: 725–736.
16 Villeneuve PJ, Leech J, Bourque D. Frequency of emergency room visits for childhood asthma in Ottawa, Canada: the role of weather. *Int J Biometeorol* 2005; 50: 48–56.
17 Grundstein A, Sarnat SE, Klein M, *et al.* Thunderstorm associated asthma in Atlanta, Georgia. *Thorax* 2008; 63: 659–660.
18 Girgis ST, Marks GB, Downs SH, *et al.* Thunderstorm-associated asthma in an inland town in southeastern Australia. Who is at risk? *Eur Respir J* 2000; 16: 3–8.
19 Marks GB, Colquhoun JR, Girgis ST, *et al.* Thunderstorm outflows preceding epidemics of asthma during spring and summer. *Thorax* 2001; 56: 468–471.
20 Pulimood TB, Corden JM, Bryden C, *et al.* Epidemic asthma and the role of the fungal mold *Alternaria alternate*. *J Allergy Clin Immunol* 2007; 120: 610–617.
21 D'Amato G, Liccardi G, Gilder JA, *et al*. Thunderstorm-associated asthma in pollinosis patients. 2005, BMJ. www.bmj.com/rapid-response/2011/10/30/thunderstorm-associated-asthma-pollinosis-patients.
22 Losappio L, Heffler E, Contento, *et al.* Thunderstorm-related asthma epidemic owing to *Olea Europea* pollen sensitization. *Allergy* 2011; 66: 1510–1511.
23 Howden ML, McDonald CF, Sutherland MF. Thunderstorm asthma – a timely reminder. *Med J Aust* 2011; 195: 512–513.
24 D'Amato G, Ruffilli A, Ortolani C. Allergenic significance of Parietaria (pellitory-of-the-wall) pollen. *In:* D'Amato G, Spieksma F, Bonini S, eds. Allergenic Pollen and Pollinosis in Europe. Oxford, Blackwell Scientific Publications, 1991; pp. 113–118.
25 Nasser SM, Pulimood TB. Allergens and thunderstorm asthma. *Curr Allergy Asthma Rep* 2009; 9: 384–390.
26 D'Amato G, Cecchi L, Liccardi G. Thunderstorm-related asthma: not only grass pollen and spores. *J Allergy Clin Immunol* 2008; 121: 537–538.
27 Taylor PE, Hagan R, Valenta R, *et al.* Release of allergens in respirable aerosols: a link between grass pollen and asthma. *J Allergy Clin Immunol* 2002; 109: 51–56.
28 Suphioglu C. Thunderstorm asthma due to grass pollen. *Int Arch Allergy Immunol* 1998; 116: 253–260.
29 Motta A, Peltre G, Dormans JA, *et al. Phleum pratense* pollen starch granules induce humoral and cell-mediated immune responses in a rat model of allergy. *Clin Exp Allergy* 2004; 34: 310–314.
30 Jato V, Rodríguez-Rajo FJ, González-Parrado Z, *et al.* Detection of airborne Par j 1 and Par j 2 allergens in relation to Urticaceae pollen counts in different bioclimatic areas. *Ann Allergy Asthma Immunol* 2010; 105: 50–56.
31 Abou Chakra O, Rogerieux F, Poncet P, *et al.* Ability of pollen cytoplasmic granules to induce biased allergic responses in a rat model. *Int Arch Allergy Immunol* 2011; 154: 128–136.
32 Hill AB. The environment and disease: association or causation? *Proc R Soc Med* 1965; 58: 295–300.
33 Newson R, Strachan D, Archibald E, *et al.* Effect of thunderstorms and airborne grass pollen on the incidence of acute asthma in England, 1990–1994. *Thorax* 1997; 52: 680–701.
34 Buters JT, Weichenmeier I, Ochs S, *et al.* The allergen Bet v 1 in fractions of ambient air deviates from birch pollen counts. *Allergy* 2010; 65: 850–858.
35 De Linares C, Díaz de la Guardia C, Nieto Lugilde D, *et al.* Airborne study of grass allergen (Lol p 1) in different-sized particles. *Int Arch Allergy Immunol* 2010; 152: 49–57.