

There was a small fall in the rate due to immaturity, with an improvement in the rate in the birthweight groups of 1500 g or less, and in 1983 there were no deaths of babies weighing more than 1500 g ascribed to this cause.

When examined within birthweight groups, with all causes aggregated, the PMR improved in each group from 1981 to 1983.

Antenatal care, obstetric management, resuscitation, and care in special-care baby units improved over the 3-year period. However, the number of larger (over 2000g) normally formed babies (of whom there were 7 in 1983) who became asphyxiated and died during labour or during the first week of life is still a cause for concern.

DISCUSSION

The PMR was consistently higher in West Glamorgan than in all Wales and in England and Wales until 1982, when, for the first time, it fell below those averages. It remained below those averages in 1983 and 1984.

As in all parts of the UK the rate had been falling in West Glamorgan before the intervention of the confidential

inquiry, but the percentage fall over the 3-year survey period was greater than that in any previous 3-year period.

The trend in the refined PMRs indicated an improvement in the quality of the health care, as did the fall in the number of avoidable adverse factors relating to the health services (unpublished data).

This form of audit contributes to an improvement in maternity and neonatal services and a consequent reduction in perinatal mortality.

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REFERENCES

1. Elbourne D, Mutch L. Do locally-based enquiries into perinatal mortality reduce the risk of perinatal death? In: Smith A, ed. Recent advances in community medicine, vol 3. Edinburgh: Churchill Livingstone, 1984: 221-29.
2. Thomas J, Collins M, Edwards J, et al. Report of the West Glamorgan Perinatal Mortality Survey. Swansea: West Glamorgan Health Authority, 1984.
3. Mallett R, Knox EG. Standardised perinatal mortality ratios: techniques, utility and interpretation. *Commun Med* 1979; 1: 6-13.
4. Wigglesworth JS. Monitoring perinatal mortality. *Lancet* 1980; ii: 684-86.
5. Macfarlane A, Chalmers I, Adelstein AM. The role of standardisation in the interpretation of perinatal mortality rates. *Health Trends* 1980; 12: 45-50.
6. Clarke M. Perinatal audit; a tried and tested epidemiological method. *Commun Med* 1982; 4: 104-07.

Environmental Health

ASTHMA OUTBREAK DURING A THUNDERSTORM

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Summary An outbreak of acute asthma occurred in Birmingham and the surrounding area on July 6 and 7, 1983. In most patients symptoms began at the time of sudden climatic changes associated with a thunderstorm. Air pollution was not a factor. The large and sudden increase in numbers of airborne fungal spores, especially *Didymella exitialis* and *Sporobolomyces*, around the time of the outbreak suggests that they may have been partly contributory, although a direct causal effect has not yet been established.

INTRODUCTION

THERE are several published reports of large epidemics of acute respiratory disease, including asthma, associated with severe air pollution and exceptional weather conditions. The best documented examples are those in the Meuse valley, Belgium (1930),¹ the Donora valley, USA (1948),² and London during a smog (1952).^{3,4} Prevalence of asthma is increased over a wide area near Yokohama and Tokyo, Japan where heavy industry is concentrated and where levels of air pollution are high.⁵ More localised epidemics of asthma have also been caused by air pollution; in separate incidents in the USA,⁶ Brazil,⁷ and South Africa,⁸ local residents were affected by discharge of castor-bean dust into the atmosphere from a nearby oil mill. Increases in hospital admission rates for asthmatic patients in The Netherlands⁹ and New York¹⁰ have been attributed to sudden falls in temperature, while peaks in childhood asthma admissions to hospitals in Philadelphia tend to occur on clear days with high barometric

pressure.¹¹ More puzzling are the reasons for recurrent asthma epidemics reported from New Orleans since the early 1950s; various factors, including air pollution,¹² climatic changes,¹³ and aeroallergens such as pollen and fungal spores,¹⁴ have been implicated. Other reports of asthma outbreaks have come from Barcelona, Spain,¹⁵ and Melbourne, Australia;¹⁶ in Barcelona, a connection with air pollution, (particularly oxides of nitrogen) was suggested, whereas the Melbourne outbreak was associated with a thunderstorm.

In the UK there is a seasonal variation in the incidence of asthma attacks, especially in children, with peaks in the early summer associated with high pollen counts, and an unexplained late-summer peak which may be related to airborne fungal spores.¹⁷ Localised epidemics of respiratory disease in the UK have ceased since air pollution over major industrial cities was brought under control in the early 1960s.

We previously reported that an abnormally high number of patients attended our hospital with acute asthma at the time of a severe thunderstorm on July 6 and 7, 1983.¹⁸ At the time we thought unusual climatic conditions were responsible, but Morrow Brown and Jackson showed that fungal spore counts in Derby, 45 km north-east of Birmingham, increased at the time of the outbreak, and suggested that a similar rise in fungal spores may have induced the onset of asthma in our patients.¹⁹ Harries and his colleagues further suggested that the particular spores involved might be those of the saprophytic fungus *Didymella exitialis*.^{20,21}

We now present more complete data on the extent of the asthma outbreak in Birmingham and on the environmental factors that may have been responsible.

METHODS

The daily figure for patients presenting in Birmingham with acute asthma and other acute respiratory disorders was enumerated for June and July, 1983. Information was obtained from eight hospitals—East Birmingham, Selly Oak, Dudley Road, Birmingham Children's Hospital, Good Hope, Birmingham General, Queen Elizabeth, and Solihull. Names of patients presenting with symptoms of respiratory disease were taken from casualty department registers. Emergency department record cards

or case-notes of these patients were then examined in order to obtain more detailed information. A diagnosis of asthma was accepted if the patient gave a history of wheeze and breathlessness of acute onset, if auscultation of the chest revealed diffuse expiratory wheezes, and if there was no history of chronic bronchitis/emphysema. For patients who presented with asthma on July 6 and 7, their time of hospital registration was noted.

Information on asthma admissions to the eight hospitals listed above, and to 13 hospitals in the West Midlands serving Coventry, Warwick, Stratford, Bromsgrove, Kidderminster, Stourbridge, Dudley, Sandwell, and Walsall was obtained from the Hospital Activity Analysis listings for 1980-84. Attack rates for acute asthma in general practice were obtained from the weekly returns system of the Royal College of General Practitioners Research Unit. The number of patients attending hospital for acute asthma in Derby was obtained for June and July, 1983.

Meteorological data for 1983 were obtained from the weather station at Birmingham University (sited 4 km south-west of the city centre). Additional information on rainfall was provided by the Severn Trent Water Authority; this included recordings from 20 rain-gauges sited in and around Birmingham, and from a radar installation in Shropshire which monitors rainfall over the West Midlands.

Daily figures for levels of smoke and sulphur dioxide for June and July were obtained from nine sampling sites in Birmingham (Birmingham Environmental Health Department).

The nearest sampling sites to Birmingham for measuring pollen or fungal spores are at Dudley, 13 km to the west, and at Leamington Spa, 20 km to the south-east. At both sites a volumetric slit sampler (a modified Hirst design)²² has been in operation since 1983. The sampler at Dudley is positioned 12 m above ground level and that at Leamington 15 m above ground in the centre of the town. At Dudley, pollen counts only were available for 1983. At Leamington, mean daily pollen and fungal spore concentrations were measured for June and July, 1983, and hourly concentrations were measured for July 6 and 7, 1983. Pollen and spore counts over Derby were also obtained for June and July.

RESULTS

Birmingham Data

Patients

The number of patients with acute asthma attending daily at the eight Birmingham hospitals is shown in fig 1. Attendances for most of June and July ranged between 2 and 20 per day; on July 6 and 7, however, there were 36 and 70 attendances, respectively. Fig 2 shows the number of patients with acute asthma attending hourly according to time of registration. Full clinical details of a representative sample will be published elsewhere, but most had had hayfever in the weeks preceding the outbreak, gave a history of summer asthma, and complained of symptoms from 2000 h onwards. From 2130 h the number of attendances increased sharply and this increase continued until mid-morning on July 7. Over the 48-hour period 106 patients (62 males, 44 females) attended; mean age was 24.7 years (range 14 months to 63 years). 32 patients were admitted. Analysis of time of attendance in relation to home address showed no special pattern.

Average daily number of admissions to the 13 other West Midlands hospitals during June and July was 6.9 (excluding patients discharged from casualty departments). Figures for July 6, 7, and 8 were 9, 29, and 20, respectively. From 1980 to 1984 there were no other significant peaks in asthma admissions at the hospitals studied.

Data on acute asthma attacks seen by general practitioners over this period had been reported as showing increased rates throughout the country;²³ re-analysis of these figures showed that the increase in asthma attacks during the week ending July 12 was predominantly in the central region (fig 3).

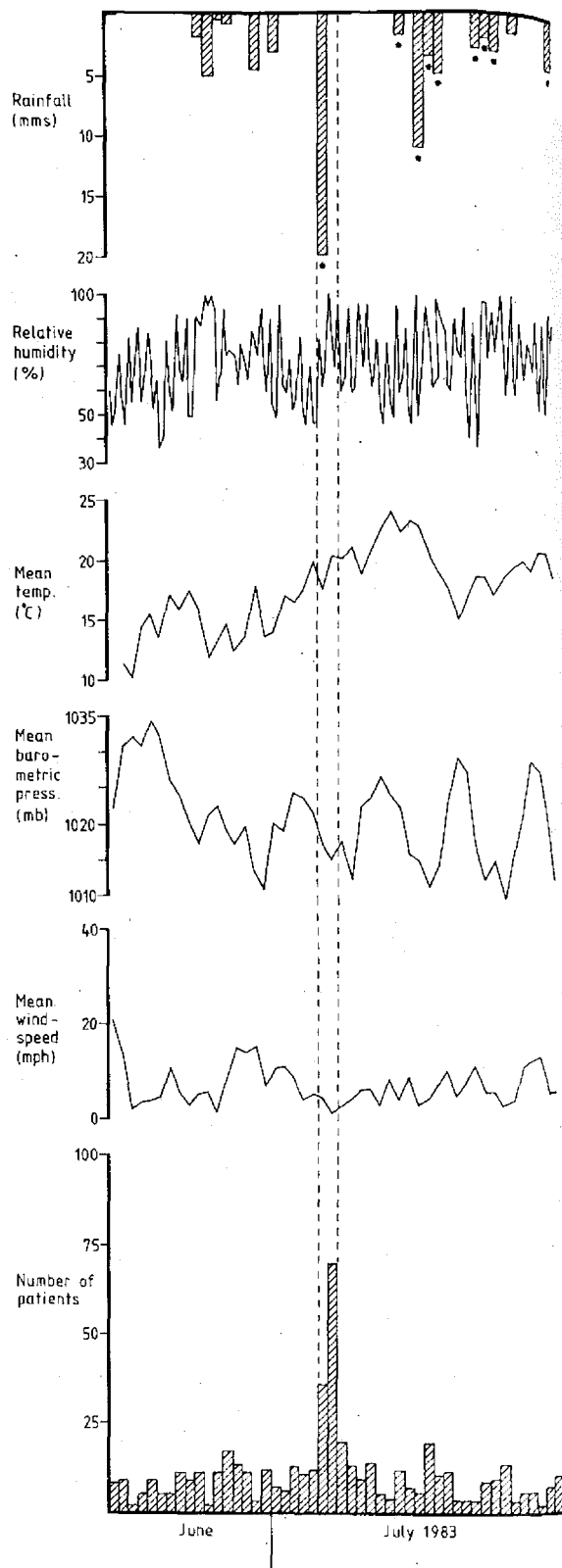


Fig 1—Climatic conditions and daily attendances for acute asthma from June 14 to July 31, 1983.

* = Thunderstorm days. Relative humidity was measured at 0600 h, 1200 h, and 1800 h. Barometric pressure was corrected to sea-level.

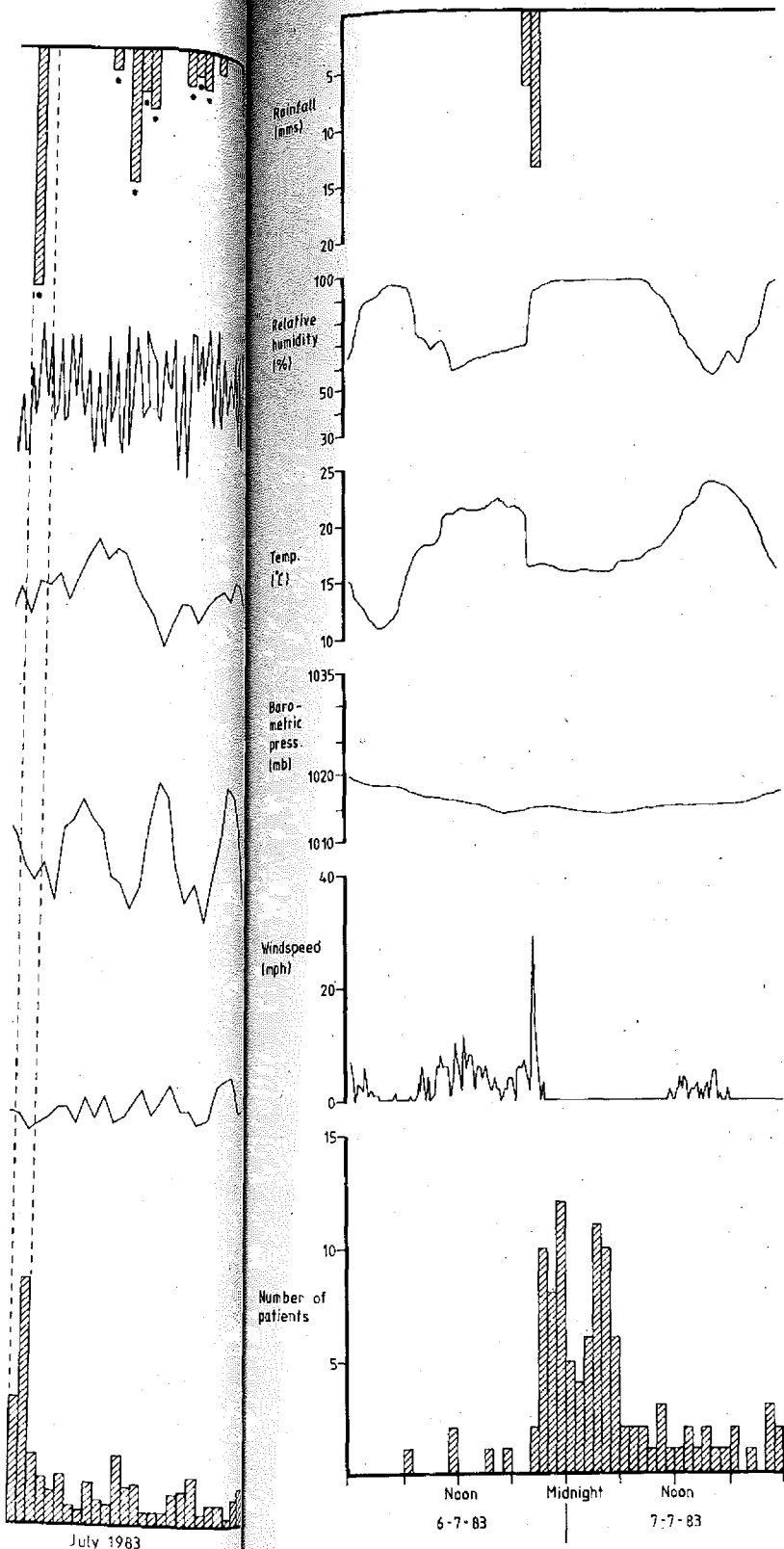


Fig 2—Hourly attendances for acute asthma, July 6-7.

Casualty data for the eight Birmingham hospitals showed that there was no increase in the incidence of other respiratory disorders at the time of the outbreak.

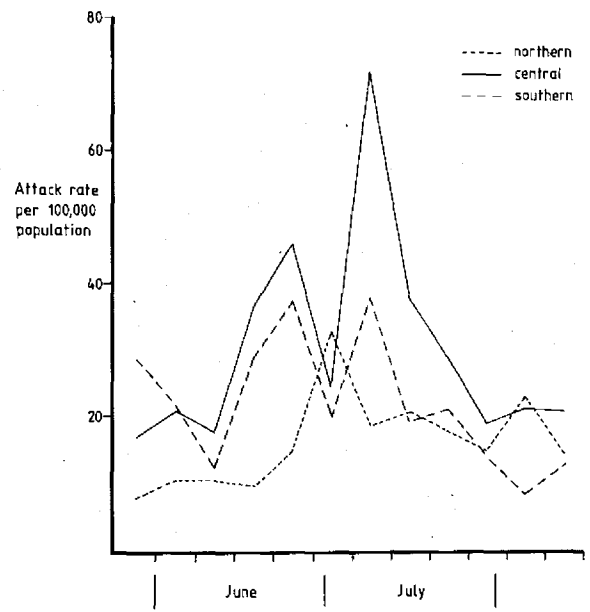


Fig 3—Weekly attack rate per 100 000 population for acute asthma seen in general practice by UK region between May and August, 1983.

Weather

At the beginning of July, 1983, the UK was covered by a ridge of high pressure from the Azores which brought sunny weather to most places; from July 2 to July 5 it was cloudless and dry over the Midlands. From July 1 to July 6 there was a steady rise in temperature, and a progressive fall in mean wind-speed. Relative humidity remained especially low during July 2-4 (fig 1). A temperature inversion was detected by radar over the West Midlands from 1900 h to 2400 h on July 5, and again from 0600 h to 1000 h on July 6. The origin of the storm was a thundery depression of 1015 millibars which developed over south-west France on July 4. The first rain to arrive in Birmingham was a trace recorded at Birmingham Airport at 1950 h; mean rainfall in Birmingham for July 6 from 20 rain-gauges was 15.6 mm (range 0.5-27.9).

Fig 2 shows weather data recorded at Birmingham University. As the storm approached there was a steady fall in barometric pressure. A squall of 35.5 mph was recorded at approximately 2030 on July 6. 19.8 mm of rain fell between 2000 h and 2200 h, most during a spell of heavy rain between 2045 h and 2130 h, and this was associated with a rapid rise in relative humidity from 60% to 100%. After the heavy rain, humidity remained high overnight, and there was a prolonged period of calm which persisted until mid-morning on July 7.

Air Pollution

Air pollution was low. Highest values for smoke and sulphur dioxide levels from July 1 to July 7 were $51 \mu\text{g m}^{-3}$ and $111 \mu\text{g m}^{-3}$, respectively.

Aeroallergens

Mean daily spore concentrations at eight sites in the Midlands have been shown to be broadly similar,²⁴ so we believe that the figures from Leamington Spa were representative of spore counts over Birmingham. However,

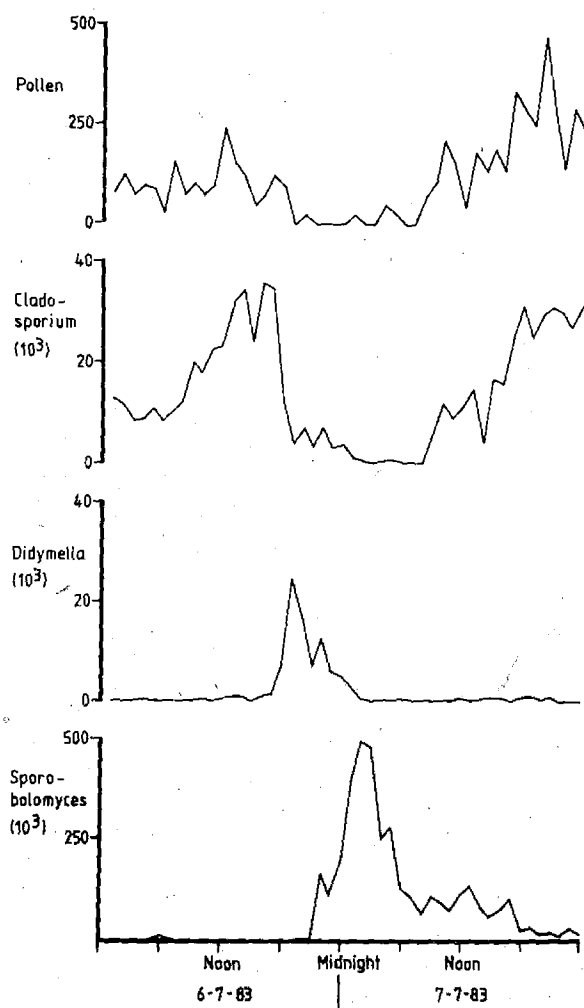


Fig 4—Hourly counts (m^{-3} of air) for total pollen and fungal spores. Counts measured at Leamington Spa on July 6 and 7, 1983.

because the storm moved in a north-westerly direction, reaching Leamington before Birmingham, changes in the air spore concentration over Birmingham would have occurred 1–2 h later than the changes recorded at Leamington.

Fig 4 shows the levels of pollen, and of known allergenic fungal spores which were prevalent before, during, and after the storm. During the morning and afternoon of July 6, spores of *Cladosporium* were most common, peaking in the late afternoon; similar concentrations of *Cladosporium* were seen in the preceding two days, and were common throughout July, 1983. Beginning at 1700 h there was a fall in *Cladosporium* spore count and a sudden rise in numbers of *Didymella exitialis* ascospores, which peaked at about 1900 h. During the period of high relative humidity after the rain, there were high concentrations of *Sporobolomyces* basidiospores. By mid-morning on July 7, *Cladosporium* was again predominant. High concentrations of *Didymella* were also noted on July 2, 23, 24, and 30, and high concentrations of *Sporobolomyces* on June 27 and 28, and July 23 and 24, although peak hourly concentrations of these spores were higher on July 6 and 7.

Total pollen counts measured at Dudley and Leamington Spa from July 2 to July 5 were high, with mean daily levels of

100–300 m^{-3} . Counts remained high during July 6, declined later in the day with the onset of rain, and increased again by the afternoon of July 7.

Derby Data

In Derby there was no increase in asthma attendances at the time of the asthma Birmingham outbreak. Moreover, the increase in fungal spores noted by Morrow Brown and Jackson¹⁹ at the time of the Birmingham outbreak was relatively small compared with the much greater increases seen over Derby later in the month. In particular, a large peak in concentrations of *Sporobolomyces*, and to a lesser extent *Didymella*, occurred at the time of a thunderstorm on July 24, and this was associated with a small rise in the number of acute asthma attendances at Derby, from an average of 0.8/day, to 3 on July 24 and 4 on July 25.

DISCUSSION

In most patients asthmatic symptoms began at the time of sudden changes in the weather associated with a thunderstorm. Unlike many previous asthma outbreaks, air pollution was not the cause and there were no deposits of "Saharan sand", as previously suggested.²⁵ Another suggestion was that airborne fungal spores were responsible,^{19–21} and we have confirmed that there was a large and sudden increase in the numbers of airborne spores, especially *Didymella exitialis* and *Sporobolomyces*, around the time of the outbreak.

Allergy to fungal spores is well recognised as a cause of summer asthma and both *Didymella exitialis*^{20,26} and *Sporobolomyces*²⁷ are known to cause respiratory allergy. Recurrent asthma epidemics in New Orleans may be related to increased levels of ragweed pollen and small basidiospores,¹⁴ although a causal link has not been established.²⁸ Unfortunately, direct comparison of spore counts in New Orleans and the UK is difficult because of differences in spore traps and methods of spore categorisation and quantification. Prevalence of asthma is increased in the coastal region of Queensland, north-eastern Australia, and high concentrations of small basidiospores and ascospores have been recorded at the time of peaks in frequency of asthma attacks.^{29,30} In Perth, south-western Australia, variations in climate and in total fungal spore levels were thought to be the two most important factors causing an increased incidence of asthma.³¹

Fungal spore release and dispersion is profoundly influenced by climatic changes, especially by rainfall; dry-air spores (eg, *Cladosporium* and *Alternaria*), can be distinguished from damp-air spores (eg, *Didymella* and *Sporobolomyces*) which are released during and after rainfall.³² The principal effect of rain is to remove particulate matter from the air ("rain-scrubbing") and this applies to fungal spores, including those that are released during rainfall. The number of airborne spores present during rainfall therefore represents the excess of those released over those removed; if rain is sustained then there is likely to be an overall reduction in damp-air spores. After abrupt periods of heavy rain, as often occurs during thunderstorms, the effect of spore release probably outweighs that of "rain-scrubbing". Hirst³³ showed that high concentrations of single-septate hyaline ascospores (probably *Didymella*) and small basidiospores, later identified as *Sporobolomyces*, were liberated over farmland after a thunderstorm and heavy rain and these findings have been confirmed.^{20,26,34–36} In Hirst's study a preceding dry spell was probably important—ie,

spores had become ripe for release but could not be liberated because of the absence of rain. The hot dry spell of four days before the Birmingham thunderstorm may have had a similar effect.

Climatic changes associated with thunderstorms are often abrupt, with sudden heavy rainfall and rapid changes in temperature, humidity, and windspeed. Rapid cooling of the airways is responsible for airway narrowing that occurs in some asthmatics in response to exercise, although this effect is attenuated if the inspired air is humidified.³⁷ It is possible, therefore, that rapid cooling of the ambient air during sudden, heavy rainfall could result in airway narrowing, but this effect might be offset by the concomitant rise in humidity.

During the growth of a thunderstorm, intense convective upcurrents of air occur within dense cumulus clouds. As the storm develops, up-currents coexist with powerful down-draughts caused by the drag exerted by numerous falling raindrops.³⁸ Down-draughts usually precede the main cloud mass, as in our case. The turbulence can draw particulate matter upwards and carry it some distance before depositing it. Thus fungal spores could have been carried in this manner, being deposited over urban Birmingham at respirable levels during the calm period which followed the storm.

A thundercloud is positively charged at the top and predominantly negatively charged at the base, causing a positive charge directly beneath at ground level.³⁸ Positive discharge from objects on the ground then results in upward movement of positive ions, an effect which is compounded by strokes of lightning. There is some evidence, that positive ions may provoke an asthmatic attack,³⁹ therefore the ionic changes during thunderstorms might in theory contribute to worsening asthma.

Temperature inversions were believed to have played a major part in the build-up of air pollutants which resulted in large outbreaks of respiratory disease in Belgium¹ and the USA,² and may have contributed to an acute asthma outbreak in Brisbane, Australia.⁴⁰ Usually, air temperature decreases with increasing altitude, but on warm cloudless days increased heat loss by radiation cools the ground and the air immediately above it. This results in a layer of cool air at ground level with warm air above it—a temperature inversion—which may occur anywhere from near ground level up to high altitude. An inversion blocks convection currents and traps particulate matter in the layers beneath it. Vertical distribution of fungal spores in the atmosphere is affected by temperature inversion;⁴¹ inversions were present over the Midlands in the 36 h preceding the thunderstorm, and, although these had cleared by mid-morning on July 6, they may have influenced spore distribution just prior to the asthma outbreak.

Many of the patients had a history of hayfever and seasonal asthma. Cockcroft⁴² has emphasised how broncho-provocation caused by allergy to pollen can cause an increase in non-specific bronchial reactivity; patients with seasonal asthma might therefore develop airway narrowing in response to non-allergic trigger factors such as cold air or inhaled particulate matter. During this outbreak, such an increase in bronchial reactivity may have occurred as the result of pollen exposure in the preceding weeks and/or climatic factors. Under these circumstances, any trigger (eg, fungal spores, cold air, excitement) could readily result in airway narrowing. It is possible that our patients, although not specifically allergic to fungal spores, may have had airway narrowing because of the sheer particle load inhaled.

During previous outbreaks of respiratory disease it has

often been difficult to relate environmental changes to the onset of symptoms because of the inevitable time lag and the various factors which determine when patients decide to seek treatment.⁴³ In addition, changes in environmental conditions have usually been examined on a daily or weekly basis; the immediate asthmatic response to a stimulus may begin within minutes,⁴⁴ making it important to look at changes on a narrower time scale—eg, at least hourly—as we have done.

It is impossible to be certain about the course of events resulting in this asthma outbreak. Our findings give some support to the suggestion that a sudden rise in *Didymella* spores^{20,21} may have contributed to the outbreak, at least in part, although the effects of other damp-air spores, especially *Sporobolomyces*, must also be considered. Why have similar episodes not occurred either in the Midlands or elsewhere in the UK? Thunderstorms often occur during the British summer, and the associated changes in air spores that we observed are not uncommon. We believe there must have been a unique combination of environmental factors at the time of the outbreak, or there may have been other factors which we have been unable to identify.

We thank Miss Felicity Jackson of the Midlands Asthma and Allergy Research Association, Derby, for providing spore data from Derby; Dr J. Hadfield for supplying details of hospital attendances for acute asthma in Derby; Dr Ursula Ullitt, Miss Felicity Jackson, Dr J. Lacey, Dr R. R. Davies, Dr R. Mullins, and Dr H. Morrow Brown for advice on measurement and identification of fungal spores; Mr J. A. Pickard, Warwick District Council Environmental Health Department, and Mr G. Brownword, Dudley Metropolitan Borough Environmental Health Department, for giving us access to pollen and fungal spore counts; and Mr J. Kings, Geography Department, Birmingham University, and Mr R. C. Goodhew, Severn Trent Water Authority, for supplying and interpreting weather data. We are grateful to the Royal College of General Practitioners Research Unit for the use of their data, and Mr R. S. Appleby, City of Birmingham Environmental Health Department, for supplying air pollution figures. We also thank the doctors in Birmingham for permitting us to examine case records of their patients.

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REFERENCES

- Batta G, Firket J, LeClerc E. Les problèmes de pollution de l'atmosphère. Paris: Masson et Cie, 1933.
- Shrank HH, Heimann, Clayton GD, Gafaer WM, Wexler H. Air pollution in Donora, epidemiology of the unusual smog episode of October, 1948: Preliminary report. Public Health Bulletin No 306, Washington, DC, 1949.
- Logan WPD. Mortality in the London fog incident. *Lancet* 1953; i: 336-38.
- Ministry of Health. Morbidity and mortality during the London fog. Rep Pub Hlth Med Subj, No 95. London: HM Stationery Office, 1954.
- Phelps HW, Koike S. Tokyo-Yokohama asthma. *Am Rev Resp Dis* 1962; 86: 55-63.
- Figley KD, Elrod RH. Endemic asthma due to castor bean dust. *JAMA* 1928; 90: 79-82.
- Mendes E, Cintra AU. Collective asthma, simulating an epidemic, provoked by castor bean dust. *J Allergy* 1954; 25: 253-59.
- Ordman D. Outbreak of bronchial asthma in South Africa affecting more than 200 persons caused by castor bean dust from an oil processing factory. *Int Arch Allergy* 1955; 7: 10-24.
- Tromp S. Biometeorological analysis of the frequency and degree of asthma attacks in the West part of the Netherlands (periods 1953-1959). In: Biometeorology. Proceedings of the Second Biometeorological Congress. London: Pergamon, 1982: 477-83.
- Greenberg L, Field F, Reed JI, Erhardt CL. Asthma and temperature change. *Arch Environ Health* 1964; 8: 642-47.
- Girsh LH, Shubin E, Dick C, Schulander FA. A study on the epidemiology of asthma in children in Philadelphia. *J Allergy* 1967; 39: 347-57.
- Kenline PA. October 1963 New Orleans asthma study. *Arch Environ Health* 1966; 12: 295-304.
- Salvaggio J, Hasselblad V, Seabury J, Heiderscheit LT. Relationship of climatologic and seasonal factors to outbreaks. *J Allergy* 1970; 45: 257-65.
- Salvaggio J, Seabury J, Schoenhardt EA, New Orleans Asthma V. Relationship between Charity Hospital asthma admission rates, semi-quantitative pollen and fungal spore counts, and aerometric sampling data. *J Allergy Clin Immunol* 1971; 48: 96-114.
- Ussetti P, Roca J, Augusti AGN, Montserrat JM, Rodriguez-Roisin R, Agusti-Vidal A. Another asthma outbreak in Barcelona: Role of oxides of nitrogen. *Lancet* 1984; i: 156.
- Egan P. Weather or not. *Med J Aust* 1985; 142: 330.

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Round the World

From our Correspondents

United States

INSURANCE TROUBLES: THE DOCTORS ARE NOT ALONE

VIRTUALLY all the large insurance companies having withdrawn from the field of medical malpractice, it seems it is now the large corporations, businesses, and individuals who must go through the economic wringer to which physicians have been subjected. Reactions are likely to be similar. The enormous costs associated with agents that cause injury have been paralleled by similar large verdicts for personal injuries due to non-medical agencies. So large have been awards against towns, municipalities, and other organisations that many are now finding it impossible to obtain cover save on terms beyond their financial means, though the Supreme Court in a recent judgment has given some relief. Moreover, the wooden buildings, often old and decrepit, in our cities and towns have been a favourite target for arsonists, sometimes subsidised arsonists—at immense cost to the insurers. Actions for personal injury and product liability have cost dearly, premiums will have to go up, and damage liability must be more precisely defined, timed, and calculated.

Thus bigger and bigger deductibles, more restrictive and carefully drawn policies, and limits on multiple claims are all foreseen and are causing concern to industry and public alike. With memories of the Bhopal disaster, there is a run for cover. Suppose it had happened here, as well it might, considering the many road and rail accidents involving dangerous chemicals, not to mention factory leaks.

Meanwhile, in New York State, the medical "go slow" and surgical curfew persist and have got the close attention of the legislature. Various proposals are being promulgated, but many have about them an air of unreality. In medical circles, there seems no willingness to admit to the large number of negligent and incompetent physicians who need to be disciplined. But one measure may affect the whole insurance scene: to make the losing side pay the costs of the case; and indeed the limits of defence costs would be laid down in policies. There then might be a far greater willingness to settle cases.

MURDERERS IN THE BOARDROOM

NOT the title of a detective novel but a note on a legal decision that has sent a shudder through the boardrooms of this country. White-collar crime is particularly prevalent just now, to judge by the newspapers; indeed nine of the ten largest suppliers of the Pentagon are under investigation for criminal or near-criminal activities.

Peculation, embezzlement, and fraud seem widespread; and activities are now being treated much more severely by the courts. In the past it was evident that prison sentences for such crimes were modest; and detention, if ordered, was passed in more pleasant surroundings than those to which persons convicted of blue-collar crimes were sentenced.

A recent decision concerns the directors and staff of a factory where silver was recovered from X-ray film by a process involving cyanide. Evidence was produced that ventilation control was inadequate, workers were exposed to cyanide fumes, and justified complaints by the exposed workers were ignored. The justification for other complaints was evident when one worker collapsed and died, and the evidence was clear that he died of cyanide poisoning after inhalation of fumes at his workplace. The time criminal charges were laid against the company and its executives. They were found guilty of murder. Of course, there will be an appeal, but the mere fact of the conviction has terrified other boardrooms, as well it might. Those responsible for spreading toxic fumes in the workplace, not to say the community at large, or those responsible for accidents in transportation of dangerous substances, have been given an awful warning.

MORE ON PRODUCT LIABILITY

SINCE a Federal judge in Cincinnati managed to consolidate about 1100 of more than 1500 'Bendectin' ('Debendox') suits against Merrell-Dow and a jury then decided that bendectin did not cause birth defects, Merrell-Dow have received calls from obstetricians across the country asking if bendectin will be put back on the market. A company spokesman said they did not intend to do that, adding that the company had redirected its energy away from obstetrics, an area with a high risk of liability.

Vaccine manufacturers are also under pressure by litigation arising from adverse reactions. Insurance costs have risen to such an extent that Wyeth Laboratories have withdrawn from the market for diphtheria, pertussis, and tetanus vaccine, leaving Lederle Laboratories and Squibb-Connaught as the sole marketers of the product in the United States.

The Senate is studying a Bill on product liability which would impose standards for four types of product liability: construction defects, breach of express warranty, design defects, and lack of warning. The last two types of liability would be applicable to the drug industry; and in such situations the conduct of the manufacturer would be taken into account. An amendment to the Bill proposes no-fault compensation as an alternative to taking a manufacturer to court. Under this system, a claimant can either sue the manufacturer or file a claim with the manufacturer: that would limit consequent economic losses. This amendment is regarded by some drug manufacturers as a trade-off. Since claimants would get money without proving whether the product was at fault, the product liability would be limited and there would be no legal costs. An essentially similar Bill had no time to make it through the 98th Congress.

G. E. PACKE AND J. G. AYRES: REFERENCES—continued

17. Khoi A, Burn R, Evans N, Lenney C, Lenney W. Seasonal variation and time trends in childhood asthma in England and Wales 1975-81. *Br Med J* 1984; **289**: 235-37.
18. Packe GE, Archer PSJ, Ayres JG. Asthma and the weather. *Lancet* 1983; **ii**: 281.
19. Morrow Brown H, Jackson F. Asthma and the weather. *Lancet* 1983; **ii**: 630.
20. Harries MG, Lacey J, Tee RD, Cayley GR, Newman Taylor AJ. *Didymella exitialis* and late summer asthma. *Lancet* 1985; **i**: 1063-66.
21. Editorial. Asthma and the weather. *Lancet* 1985; **i**: 1079-80.
22. Hirst JM. An automatic volumetric spore trap. *Ann Appl Biol* 1952; **39**: 257-65.
23. Birch D, Alderson MR. Asthma and the weather. *Lancet* 1983; **ii**: 630-31.
24. Morrow Brown H, Jackson AF. Aerobiological studies based in Derby II. Simultaneous pollen and spore sampling at eight sites within a 60 km radius. *Clin Allergy* 1978; **8**: 599-609.
25. Walter RE. Asthma and the weather. *Lancet* 1983; **ii**: 452.
26. Frankland AW, Gregory PH. Allergenic and agricultural implications of airborne ascospore concentrations from a fungus, *Didymella exitialis*. *Nature* 1973; **245**: 336-37.
27. Evans RG. *Sporobolomyces* as a cause of respiratory allergy. *Acta Allergol* 1965; **20**: 197-205.
28. Salvaggio J, Ankrust L. Mold-induced asthma. *J Allergy Clin Immunol* 1981; **68**: 327-46.
29. Wright GT. Research in general practice: The Queensland asthma survey. *Medical World* 1965; **102**: 130-40.
30. Derrick EH. Asthma and the Brisbane climate. *Aust N Z J Med* 1972; **3**: 235-46.
31. Hobday JD, Stewart AJ. The relationship between daily asthma attendance, weather parameters, spore count and pollen count. *Aust NZ J Med* 1973; **3**: 552-56.
32. Gregory PH. *The Microbiology of the Atmosphere*. London: Leonard Hill, 1973.
33. Hirst JM. Changes in atmospheric spore content: Diurnal periodicity and the effects of the weather. *Trans Br Mycol Soc* 1953; **36**: 375-93.
34. Last FT. The spore content of air within and above midew-infected cereal crops. *Trans Br Mycol Soc* 1955; **38**: 453-64.
35. Hamilton ED. Studies on the air spora. *Acta Allergol* 1959; **13**: 143-75.
36. Pady SM. *Sporobolomyces* in Kansas. *Mycologia* 1974; **66**: 333-38.
37. Strauss RH, McFadden ER, Ingram RH, Chandler DF, Jaeger JJ. Influence of heat and humidity on the airway obstruction induced by exercise in asthma. *J Clin Invest* 1978; **61**: 433-40.
38. Pedgley DE. *Elementary Meteorology*. London: HM Stationery Office, 1981.
39. Podleski WK. Asthma and ionisation. *Lancet* 1980; **ii**: 1035.
40. Morrison I. It happened one night. *Med J Aust* 1960; **i**: 850-52.
41. Hirst JM, Stedman OJ, Hogg WH. Long distance spore transport: Methods of measurement, vertical spore profiles and the detection of immigrant spores. *J Gen Microbiol* 1967; **48**: 329-55.
42. Cockcroft DW. Mechanism of perennial allergic asthma. *Lancet* 1983; **i**: 253-55.
43. Bennett AE. Limitations of the use of hospital statistics as an index of morbidity in environmental studies. *J Air Pollut Control Assoc* 1981; **31**: 1276-78.
44. Pepsys J, Huchcroft BJ. State of the art. Bronchial provocation tests in etiologic diagnosis and analysis of asthma. *Am Rev Resp Dis* 1975; **112**: 829-59.

Letters to t

BUS CONFERENCE HEART

would like to res development confe to prevent heart dis mens in your Point of Applications of I Development Prog e with the National shows a limited r as of consensus co knowingly, organis that the outcom ed because the pan predictably, say that States are too high ces, he claims, are with the "implicit pressure". Develo es no place for the "agreements". 7 strongly disagree. 7 on some aspe versy in treatment e for clinicians at n members are se ble research work entologists or stati ough some or perlu ar with the issue ate who have taken independent planni conference topic a e on the planning e agree on the ne panel but disag estions for the chol heart of the CDC l for every CDC mmittee that choe session. The consensus pr free to disagree ightforward ex ambiguous co: prolonged, and if uestions posed at he evidence preat alleged constraint Oliver notes t presentation of a presenting data: major views are CDC on cholest speakers, and r enter into disc at hand. Again extensive pre- months earlier The research any particula panel memb have some fa or leisure presentation Oliver wa is finalised,