Thunderstorms: a risk factor for asthma attacks

J M Antó, J Sunyer

Sometimes thunder and wheezes come together. One of the last occasions when such a combination was detected was in London during the summer of 1994. At 18.00 hours on 24 June there was a heavy thunderstorm with about 50 ground strikes of lightning, a fall in temperature from 26.5°C to 18.7°C, a rise in humidity from 50% to 87%, and rainfall of 46 mm.[1] A few hours later there was a massive increase in the number of patients presenting at the accident and emergency departments of several hospitals with wheezing and shortness of breath. In total, 604 patients were seen in 12 accident and emergency departments compared with an expected number of 66.[2] Was there any causal link between these two notable and uncommon events?

Strong arguments in favour of the possibility that asthma outbreaks are caused by thunderstorms have been provided by researchers in Melbourne where both events have occurred simultaneously on several occasions.[3] The outbreaks of asthma in Melbourne were of a similar relative size to that in London with about a tenfold increase in the number of patients presenting to the accident and emergency departments with asthma. Patients affected during these outbreaks were more likely to be sensitised to rye grass pollen than non-epidemic asthmatic patients, and subsequently showed well defined responses to inhalation challenge tests with isolated starch granule suspensions containing *Lol p* IX obtained from pollen grains. Thunderstorms were thought to play a part by providing the appropriate circumstances for the pollen grains to break and release hundreds of starch granules of a respirable size, and in vitro evidence for this hypothesis was provided.[4] Although the Melbourne outbreaks were suited to the investigation of their mechanisms, well defined epidemiological evidence of an association between the occurrence of asthma outbreaks and thunderstorms was not investigated; in addition, the studies of the mechanisms were performed in a small number of subjects so some doubt over the causality of the association remains.[5]

The simultaneous occurrence of asthma outbreaks and thunderstorms was also noticed by Packe and Ayres when describing an outbreak of asthma which occurred in Birmingham in 1983.[6] In this paper the authors cited papers published since the 1950s which showed that rainfall may favour the release of fungal spores into the atmosphere, suggesting that, after thunderstorms with heavy rain, the effect on spore release may outweigh the effect of rain on the removal of suspended particles.[7 8]

Asthma outbreaks are not easily amenable to epidemiological investigation because they tend to be rare. When a suspected association is not testable because of the small number of outbreaks, one can focus on the usual variations of asthma and determine whether thunderstorms are a risk factor for asthma admissions in a large population. The study in this issue of *Thorax* by Newson *et al* is an important contribution to this latter approach.[9]

The authors looked for evidence of an independent association between the occurrence of thunderstorms and the number of asthma admissions in hospitals of several regional health authorities in England, and also determined whether that association was modified by the level of airborne grass pollen. With this approach the authors were able to test the behaviour of a population of several million, including several hundreds of thousands of asthmatic subjects, on about 1200 different days. A log linear model with a modification to account for repeated measures was used, which is one of the statistical methods to be used with time series. This type of technique is widely available and has recently been reliably used in a large multinational European study on the short term health effects of air pollution.[10] A contribution of the study, which is of interest for future epidemiological studies, is the measure of thunderstorms using records of lightning flashes as provided by the Meteorological Office. Each flash of lightning corresponds to a sferic, which consists of an electromagnetic signal emitted during the lightning discharge. According to Lee[11] this method provides much greater spatial resolution than records of ground based observers.

Using 15 hour lagged values of sferics an association was found between thunderstorms with strong activity (high sferic density) and asthma admissions for both children and adults (RR 1.25; 95% CI 1.15 to 1.36), but there was a smaller association with low sferic density compared with the absence of sferics. This association was geographically consistent as it was present in 12 of the 14 geographical areas for those aged 14 years and over and in 10 of the 14 areas for those aged 0–14 years.

The association of a high level of pollen (≥ 50 g/m^3/day) with asthma admissions, assessed in only five geographical areas, was modified by the sferics. Whereas there was a non-significant increase of only 3% in asthma admissions on days with a high pollen count and no sferics, the increase in asthma admissions was approximately 31% when both high pollen counts and high sferic density coincided (RR 1.31; 95% CI 1.21 to 1.42). This pattern was again geographically consistent.

The findings reported by Newson *et al* deserve consideration from two distinct angles – namely, aetiology and public health. They are important from an aetiological perspective because they provide additional evidence in support of a causal relationship between thunderstorms and attacks of asthma. Unfortunately the categorisation of sferics resulted in very small numbers in the high sferic density group for which an association was observed, and the criteria for stratification of sferics was not given. The relationship between thunderstorms, pollen counts, and asthma admissions was carefully adjusted for day-of-the-week and seasonal effects as well as for the potential impact of administrative changes in the area covered by some health authorities. Could other non-reported factors have confounded the reported association? Viral infections have been found to be a risk factor for asthma attacks both in children[12] and adults[13] and, at least in theory, may have been seasonally correlated with the distribution of thunderstorms. Whether adjustment for the season could have involved partial adjustment for viruses is difficult to say. Air pollution may also be considered as a potential confounder if days with high sferic densities tended to display higher air pollution levels than days without sferics. Air pollution has been found to be associated with asthma admissions in the UK[14] and elsewhere,[15] although the strength of this association is substantially weaker than the one reported for thunderstorms. Although the authors

point out that the asthma outbreak that occurred in London in June 1994 did not coincide with high air pollution levels, in the current study adjustment for air pollution was not provided. However, since air pollution has been found to increase bronchoconstriction in asthmatic subjects after controlled exposure to allergens,[16 17] pollutants could in theory have had a double role as both a confounder and a synergistic factor, and this complexity may have made the authors reluctant to include air pollution in the time series models.

Another relevant issue on aetiology is whether the presence of thunderstorms is an indirect or a related variable to other meteorological factors more directly linked to the mechanisms that supposedly trigger asthma attacks. As previously mentioned, in vitro experiments by Suphioglu *et al* have shown that pollen grains are ruptured in rain water by osmotic shock, each grain releasing hundreds of starch granules of a respirable size (<3 μm) which contain *Lol p* IX.[4] A measure of osmotic shock may perhaps have been able to reveal a more direct mechanism and thus a larger relative risk. During the London outbreak marked changes were noted in humidity, temperature, and rainfall, and we do not know to what extent inclusion of these variables in the study by Newson *et al* could have provided different results. Future studies based on the same approach are needed to investigate other thunderstorm-associated variables potentially involved in triggering asthma attacks.

The importance of the role of thunderstorms in asthma attacks from a public health perspective was demonstrated by the large outbreak that occurred in London when, in a few hours, many casualty departments were overwhelmed by patients with asthma attacks, some having run out of medicines. For this reason predicting the occurrence of thunderstorms likely to cause asthma outbreaks may be important for public education. Unfortunately, the present study tell us little about the relation between thunderstorms and asthma outbreaks since there was no indication of days with an unusually high occurrence of asthma during the period of the study. However, the occurrence of asthma outbreaks cannot be totally ruled out since the study included only hospital admissions for asthma and not visits to the accident and emergency departments. Most reported asthma outbreaks have shown that a substantial proportion of affected people were able to return home after treatment at the emergency department without needing to be admitted. In the London outbreak only 16% of those seen at the emergency departments were admitted.[2]

Is the reported association sufficiently relevant to recommend warning systems to predict thunderstorms likely to affect asthmatic subjects and to establish appropriate informative strategies for the patients? Newson and coauthors speculate that, assuming high pollen counts amplify the high sferic effect as much as the low sferic effect, the simultaneous presence of both high pollen levels and high sferic density may increase the number of asthma admissions by 45% in children and nearly double it in adults. In the National Institutes of Health review on the global strategy for asthma management and prevention published in January 1995 it was recognised that, although adverse weather conditions have been associated with asthma exacerbations, these factors have not been examined systematically and in depth.[18] Taking into account the evidence provided by the researchers in Melbourne, the study by Newson *et al* has provided additional evidence that thunderstorms increase the risk of asthma exacerbations and future reviews should certainly consider this point.

Respiratory and Environmental Health Research Unit, J M ANTÓ
Institut Municipal d'Investigacio Mèdica, J SUNYER
Universitat Autonoma de Barcelona,
Doctor Aiguader 80,
08003 Barcelona,
Spain

1 Celenza A, Fothergill J, Kupek E, Shaw RJ. Thunderstorms associated asthma: a detailed analysis of environmental factors. *BMJ* 1996;**312**: 604–7.
2 Davidson AC, Emberlin J, Cook A, Venables KM and Thames Region Accident and Emergency Trainees Association. A major outbreak of asthma associated with a thunderstorm: experience of accident and emergency departments and patients' characteristics. *BMJ* 1996;**312**:601–4.
3 Bellomo R, Gigliotti P, Treloar A, Holmes P, Suphioglu C, Singh MB. Two consecutive thunderstorm associated epidemics of asthma in Melbourne. *Med J Aust* 1992;**156**:834–7.
4 Suphioglu C, Singh M, Taylor P, Bellomo R, Holmes P, Puy R, *et al.* Mechanism of grasspollen-induced asthma. *Lancet* 1992;**339**:569–72.
5 Antó JM. Asthma outbreaks: an opportunity for research? *Thorax* 1995;**50**: 220–2.
6 Packe GE, Ayres JG. Asthma outbreak during a thunderstorm. *Lancet* 1985; **ii**:199–203.
7 Hirst JM. An automatic volumetric spore trap. *Ann Appl Biol* 1959;**39**: 257–65.
8 Frankland AW, Gregory PH. Allergenic and agricultural implications of airborne ascospore concentrations from a fungus, *Didymella exitialis. Nature* 1973;**245**:336–7.
9 Newson R, Strachan D, Archibald E, Emberlin J, Hardaker P, Collier C. Effect of thunderstorms and airborne grass pollen on the incidence of acute asthma in England, 1990–94. *Thorax* 1997;**52**:680–5.
10 Schwartz J, Spix C, Touloumi G, Bachárová L, Barumamdzadeh T, le Tertre A, *et al.* Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Community Health* 1996;**50**(Suppl 1):S3–11.
11 Lee ACL. Ground truth confirmation and theoretical limits of an experimental VLF arrival time difference lightning flash locating system. *Q J R Met Soc* 1989;**115**:1147–66.
12 Johnston S, Pattemore PK, Sanderson G, Smith S, Lampe F, Joseph L, *et al.* Community study of role of viral infections in exacerbations of asthma in 9–11 old children. *BMJ* 1995;**310**:1225–9.
13 Nicholson KG, Kent J, Ireland DC. Respiratory viruses and exacerbations of asthma in adults. *BMJ* 1993;**307**:982–6.
14 Walters SM, Phupinyokul M, Ayres JG. Hospital admissions rates for asthma and respiratory diseases in West Midlands: their relationship to air pollution levels. *Thorax* 1995;**50**:848–54.
15 Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 1993;**147**:826–31.
16 Tunnecliffe WS, Burge PS, Ayres JG. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 1994;**344**:1733–6.
17 Devalia JL, Rusznack C, Herdman MJ, Trigg CJ, Tarraf H, Davies RJ. Effect of nitrogen dioxide and sulphur dioxide on airway response of mild asthmatic patients to allergen inhalation. *Lancet* 1994;**344**:1668–71.
18 *Global strategy for asthma management and prevention.* NIH Publication No. 95-3659, January 1995.