

# Thunderstorm-related asthma – the epidemic of 24/25 June 1994

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## Summary

**Background** A large epidemic of asthma occurred following a thunderstorm in southern and central England on 24/25 June 1994. A collaborative study group was formed.

**Objectives** To describe the epidemic and the meteorological, aerobiological and other environmental characteristics associated with it.

**Methods** Collation of data from the Meteorological Office, the Pollen Research Unit, the Department of the Environment's Automatic Urban Network, from health surveillance by the Department of Health and the National Poisons Unit, from clinical experience in general practice and hospitals, and from an immunological study of some of the affected cases from north east London.

**Results** The thunderstorm was a Mesoscale Convective System, an unusual and large form of storm with several centres and severe wind gusts. It occurred shortly after the peak grass pollen concentration in the London area. A sudden and extensive epidemic occurred within about an hour affecting possibly several thousand patients. Emergency services were stretched but the epidemic did not last long. Cases had high serum levels of IgE antibody to mixed grass pollen.

**Conclusion** This study supports the view that patients with specific IgE to grass pollen are at risk of thunderstorm-related asthma. The details of the causal pathway from storm to asthma attack are not clear. Case-control and time series studies are being carried out.

**Keywords:** asthma, allergy, weather, thunderstorm, epidemic, outbreak, aerobiology, pollen

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## Introduction

A few well documented epidemics of asthma attacks have occurred in relation to summer thunderstorms, notably in Birmingham, UK in 1983 [1], Nottingham, UK in 1984 [2] and Melbourne, Australia in 1984, 1987 and 1989 [3,4]. The

common feature of these epidemics was that most affected patients had a history of hay fever, although some had not previously suffered from asthma. Levels of gaseous air pollutants were not unusually high at the time of these outbreaks but, in general, thunderstorms are known to have a marked effect on fungal spores, pollens, and dust and smoke particles.

Rain produces a profound change in the content of the air [5]. Dust, smoke particles, pollen and those fungal spores typical of dry air are washed out after rain, sometimes after a transient rise in spores such as *Cladosporium*. Ascospores,

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the spores of the sexual state of ascomycete fungi, which need water for their release, then take their place in the air. 'Mirror yeasts' can then grow and sporulate on moist leaves. In the Birmingham epidemic airborne spore counts of *Didymella* and the 'mirror yeast' *Sporobolomyces* rose markedly shortly after the storm [1]. While some patients with late summer asthma are allergic to *Didymella* extracts [6] it is not clear if fungal allergy was important in the Birmingham epidemic [7].

In Melbourne, patients admitted during the episode were highly allergic to rye grass pollen and one of the potent rye grass allergens *Lol p IX* has been identified in starch granules contained within pollen grains [8]. These granules are released in large numbers when the pollen comes in contact with water, are small enough to enter the pulmonary airways, and are capable of provoking bronchoconstriction in experimental situations [8]. This suggests that allergic reactions to osmotically disrupted grass pollen may contribute to the occasional reports of asthma epidemics in relation to early summer thunderstorms.

A further epidemic of asthma occurred across southern and eastern England in association with a thunderstorm on 24 June 1994 [9,10]. The characteristics of patients presenting in the London area have been reported [10] and the high prior grass pollen count and fall in temperature have been suggested as possible co-factors in London [11]. In this paper we take a wider geographical perspective and summarize work nationwide describing the meteorological features of the storm, the associated aerobiology and pollutant levels, the resulting epidemic and the response of the health services.

### Meteorology

On the afternoon of Friday 24 June 1994 a major thunderstorm system developed in the region of the Cherbourg peninsula, and moved steadily north and east over south east England. Figure 1 shows the synoptic weather chart at 1200 UTC (1300 BST). (UTC = Universal Time Clock, an atomic standard of time which has replaced Greenwich Mean Time (GMT), an astronomical standard of time. BST = British Summer Time, 1 h ahead of UTC/GMT.) Over the preceding 6 h a cold front moved slowly from the west becoming stationary over the east of Ireland and west of Cornwall. Warm moist air at low levels was carried northwards from France, and weak low level convergence occurred into an area running roughly north-south through London.

The air over southern England and the Channel was potentially unstable, and by midday this instability was being released over south west England and central southern England, continuing during the afternoon. However, during the late afternoon explosive development occurred over the Channel, as shown by the rapid expansion of the cloud

tops in Fig. 2. Several thunderstorm cells merged to form a Mesoscale Convective System (MCS) as described by Browning and Hill in 1984 [12]. In fact, two main cells are evident from data recorded by the Meteorological Office Sferics (lightning) Arrival Time Difference system, Fig. 3; one moving northwards slightly to the west of London, and the other moving north-eastwards over east London into East Anglia. During the late evening the thunderstorm outflows produced convergence leading to the development of further thunderstorm cells in the north of England (Fig. 2).

Work is continuing aimed at understanding the dynamics of the MCS using mesoscale observational analysis and simulations with the Meteorological Office Large Eddy Simulation numerical model. It is already evident that the system produced an unusually strong low level outflow in the direction of movement of the storm. Winds gusted up to about 50 knots (25 m/s) over a relatively narrow corridor running from the south coast centred east of Brighton through to east London and into East Anglia. Smaller, although still strong, outflows were also associated with the other main convective cells north of Chichester and later around Sheffield. Figure 4 shows the sudden increase in wind speed observed at Gravesend, and the associated 5 °C decrease in temperature and greater than 30% increase in relative humidity observed at the London Weather Centre. These changes were followed about half an hour later by a burst of heavy rainfall.

### Aerobiology

Grass pollen was the most significant aeroallergen present in the period leading up to the storm. In central and southern England the peak of the 1994 grass pollen season occurred a few days before 24 June. Warm dry weather caused a rapid rise in pollen concentrations on 20 June and the highest counts of the year occurred over the next two to three days. London had an exceptionally high daily average pollen count for the urban area (258 grains per cubic metre) on 22 June (Fig. 5). This was the highest count recorded for 6 years. The concentration of grass pollen remained very high until it rained during the evening of 24 June. After this period the count returned to moderate or low.

The other main pollen types in the atmosphere during this period were *Tilia* (lime), *Plantago* (plantain), *Rumex* (dock), *Castanea* (sweet chestnut), *Sambucus* (elder) and *Urtica* (nettle). The daily average concentrations of these exhibited the same general patterns as the grass pollen counts but the counts were low.

Before the storm the fungal spores in the air in central and southern England were typical of a dry day at that time of year. Spores of the common saprophyte *Cladosporium* and the cereal smut *Ustilago avenae* were particularly common.

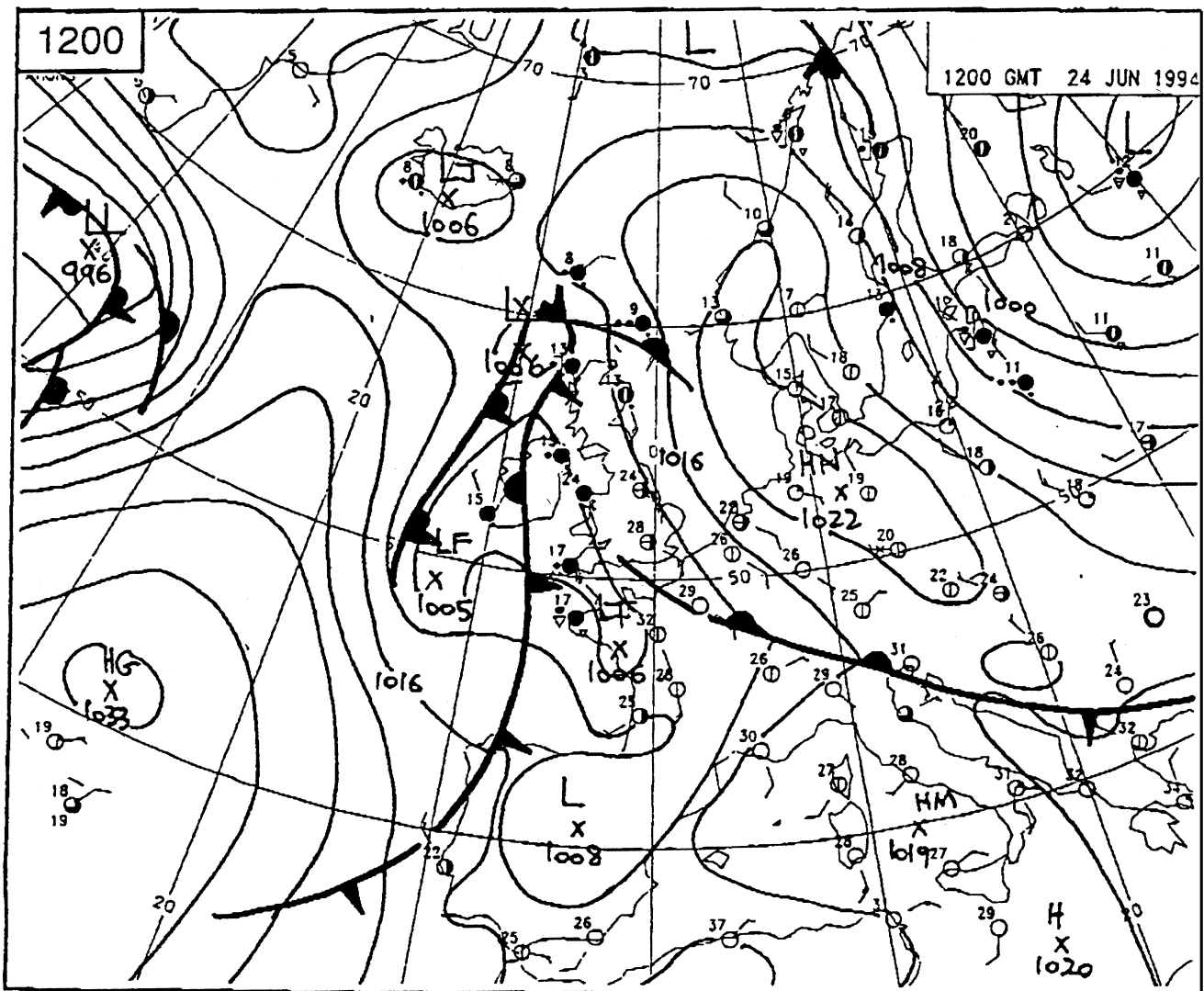


Fig. 1. Synoptic meteorological situation at 12.00 UTC 24 June 1994.

There were differences from site to site in the changes in fungal spore content in the air during the storm. At London and Runnymede there was an increase in dry powdery wind-released spores just before the rain, presumably because of the strong gusts of wind before the storm. At both Cambridge and Leicester there was a transient increase in *Cladosporium* spores (Fig. 6). After the rain there were ascospores from many different species except in London, where there were few ascospores. This was particularly so in Cambridge and Norwich, where the ascospores of *Phaeosphaeria nigrans* and of the fungal family *Diatrypaceae* were present in unusually high concentrations. *Sporobolomyces* spores did rise to extremely high levels (Fig. 6) but not until 05.00 BST (04.00 UTC) the next day, hours after the epidemic of asthma occurred. Concentrations of

*Didymella* ascospores were not unusually high at any of the sites examined to date.

#### Pollution

Concentrations of air pollutants in the London area recorded by the Department of the Environment's Automatic Urban Network (AUN) during the week of the storm were highest on the day of the storm, probably because of the calm, still conditions that preceded the storm. In the London area the highest hourly average concentrations of  $O_3$ ,  $NO_2$  and  $SO_2$  were 44, 126 and 48 p.p.b. (88, 237 and  $137 \mu g/m^3$ ), respectively. None of these values exceeded the WHO air quality one hour guidelines. In southern rural areas the maximum hourly  $O_3$  concentration was, as would be expected, higher:

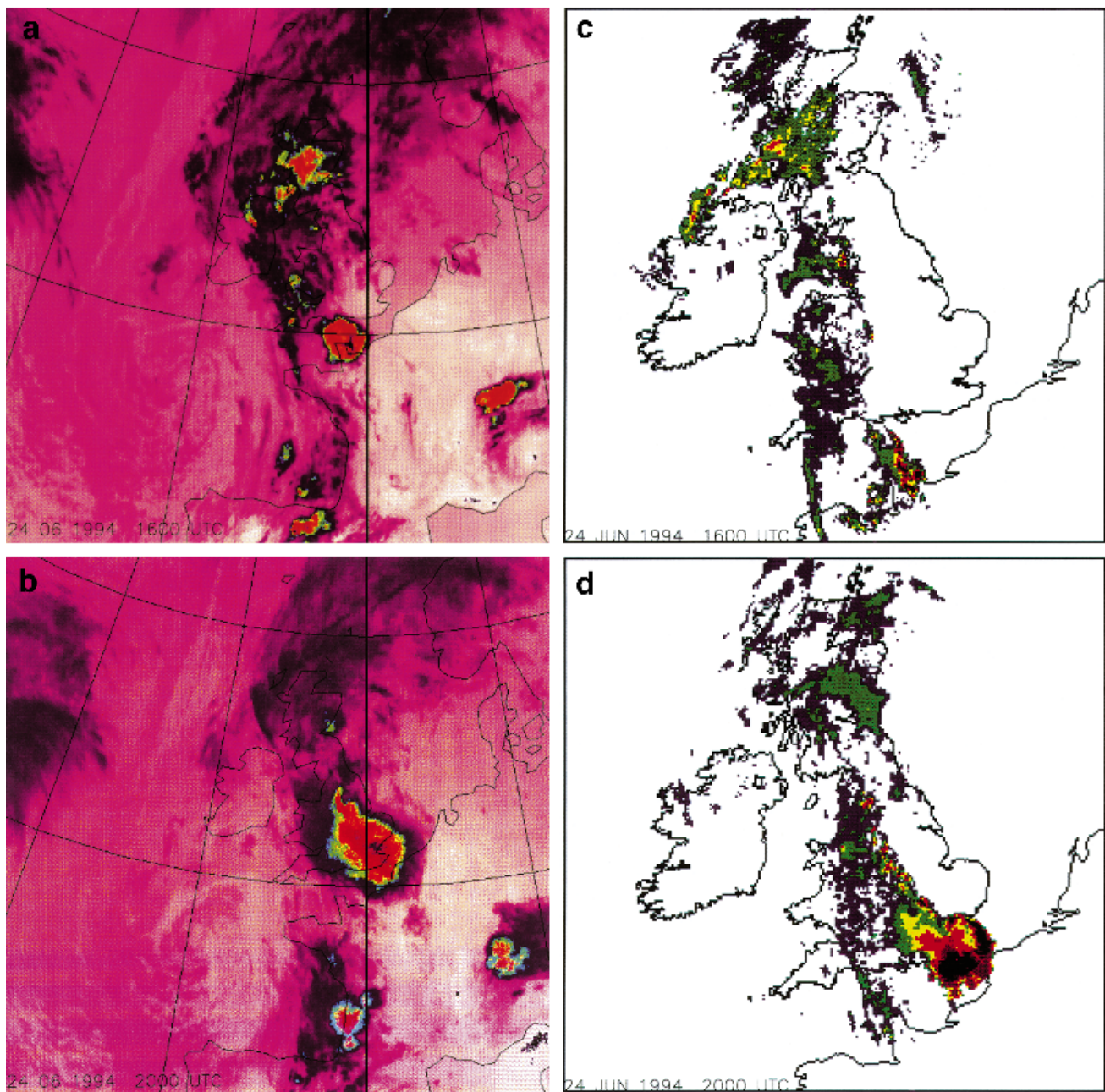


**Fig. 2.** Rapid development of cloud tops (left hand figures) between 16.00 and 20.00 UTC on 24 June 1994 as observed by the infrared radiometer channel on the European satellite Meteosat. The colours represent cloud top temperatures. Also shown are corresponding radar images (right hand figures). In this case the colours represent instantaneous rainfall rates with red and black being heavy rainfall. Each small square represents the rainfall averaged over a 25 km<sup>2</sup> area.

84 p.p.b. (168  $\mu\text{g}/\text{m}^3$ ) at Sibton. The highest hourly average concentration of PM<sub>10</sub> recorded by the AUN in the London area was 102  $\mu\text{g}/\text{m}^3$  at 09.00 BST (08.00 UTC) at the Bloomsbury site. These levels were not unusually high and would not have been expected to cause serious health effects.

The PM<sub>10</sub> monitors in the AUN are not expected to record intact grass pollen although smaller fragments may be detected. The Burkard slide results from a pollen monitoring site at Holloway, North London, showed that during the day there was a steady and marked accumulation of particulates especially in the respirable fraction (<10  $\mu\text{m}$ ) from about

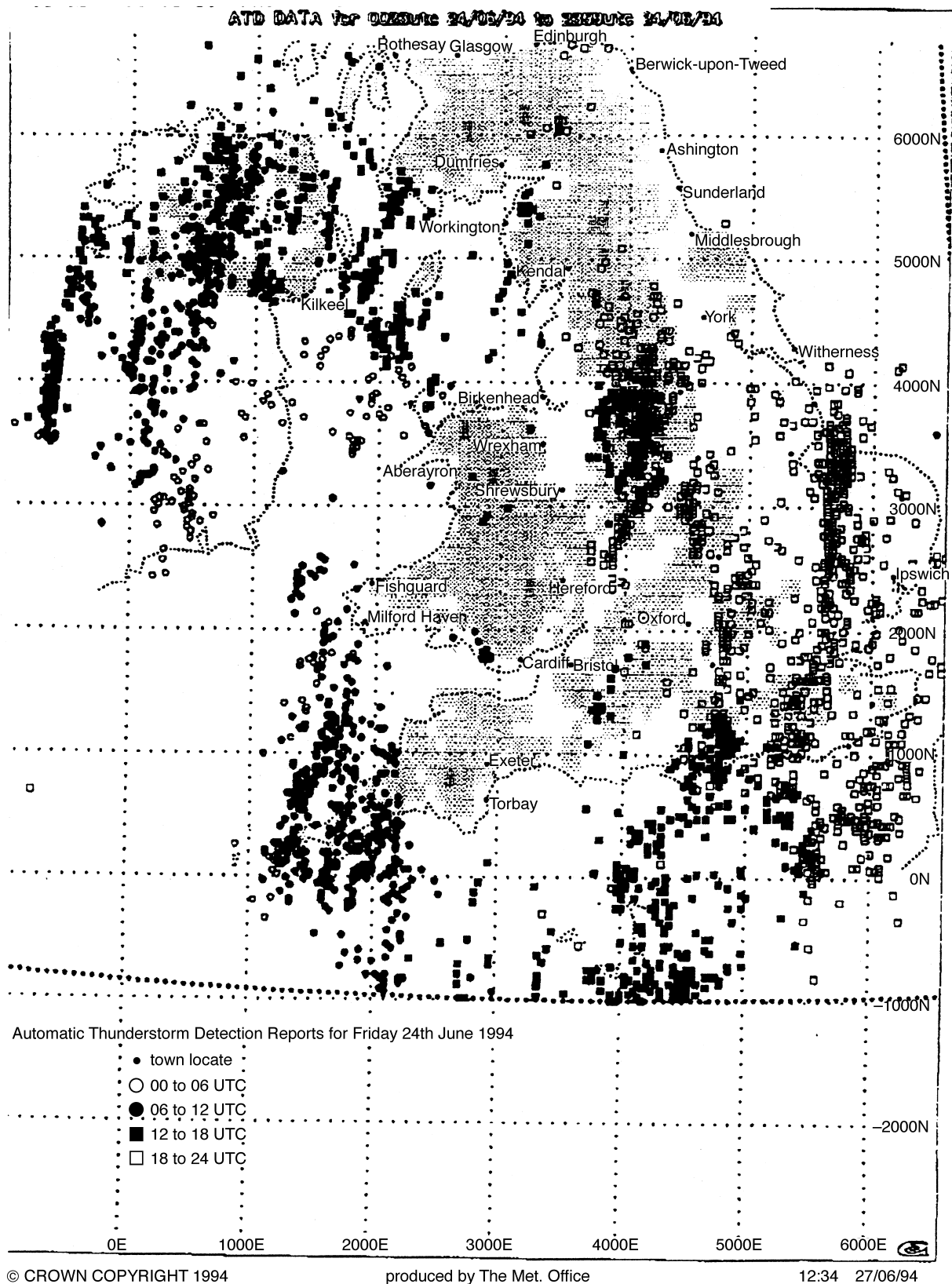
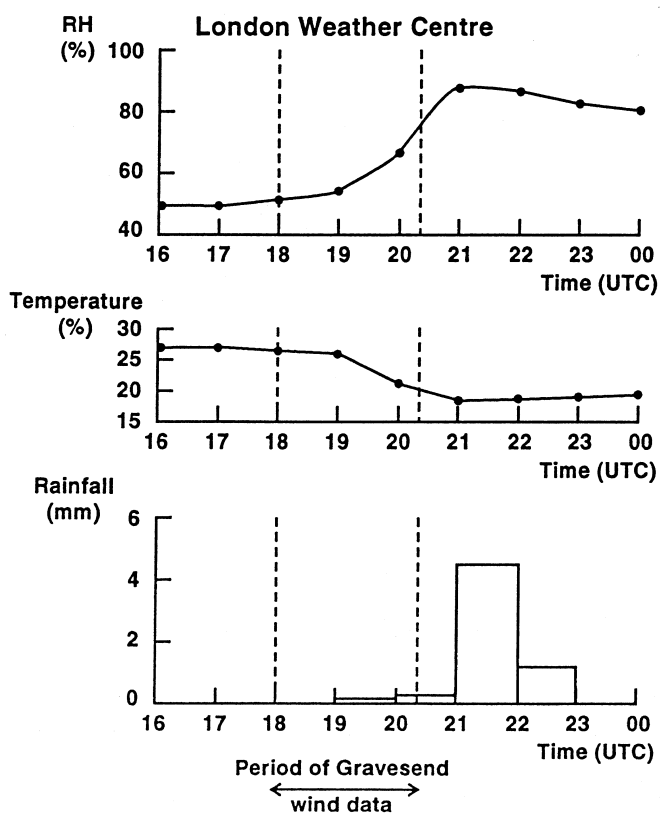
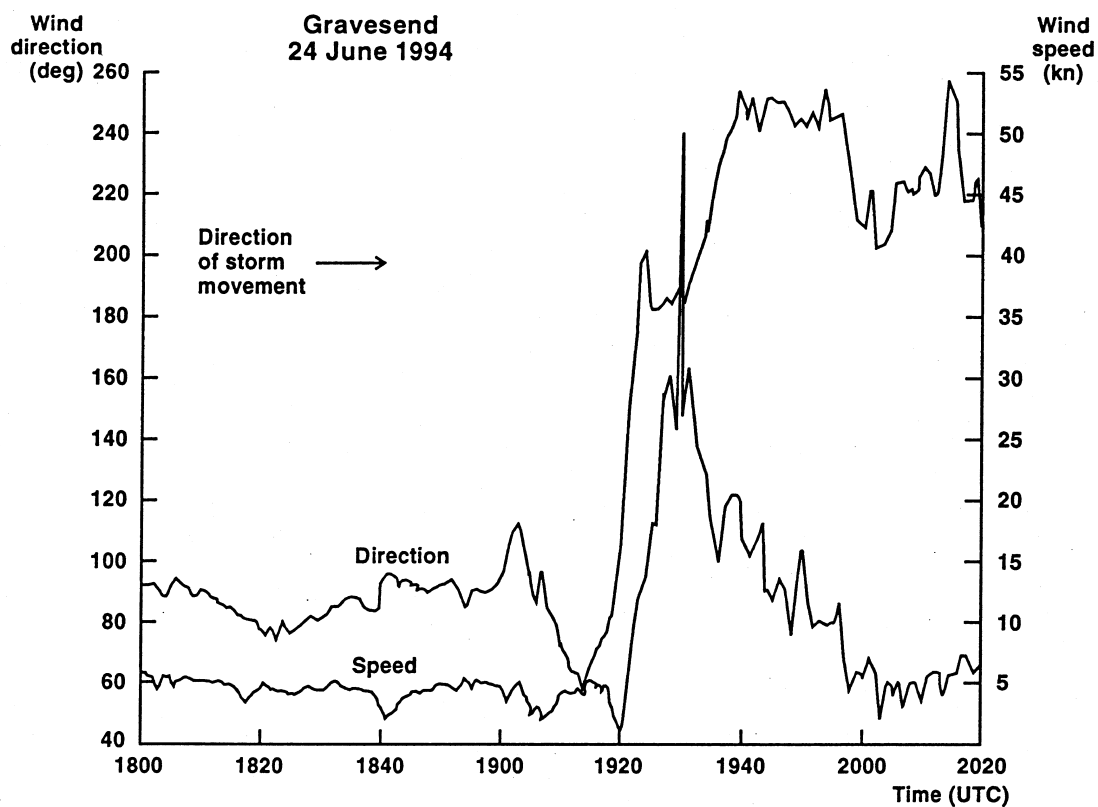


**Fig. 3.** ATD sferics data between 00.23 and 23.59 UTC on 24 June 1994 (courtesy E. Spackman, Meteorological Office).



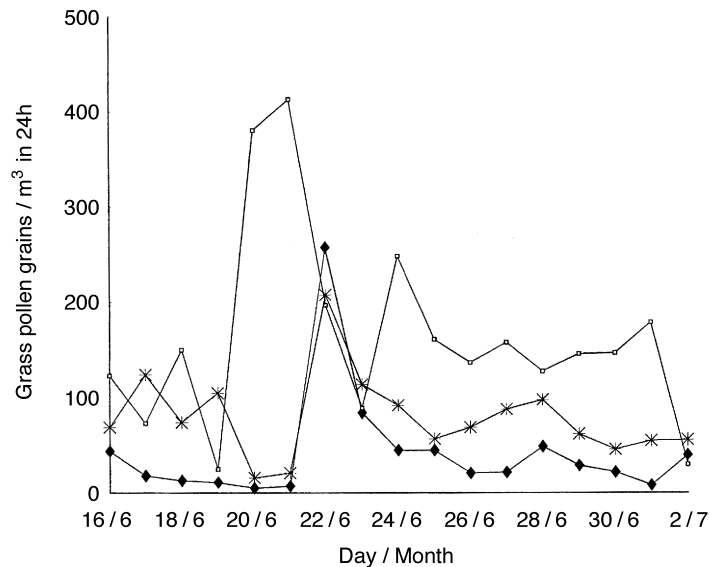


Fig. 5. Airborne grass pollen concentrations in the days around 24 June. —□—, Derby; \*, Cambridge, —◆—, London.

09.00 BST (08.00 UTC) increasing to a peak at about 19.00–20.00 BST (18.00–19.00 UTC) (Fig. 7).

#### General practice

One of us (J.H.) was second on call for a doctor's deputizing service in Luton, north of London, on 24 June and watched a spectacular display of lightning and thunder before going to bed. In the early hours of the morning J.H. was called in to help the regular deputizing doctors who had been overwhelmed by the number of calls from patients with difficulty in breathing. Examining the records that evening in the control room it was clear that the storm was of sufficient ferocity that it was unsafe to drive and the doctors on call had to pull their cars off the road. Meanwhile within 20 min of the onset of the storm an ever increasing number of people started to call for visits. Their main complaint was of hay fever with breathlessness, for these were people who did not recognize their asthma, never having suffered from it before [13].

Because it was clear that something very unusual had happened the records from the 29 other offices of the deputising service were subsequently examined. These showed that the epidemic of asthma had affected an area from the south coast to Yorkshire. It was possible to estimate the time of the storm from the peaks in the number of calls, from 20.00 BST (19.00 UTC) on the

south coast to 23.00 BST (22.00 UTC) in Yorkshire. Extrapolation of the figures from those calling the deputizing service suggests that, nationally, about 1600 extra patients with acute asthma associated with the thunderstorm called their GP that night. A full report on the impact on general practice is about to be published [13a].

#### Accident and Emergency

At the same time, patients with asthma were arriving by ambulance or their own transport at Accident and Emergency (A&E) departments over a wide area of the UK. At least 37 hospitals are known to have been affected. At Whipps Cross Hospital in North East London, so many arrived in such a short period that it was thought important to exclude the possibility of a major gassing incident and the National Poisons Unit at Guy's Hospital was contacted. Informal surveillance of A&E departments revealed  $\approx 1000$  excess attendances for asthma that night in the UK [9].

In affected hospitals, emergency attendances for asthma were about ten times the usual number in 24 h. The rate of attendance increased from about 20.00 BST and reached a peak in the early hours of the morning, falling by 08.00 the following morning but with a secondary, smaller peak of attendances later in the morning. In North Thames, one of the most affected health service regions, seven hospitals had more than 50 attendances and two had more than 100 in the

Fig. 4. Wind speed and direction recorded at Gravesend between 18.00 and 20.20 UTC on 24 June 1994. Also shown are the variations of temperature, relative humidity and rainfall at the London Weather Centre between 16.00 and 23.59 UTC on the same day.

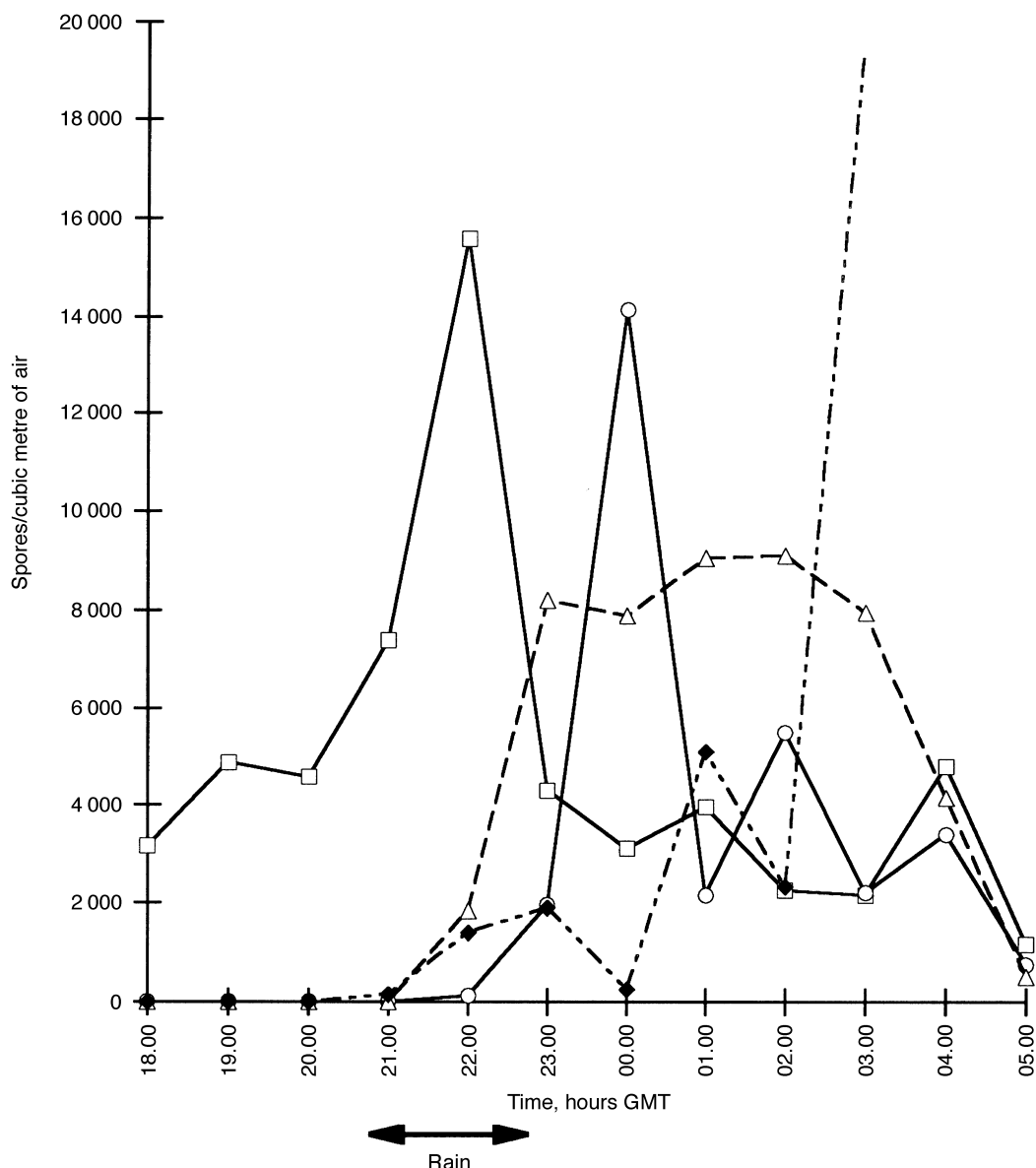


**Fig. 6.** Airborne fungal spore concentrations during the storm of 24 June in Cambridge. □, *Cladosporium*; ○, *Phaeosphaeria nigrans*; △, *Diatrypeaceae*; ◆, *Sporobolomyces*.

period 22.00 h 24 June to 13.00 h 25 June. The expected number of attendances was of the order of five to 10.

The patients were predominantly young adults, several hospitals noting that about half were in their 20s or 30s. Typically, there was a history of hay fever, but not of asthma. In general, those patients with a diagnosis of asthma usually had mild and intermittent symptoms and were not taking inhaled prophylactic steroids.

The asthma attacks in the epidemic were severe but, in general, of short duration and most patients were discharged home from A&E. The A&E departments became crowded with patients requiring nebulized bronchodilator therapy;

many departments needed additional oxygen cylinders, nebuliser facemasks or other equipment; pharmacists or other staff were called from home and nurses worked under considerable pressure. One A&E specialist commented that the epidemic was comparable in scale to a major accident affecting each hospital (D. Williams, pers. comm.). A case series from 12 London hospitals gives more detail [10].

#### *The Chief Medical Officer's request for information*

Subsequently, the Chief Medical Officer approved of a

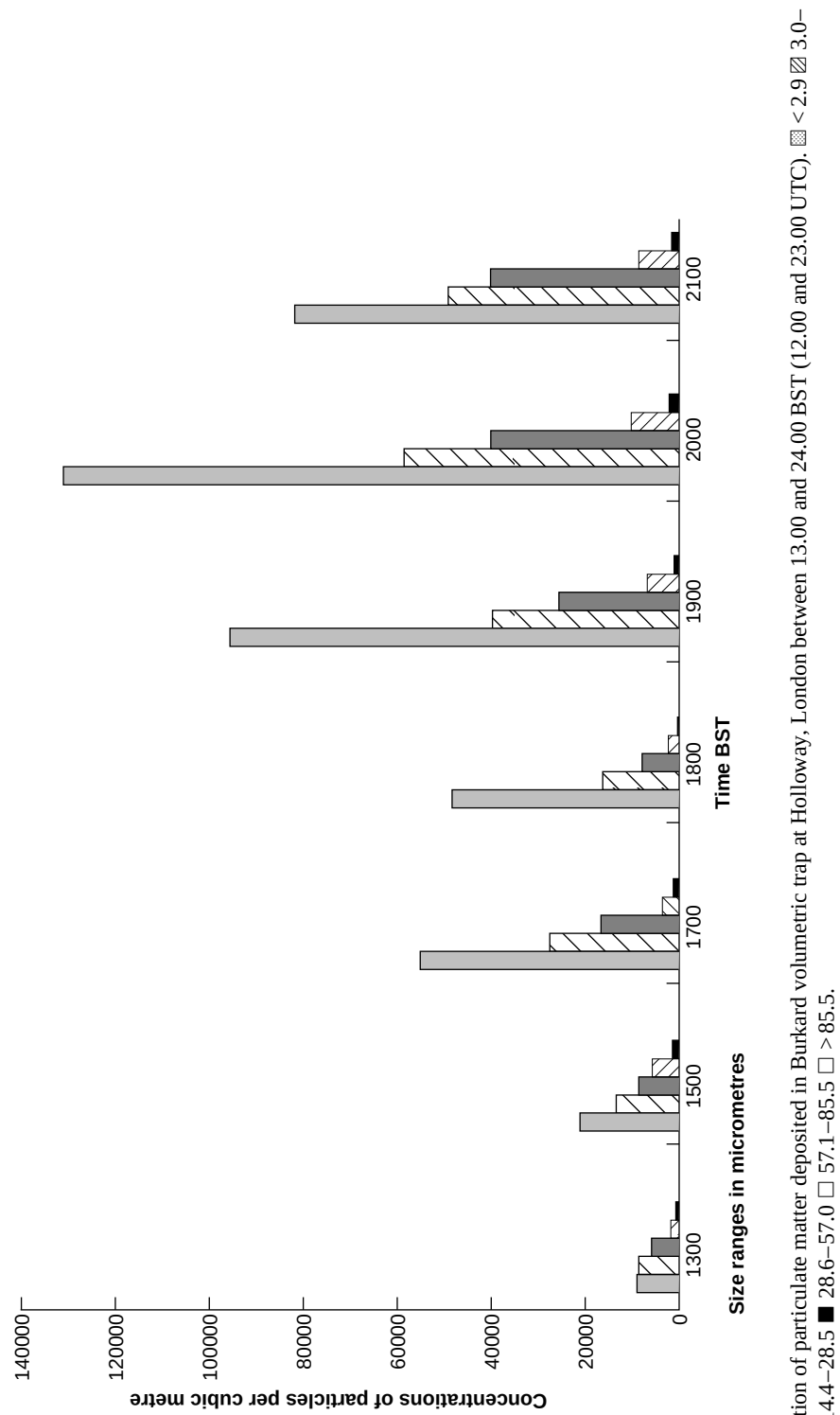


**Fig. 7.** Size distribution of particulate matter deposited in Burkard volumetric trap at Holloway, London between 13.00 and 24.00 BST (12.00 and 23.00 UTC). ■ < 2.9 µm, ▨ 3.0–5.7 µm, ▩ 5.8–14.3 µm, ▤ 14.4–28.5 µm, ▥ 28.6–57.0 µm, ▦ 57.1–85.5 µm, ▧ > 85.5 µm.

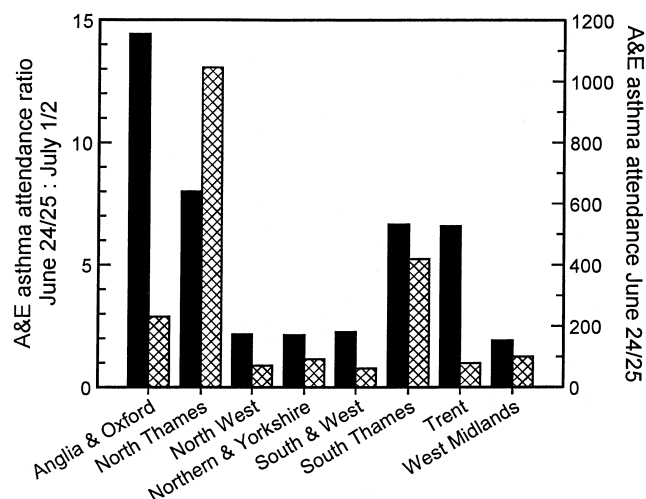


Fig. 8. Number of A & E attendances with asthma (▨) on 24/25 June and ratio of asthma attendances compared with 1/2 July (■).

message going out in his name on the Department of Health EPINET system to seek information from District Health Authorities (DHAs) in England to help define the scale and extent of the epidemic. The information requested included:

'The numbers attending Accident and Emergency Departments on the evening/night of 24/25 June (18.00 h–06.00h, or the nearest available 12-h period), compared with the following Friday/Saturday (1/2 July); cases reported should be restricted to those suffering from asthma/wheeze which responded to treatment with bronchodilators. Both paediatric and adult cases should be reported.'

Similar information in respect of admissions and any deaths was also sought, along with any information on difficulties in the provision of services, and such GP data as were available. The 1/2 July control period was selected

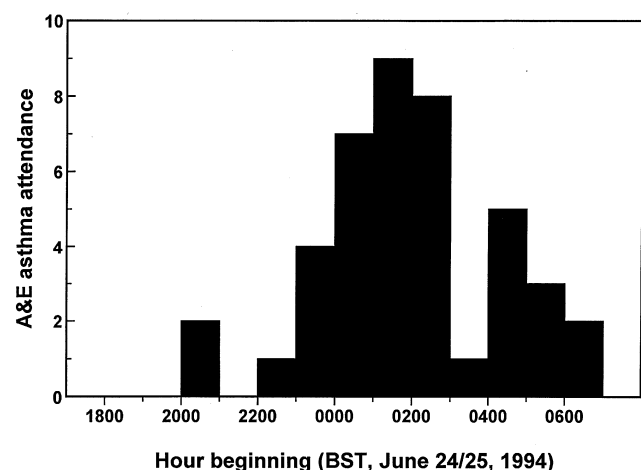


Fig. 9. Time course of attendances with asthma or wheeze at the A&E department, Addenbrooke's Hospital, Cambridge, 24/25 June 1994 [14].

as an equivalent time period for comparison purposes without placing too great a burden on staff obtaining the data.

Responses were received from 95% of the DHAs. Not all were able to supply all the information requested. Based on data from those hospital A&E departments which made returns for both nights, it was apparent that four of the eight Regional Health Authorities (RHAs) in England had experienced a more than sixfold greater attendance with asthma/wheeze on the epidemic night than on the following week. These RHAs were: Anglia and Oxford, North Thames, South Thames, and Trent (Fig. 8). There was a twofold increase in the other regions. In terms of the total reported numbers attending on the night of June 24/25, North Thames and South Thames were the most severely affected of the RHAs (Fig. 8). Data on the time-course of attendance had not been requested but were supplied by some hospitals. At Addenbrooke's Hospital, Cambridge, the peak occurred at 0100–0200 h 25 June (Fig. 9) [14].

Figures for admissions were less informative. Only one associated death was reported from the main affected RHAs. Eleven DHAs, predominantly in the North Thames region, reported that there had been difficulties in the provision of services at A&E departments. In most instances these involved the necessity to restock with bronchodilators. These, and staff shortages, contributed to unavoidably long waiting times. In all cases the difficulties were resolved locally.

#### Immunological aspects

Blood was available from 16 subjects who had attended London A&E departments with respiratory symptoms following the thunderstorm.

In order to see if these patients were hypersensitized to common aeroallergens their specific IgE antibodies were measured by CAP (Pharmacia) to a grass pollen mix ( $g \times 1$ ) and a mould mix ( $m \times 1$ ). Twelve of the patients had very high levels of IgE to pollen (classes 4–6), three of whom had a low or moderate IgE level to mould (classes 1–2). Only two of the patients had a negative IgE response to pollen whereas 13 were negative to mould.

#### Discussion

The sequence of events seems to be consistent with two processes. The first is that during the day there was a steady accumulation of airborne particulates measured by pollen monitors. This was very marked in London where the particulate matter was exceptionally abundant especially in the respirable fraction ( $<10 \mu m$ ) from about 09.00 BST (08.00 UTC) increasing to a peak at about 19.00–20.00 BST (18.00–19.00 UTC) (Fig. 7). This steady accumulation of particulates was probably the result of convergence and atmospheric stability.

The second process was shown by a sharp increase in the concentrations of airborne particulate matter for about an hour just before the storm. This is likely to have resulted from the increased gusting of wind lifting particles from surfaces. This gusting coincided with the peak in grass pollen concentrations which usually occurs in the early evening [15]. As the rain began to fall the amounts of larger particles ( $>10\mu\text{m}$ ) decreased but small particles remained suspended for some time after the storm began.

The high frequency of specific IgE antibody to grass pollen in the immunology pilot study, together with the history of hay fever in many affected patients, suggests that the patients may have been responding to grass pollen allergen, as Suphioglu *et al.* (1992) suggested was the case in the Melbourne epidemic [8]. These findings are being examined in a case-control study comparing epidemic asthma cases with asthmatic patients unaffected by the storm. The pilot study was set in London and it is possible that other pollens, or fungal spores, were the responsible aeroallergens in other parts of the country.

Summarizing, it is likely that large amounts of grass pollen were released in the day or two before the storm, settling on ground and vegetation. The strong gusting winds immediately before the storm lifted the pollen into the air to make it more readily inhalable. It is possible that the gusts led to further release of attached pollen. The rain associated with the storm then disrupted the pollen grains into a much larger number of smaller allergenic starch particles. These were readily inhaled. People with high levels of specific IgE to grass pollen were vulnerable to this 'allergen challenge', particularly those not protected by medication.

Although considerable electrical activity was evident during the storm (Fig. 3) early indications suggest that lightning occurred somewhat behind the surface gust front. It remains to be explored what, if any, was the role of electrical charge; this question was also asked after the Birmingham outbreak [1]. Work continues to examine the details of the meteorological mechanisms.

Air pollution may play a role in thunderstorm-related asthma by lowering the threshold for triggering an asthma attack [16–18] or by increasing the severity of the attack. Air pollution levels in the London area were not unusually high on 24 June and did not exceed air quality 1-h guidelines (World Health Organization 1987 [19]) but did peak during the calm, still period before the thunderstorm. Further work is needed to clarify the role of air pollution. It is of interest that a recent air pollution episode was not associated with an epidemic of asthma [20].

Whatever the mechanisms, this was a substantial epidemic of asthma with sufficient impact to trigger an enquiry to the National Poisons Unit. The Chief Medical Officer's survey shows that there was an appreciable, though fortunately not overwhelming, burden on the emergency services.

All severe asthma attacks are potentially life-threatening so the epidemic will be studied further on its own merits. A historical review of daily fluctuations in hospital admissions for asthma in England is in press. The study aimed to identify in a systematic manner both local and widespread epidemics of acute asthma and to explore their relation to weather conditions and airborne pollen levels. It also examined the feasibility of forecasting the occurrence of asthma epidemics [20a].

The frequency of acute attacks of asthma is variable within individuals, over time, and between populations. Knowledge of the causal factors for this epidemic, and the way they interact, may be important in understanding the aetiology of acute asthma.

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