and sex, joint symptoms being more common in susceptible women or in those receiving primary vaccination and being rare in children, particularly those aged over 5 years.¹³

If the measles-rubella campaign had not been conducted, large numbers of children would have had measles (with a much higher risk of long term serious effects than that potentially associated with vaccination) and rubella (with its established risk of chronic arthritis).¹⁴ To prevent illness in susceptible children, in the absence of a rapid, inexpensive, and reliable measure of immunity that could be used to screen seven million schoolchildren, it was more cost effective to vaccinate all children.

A routine second dose of measles and rubella vaccines could have been introduced, but such high coverage may not have been achieved, and it would have taken longer to reduce transmission. The incidence of temporally related adverse events is expected to be the same whether vaccine is delivered by a mass campaign or by a routine two dose schedule. Increased rubella immunity in schoolgirls now means less need for vaccination of adult women later, with a consequent reduction in risk of vaccine related joint symptoms.

The measles-rubella campaign has raised awareness both of the hazards of measles disease and of the potential for adverse events after vaccination. Success in controlling disease must be matched with improved monitoring of and research into adverse events, including in depth investigation of reports of serious adverse events, long term follow up of affected children to assess persistent morbidity, increased use of large databases such as the record linkage method proposed by Farrington *et al.*,⁴ and registries for the rare conditions that have been related temporally to vaccination. Public health professionals have a responsibility not only to develop the best

possible adverse events monitoring systems but also to ensure that perceived risks associated with vaccines do not undermine confidence in essential and effective preventive health programmes.

I thank Professor Paul Fine for helpful comments on earlier versions of this paper.

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Asthma associated with thunderstorms

Grass pollen and the fall in temperature seem to be to blame

See p 601, 604

Despite its increasing prevalence, the aetiology of asthma remains mysterious. However, a panoply of factors continues to be associated with acute episodes of asthma. In recent years, environmental epidemiologists have conducted numerous studies investigating the relations between indoor and outdoor air pollutants and asthma, particularly focusing on exposures to ozone, sulphur dioxide, nitrogen dioxide, and particulate matter of respirable diameter. Researchers have found it difficult to standardise measures of exposure, and most have used an ecological design, searching for associations between exposures measured in environments and outcomes measured in individuals (typically asthma attacks or health care utilisation). Despite the large amount of research, the evidence that air pollution causes asthma remains controversial.¹

Research into the causes of chronic diseases, such as asthma, seldom produces the immediate gratification of the epidemic curve seen in acute infectious disease, which can then be followed sleuth-like, by searches for causal agents. However, asthma has recently obliged with several epidemics linked to environmental factors. Some reports suggest a point source of an environmental causative agent, as in the Barcelona asthma "epidemic," which was linked to soya bean dust.²

More complex environmental mechanisms have been

proposed to explain "thunderstorm associated asthma," described in two papers in this issue of the *BMJ*.^{3,4} This phenomenon was recognised over a decade ago in Britain⁵ and has been reported as a recurrent seasonal phenomenon in Australian studies.⁶ These studies have consistently suggested that this phenomenon occurs in late spring, is associated with thunderstorms or a drop in temperature, and is followed by a rapid increase in attendance with asthma at hospitals or in general practice. No unusual levels of air pollutants were noted at the time of these epidemics. Packe reported that increases in airborne fungal spores (*Didymella* and *Sporobolomyces* spp) might act as a trigger.⁵ Recent studies have shown strong associations with rye grass pollen and have described a mechanism whereby allergenic starch particles may be released from rye grass, triggered by the atmospheric changes preceding and during thunderstorms.^{6,7}

Celenza *et al* (p 604) report an epidemic of asthma related to a thunderstorm that occurred in London in June 1994.³ Earlier reports of this epidemic suggested that it occurred across southern England.⁸⁻¹⁰ Some observers speculated that this epidemic might be related to air pollution,⁹ although all previous studies that have measured air pollution during thunderstorm related epidemics of asthma have found it to be normal for the time of day and month of the year.^{5,6}

Celenza *et al* have conducted a detailed analysis that

further our understanding of this phenomenon.³ By analysing asthma attendances and environmental factors during the epidemic period and the surrounding two months, they noted different patterns of association. The acute epidemic period of 48 hours showed a "classical" rise in attendances, associated with a preceding fall in temperature and substantial change in levels of airborne grass pollen, rising a few hours before the asthma attendance peaked and falling within nine hours. Asthma attendances outside the period of the epidemic were associated with a complex and different set of environmental variables, suggesting that the epidemic itself had a unique aetiology.

The hypothesis that grass pollen was to blame gains further support from the paper by Wallis *et al* (p 601).⁴ The observation that 44% of the patients had never previously had asthma strengthens the possibility that there is a group of atopic individuals who are sensitive to grass pollen^{4,6} and who react acutely to this seasonal but infrequent interaction of biological and meteorological conditions. Young adults seem to be most affected, a group who are not usually the most frequent attenders at emergency departments.

Doctors should be aware of this phenomenon, but it may not merit the "asthma weather alerts" seen on television in Melbourne, Australia, last November. Sensationalist

portrayals of asthma are a favourite of the mass media,¹¹ but fuelling the tabloid press is counterproductive to developing a wider understanding of asthma and its prevention. This group of pollen sensitive asthmatic individuals might be further investigated from a clinical perspective, with a view to developing targeted approaches to primary or secondary prevention.

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Secondary prevention of meningococcal disease

High risk contacts should be given chemoprophylaxis and preventive treatment with penicillin

Meningococcal disease puts huge pressure on local health officials. General practitioners must of course ensure that the correct diagnosis is made and that initial treatment is given, but referring the patient to hospital is not the end of the job. Meningococcal disease brings anxiety to the affected population, which may itself become a health problem. Doctors are expected to stop the disease spreading, to provide prophylaxis to those at risk of developing the disease, and to calm people's fears. Several weapons are at hand: chemoprophylaxis, protective chemotherapy, vaccination, and information.

In Britain, as well as in many other countries, chemoprophylaxis (in the form of rifampicin) is given to close contacts of the patient (household members, kissing contacts, and friends who have stayed in the patient's home for hours during the past week) to eradicate meningococci. Typically, 15% of contacts carry meningococci in their throat, but only 3-4% carry the pathogenic strain.¹ Too liberal use of chemoprophylaxis may lead to bacterial resistance. It may also impair the body's ability to develop antibodies against meningococci, which may be formed in response to the carriage of pathogenic and non-pathogenic meningococci.² Therefore, chemoprophylaxis should be restricted to those close contacts who are at highest risk of carrying the pathogenic strain.

Because of the risk of bacterial resistance, chemoprophylaxis is not recommended in Norway. Instead, household members below 15 years of age, who are at highest risk of developing secondary infection, are given penicillin for seven days to protect them from contracting the disease.³

Although chemoprophylaxis and protective treatment may prevent secondary infection, failures have been reported. Rifampicin prophylaxis eradicates meningococci from the throat, but it may not stop systemic infection.^{4,5} Protective penicillin treatment prevents systemic disease, but it may not

eradicate meningococci from the throat or protect against infection from an untreated contact.³ In our opinion, a higher degree of protection would be achieved by combining these approaches. All close contacts should be given chemoprophylaxis, while household members aged under 15 should receive both chemoprophylaxis and preventive treatment with penicillin. We cannot recommend preventive treatment with penicillin without chemoprophylaxis.

Dealing with an outbreak

Rifampicin is the drug usually used for chemoprophylaxis; taken every 12 hours for two days (600 mg in adults and children aged over 12 years, 10 mg/kg in children aged 1-12, and 5 mg/kg in children below 1 year).⁶ Alternative drugs are sulphonamides (if the strain is sensitive to sulphonamides), ceftriaxone, or one of the quinolones.⁷ More studies are needed to see if quinolones or other antibacterial drugs alone may prove effective for both chemoprophylaxis and protective chemotherapy.⁸

When an outbreak of group A or group C meningococcal meningitis occurs, all contacts should be vaccinated. Available vaccines are highly protective and have no serious side effects, so there is no reason to restrict vaccination to close contacts. As protection begins only 10-14 days after vaccination, close contacts should also be given chemoprophylaxis and preventive treatment with penicillin. There is no reason to believe that this will interfere with the action of vaccine. Recently, a new conjugated polysaccharide vaccine against group A and group C meningococcal disease was shown to give improved protection in infants and children.⁹ Protein vaccines against certain serotypes of group B meningococci have been tested, but no vaccine exists that covers all group B strains.¹⁰

People should be provided with information as soon as