

## Communicable Disease Report

### Outcome of pregnancy after maternal reinfection with rubella

*P Morgan-Capner, E Miller, J E Vurdien, M E B Ramsay*

#### Introduction

Until recently, rubella reinfection in pregnancy was not thought to present a significant risk to the fetus. However, there have now been a number of reports of congenital rubella following confirmed maternal reinfection<sup>1</sup>, suggesting that the risk may be greater than previously supposed. Two prospective studies<sup>2,3</sup> of the outcome of pregnancy after maternal reinfection found no evidence of fetal infection, but only 40 pregnancies were followed to term and, of these, only 4 were in women whose initial antibody was known to be vaccine-induced. In order to define the risk of reinfection in pregnancy more accurately, a prospective study was set up by the PHLS in October 1987 at the request of the MRC Rubella Sub-Committee. The outcome of 47 pregnancies followed up by March 1991 is reported below.

#### Patients and methods

Cases of suspected reinfection in pregnancy were identified from reports to the Communicable Disease Surveillance Centre (CDSC) and Communicable Diseases (Scotland) Unit (CDSU) by laboratories in England, Wales and Scotland. Maternal cases were diagnosed as confirmed reinfections if there was acceptable evidence of pre-existing rubella antibody or the serological characteristics at the time of infection were indicative of reinfection. The former required at least two previous antibody positive reports by a reliable laboratory method or a documented history of rubella vaccination followed by at least one antibody positive report<sup>4</sup>. Vaccination was considered documented if the date, batch number and/or manufacturer was recorded. Cases were considered probable reinfections when the history or serological features were consistent with reinfection but did not satisfy the above criteria. Where appropriate, maternal sera were tested for rubella-specific IgG avidity using the diethylamine shift method<sup>5</sup>, and for specific IgG subclasses<sup>6</sup>. Reinfection is associated with specific IgG of high avidity whereas low avidity indicates recent primary infection<sup>5</sup>. Specific IgG3 is present in all cases of primary infection but only some cases of reinfection; failure to demonstrate IgG3, therefore, supports the diagnosis of reinfection<sup>6</sup>. The stage of pregnancy at which maternal reinfection occurred was defined as the number of completed weeks between date of the last menstrual period (LMP) and date of the first sample showing a significant rise in titre or the presence of rubella-specific IgM antibody. Evidence of fetal infection was sought by testing products of conception (POC) for rubella virus, or testing infants for IgM antibody at birth and/or the persistence of IgG antibody at one year.

#### Results

During the three and a half year study period, 47 cases of suspected maternal reinfection in pregnancy were reported prospectively to CDSC or CDSU; only 3

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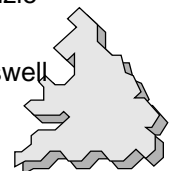
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**Table 1 Outcome of pregnancy for 47 cases of suspected reinfection reported to CDSC between October 1987 and March 1991**

Type of maternal case	Total cases	Outcome of pregnancy		Infant/POC	
		Continued to term	Termination	Tested for evidence of congenital infection	Positive
Confirmed reinfection	34	26	8	31*	3
Probable reinfection	8	4	4	5	0
Persistent IgM reactivity	5	5	—	6*	0
Total	47	35	12	42	3

POC - products of conception

\* includes one pair of twins

cases having occurred since January 1990 (Figure). Confirmed reinfection was diagnosed in 34 (72%) women and probable reinfection in a further 8 (17%). Persistent IgM reactivity was diagnosed in the remaining 5 women by the absence of a significant change in titre following contact and the detection of IgM antibody at a similar concentration in follow up sera taken between 6 weeks and 6 months after the initial IgM positive sample. Further sera taken 2 years later from two of these women were IgM negative.

#### Figure

Five (11%) of the 47 women had a rash whose date of onset was consistent with the serological findings (3 with confirmed and 2 with probable reinfections). No symptoms consistent with rubella were reported in the remaining 42 women who presented with a history of recent rubella contact, usually with their own children. Thirty seven (79%) of the women were parous and a documented history of rubella vaccination was obtained in 12, of whom 7 had been vaccinated at school and 5 post-partum. Only one of the 5 symptomatic reinfections occurred in a confirmed vaccinee.

The outcomes of the 47 pregnancies are shown in Table 1. A total of 6 POC were cultured for rubella virus, 33 cord/neonatal bloods tested for rubella-specific IgM antibody and 16 blood samples tested at one-year IgG for antibody. All 5 women with persistent IgM reactivity continued to term and their infants were IgM antibody negative at birth;

the initial IgM positive samples in these women were obtained between 7 and 16 weeks of pregnancy.

Among the 42 cases of confirmed/probable reinfection, evidence of congenital infection was found in 2 of 6 POC examined and in one of 30 infants tested in this group (Table 2); all 3 congenital infections were the result of confirmed asymptomatic maternal reinfections during the first trimester. None of the 3 mothers had a documented history of rubella vaccination. In one of the two positive POC, virus was isolated from fetal heart, eye and lungs; in the other, virus was isolated from mixed products, presumably containing both fetal and placental tissues. The infant with serological evidence of congenital infection was not tested for IgM antibody at birth but was positive for IgG antibody at 12 months (antibody titre by immunofluorescence of 1 in 256, IgM antibody negative). A second sample taken 2 months later, one week after measles/mumps/rubella (MMR) vaccination, had a titre of 512 and was also IgM antibody negative. The infant had no abnormalities at birth and was developing normally at a year; maternal reinfection had occurred 12 weeks after LMP.

Of the 5 women with symptomatic reinfection, 2 continued

**Table 2 Stage of gestation of maternal reinfection (confirmed and probable cases combined)**

Outcome of pregnancy	Gestation (weeks from LMP)			Totals
	0-12	13-26	27-term	
Termination:				
total pregnancies	7	5	—	12
POC examined	3 (2)	3	—	6
Continued to term:				
total pregnancies	7	20	3	30
infants examined	* 7 (1)	20	3	30

Numbers in brackets are for those with evidence of congenital infection

\* includes one pair of twins

to term and both infants were IgM antibody negative at birth (maternal rashes at 17 and 32 weeks after LMP). The remaining 3 women with a rash had their pregnancies terminated (POC not examined).

## Discussion

The proportion of parous women in this study was high at 79% compared with 50% of the 229 women with confirmed primary infection and known parity reported to CDSC over the same period. This difference reflects the more frequent testing of parous pregnant contacts as a result of exposure to their own children with rubella. The marked decline in the number of diagnosed reinfections during 1990/91 is consistent with other evidence that the introduction of MMR vaccine in October 1988 has already reduced the risk of exposure of pregnant women to children with rubella<sup>7</sup>. As a result, there will be little opportunity in the UK to study the outcome of pregnancy after maternal reinfection in the future.

The observed risk of congenital infection after maternal reinfection during the first 12 weeks of pregnancy was 3 out of 10 (30%). Two congenital infections were diagnosed by virus isolation and have been reported previously<sup>4</sup>. The third was diagnosed by the detection of persistent IgG antibody; post-natal infection in this child seems unlikely as it was born in November 1989 when the prevalence of acquired rubella in the community had been substantially reduced by MMR vaccination<sup>7</sup>. Before the introduction of mass vaccination, acquired rubella during infancy was sufficiently rare for detection of IgG antibody at a year to be considered strong evidence of congenital infection.

In two earlier prospective studies<sup>2,3</sup>, a total of 27 infants whose mothers had a reinfection during the first 12 weeks of pregnancy were examined serologically; no cases of congenital infection were found. The observed risk of fetal infection in our study (3/10) was significantly higher ( $p = 0.003$ , Fisher's exact test). Congenital infection was not associated with vaccine-induced maternal immunity nor with symptomatic reinfection. One of our 3 cases of congenital infection was diagnosed by detecting virus in mixed POC, and its tissue of origin may therefore have been placental not fetal; placental infection without fetal infection has been described after primary rubella<sup>8,9</sup>. In a second case, acquired rubella in infancy cannot be finally excluded as neonatal serum was not tested for IgM antibody.

## Conclusion

The precise risk of rubella reinfection in pregnancy remains uncertain despite a three and a half year national study. Our results indicate, however, that congenital infection may be more common than previously supposed. The observed risk of congenital infection after maternal reinfection in the first 12 weeks of pregnancy, obtained by combining the results of this study and two previous reports<sup>2,3</sup>, is 3/37 (8%), with 95% confidence intervals (CI) from 2% to 22%. The observed risk of fetal infection after primary rubella during the first 12 weeks of pregnancy is over 80%<sup>10</sup>. Exclusion of our two cases in which fetal infection may not have occurred reduces the observed risk to 2.7% (95% CI 0.07% to 14%). Clearly, further studies are required, but they can only be carried out in countries where rubella is still sufficiently prevalent for there to be both the opportunity and the need to determine the outcome of pregnancy after confirmed maternal reinfection.

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# Meningoencephalitis associated with MMR vaccine

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## Introduction

Combined measles/mumps/rubella (MMR) vaccine was introduced into the UK immunisation schedule in October 1988, replacing single measles vaccine for children aged 12 to 15 months. Reactions to MMR are generally mild<sup>1</sup> but meningoencephalitis occurring in the third and fourth weeks post-vaccination has been reported in the UK and elsewhere<sup>2-11</sup>. In some of these cases, mumps virus has been cultured from cerebrospinal fluid (CSF) and nucleotide sequencing of virus isolates has enabled strains of vaccine origin to be differentiated from wild strains<sup>12</sup>.

The routine post-licensing surveillance of adverse reactions to vaccines is through the "yellow card" reporting scheme administered by the Committee on Safety of Medicines (CSM). Reporting to CSM is inevitably incomplete: standardised criteria are not used, and there is no clinical follow-up to determine the outcome of reported reactions. In order to supplement the yellow card scheme, and to provide information on clinical outcome, a two year study of meningoencephalitis associated with MMR vaccine has been mounted by the British Paediatric Surveillance Unit (BPSU) in collaboration with the Communicable Disease Surveillance Centre (CDSC) and the Department of Community Paediatrics, Oxford Health District. This paper reviews the epidemiological and laboratory features of cases reported in the first year of the study.

## Methods

The BPSU reporting scheme has been described fully elsewhere<sup>13</sup>. Starting in February 1990, paediatricians were asked to report all cases occurring within six weeks of MMR vaccination with one or more of the following: neck stiffness (or other signs of meningism), altered consciousness, extreme irritability, convulsions, unexplained screaming attacks, motor or sensory deficit, visual deficit, visual disturbance, or speech disturbance. A questionnaire requesting information about clinical features, laboratory findings and vaccination details was subsequently sent to the reporting paediatrician. Where mumps virus was cultured from CSF, the strains were forwarded to the National Institute for Biological Standards and Control (NIBSC) for characterisation as wild

or vaccine-like.

Cases reported were classified as definite, probable or unrelated. **Definite** cases were those with a vaccine-like strain of mumps virus cultured from CSF. **Probable** cases were those with onset of symptoms between 12 and 28 days following vaccination, but without mumps virus cultured from CSF. **Unrelated** cases were those whose clinical or laboratory features were inconsistent with meningoencephalitis or with an onset of symptoms less than 12 or more than 28 days following MMR vaccination.

## Results

Forty two cases were reported between 1 February 1990 and 30 January 1991. In two cases, the interval between vaccination and onset of symptoms was greater than six weeks. These cases, which did not meet the criteria for reporting to BPSU, were excluded from the analysis. Of the remaining 40 cases, 7 were classified as definite, 17 probable and 12 unrelated. In a further 4 cases, data were insufficient to enable classification.

Since the introduction of MMR, an average of 1.65 million doses of vaccine have been distributed each year. The estimated incidence of vaccine-associated mumps meningoencephalitis during the study period was thus 14.5 per million doses (definite and probable cases) and 4.2 per million doses (definite cases only).

The interval between vaccination and onset of symptoms was known for 34 cases (Figure 1). It ranged from 0 to 35 days, with 23 cases (68%) occurring between days 12 and 25. A vaccine-like strain of mumps virus was recovered from 7 cases, 6 with onset of symptoms between days 14 and 21. In the seventh culture-positive case, symptoms developed 35 days after vaccination.

The age and sex was known for 39 cases, of whom 23 (59%) were aged between 12 and 23 months at the time of onset of illness, although only a third of the definite cases occurred in this age group. Four cases (3 probable) occurred in children aged 5 years or more. The oldest reported case was in a 13 year old boy who developed symptoms 20 days following vaccination. There was no sex difference in reported cases overall, although an excess of males (2:1) was reported in the definite and probable categories.

**Figure 1** Interval between vaccination and onset of symptoms

## Discussion

The risk of meningoencephalitis following MMR vaccination, based on one year of reporting to the BPSU scheme, is not very different from that observed in Canada (5-10 per million doses<sup>6</sup>), West Germany and the US (less than 1 per million doses<sup>7,14</sup>). This must be compared to the risk of meningoencephalitis following natural mumps infection, estimated to occur in 1 per 400 cases<sup>15</sup>. It should not be forgotten that, before the introduction of MMR vaccine, mumps accounted for a fifth of all reported cases of viral meningitis in the UK<sup>16</sup>. Since the vaccine was introduced the incidence of mumps and therefore of related neurological complications has declined rapidly. Between 1989 and 1990, annual notifications fell from 20,713 to 4,270, and laboratory reports to CDSC fell from 265 to 94.

A clustering of reported cases between days 12 and 24 following vaccination was observed in this study. This is similar to the intervals reported in other countries for cases with virus isolated from CSF<sup>5-10,17</sup>. In a study of post-vaccination aseptic meningitis in Yugoslavian children, the onset of symptoms was between 11 and 25 days following vaccination in 92 per cent of cases<sup>11</sup>.

Subclinical meningoencephalitis (pleocytosis in CSF) has been reported in over 65 per cent of patients with mumps parotitis<sup>15</sup>. The proportion of vaccinees with pleocytosis or mumps virus in CSF is unknown. Thus, the isolation of a vaccine-like virus from the CSF of a vaccinated child who develops symptoms of meningoencephalitis may be suggestive, but not proof, of a causal relationship. In the absence of an appropriate temporal relationship such a finding may be coincidental. One culture positive case in this review occurred 35 days post vaccination, which is longer than the normally accepted incubation period for mumps<sup>18</sup>. In the absence of an alternative aetiology this case was classified as definitely vaccine-associated.

Although mumps occurs equally in both sexes<sup>15</sup>, meningoencephalitis following both wild infection and mumps vaccination has been reported more frequently among males than females<sup>7,10,11,16</sup> with ratios ranging from 3:1 to 5:1. In this study, the male:female ratio among definite and probable cases was 2:1.

Clinical data have not been reported here. Vaccine-related mumps meningoencephalitis is usually mild and short lived<sup>19</sup> although permanent sensorineural deafness has been reported<sup>20</sup>. All cases reported to the BPSU will be followed-up twelve months after the acute illness. Follow-up will include a general neurological examination, a Denver-development assessment and a hearing test. The published evidence suggests that neurological complications following MMR vaccination are rare and, unlike the natural disease, do not lead to permanent sequelae. The current programme, which has reduced the incidence of measles, mumps and rubella to an all-time low<sup>21</sup>, should continue to be vigorously promoted.

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# Immunisation for travellers

## Travel information and education

A meeting between a traveller and a health care worker is an opportunity to educate and the importance of general health measures should be emphasised whenever travel advice is sought. Before approaching the topic of immunisation, a traveller should be given general advice on ways of avoiding illness while abroad<sup>1</sup>. This includes personal hygiene, risks of sexually transmitted diseases, food and water consumption, and prevention of insect bites. No vaccine is 100 per cent effective<sup>2</sup> and travellers need a broader perspective than discovering which "shots" are required<sup>3</sup>.

## Target the traveller according to risk

It is important to target the information at the particular traveller and his travel circumstances. Questions requiring consideration include:

1. Who is the traveller and what type of trip is he or she undertaking eg, business, holiday, trekking?
2. To which countries (and areas) is the person travelling?
3. How long is the visit?
4. What previous immunisations has the person received?
5. Does the traveller have any relevant medical conditions?

The adviser should attempt to determine the risk of a particular traveller coming into contact with a potential vaccine preventable disease and weigh this against the small risk of a vaccine reaction. Certain travellers will be at greater risk than others. For example, businessmen and women who visit major cities for a few days are at less risk of many infections than holidaymakers who travel overland and people who live and work abroad for long periods. Although business personnel may require fewer immunisations, they nevertheless should be educated in ways of avoiding disease.

Japanese B encephalitis can be taken as an example. This is a rare disease among travellers but has a high case fatality in adults. The risk of an individual traveller coming into contact with this virus must be assessed before deciding whether to give the vaccine (see p R64).

A list of risk areas, routes of transmission, and available vaccines is given in the accompanying table.

## Diseases imported into the United Kingdom

Two vaccine preventable diseases, typhoid and hepatitis A, affect significant numbers of UK travellers each year. Of 1735 typhoid cases reported to the Communicable Disease Surveillance Centre (CDSC) between 1980 and 1989, 1517 (87%) were reported as having been infected abroad, mainly

in the Indian subcontinent. Similarly, of the 576 paratyphoid infections reported, 555 (96%) were considered to have been acquired abroad. Typhoid vaccine should be considered for any UK resident travelling to areas where the disease is endemic, particularly the Indian subcontinent.

The Office of Population Censuses and Surveys were notified of 723 cases of hepatitis A (between 1987 and 1989) as having been acquired abroad (with visits to India, Pakistan, North Africa and the Canary Islands, West Africa and South America reported in descending order of frequency). This figure will, however, be an underestimate of the actual number of cases acquired abroad. The risk of infection depends on the country visited, duration of stay and the individual's susceptibility. Regular travellers to endemic areas should be screened for hepatitis A antibody and, if seronegative, offered normal immunoglobulin. Notes concerning the prevention of Japanese B and tick-borne encephalitides are set out on pages R64-5.

Many diseases acquired by travellers are not preventable by vaccination. Cases of malaria continue to increase, particularly among travellers to West Africa<sup>4</sup>, and travellers should be advised of mosquito prevention methods (eg nets, repellants) and the use of chemoprophylaxis. A recent review highlights the steady increase in cases of falciparum malaria imported into the United Kingdom<sup>5</sup>. HIV infection acquired by travellers abroad is also increasing<sup>6</sup>. Travellers should be informed and educated about sexually transmitted diseases.

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**Table Immunisation for travel<sup>§</sup>**

<b>Disease</b>	<b>Risk areas</b>	<b>Transmission</b>	<b>Immunisation #</b>	<b>Supply *</b>
<b>Cholera</b>	Africa, Asia, Middle East	Ingestion of contaminated food or water	2 doses 1–4 weeks apart. Certificate valid for 6 months	Wellcome (Tel: 0270 583151)
<b>Diphtheria</b>	Tropical countries under conditions of crowding and poor hygiene	Direct contact with an infected person	3 doses at monthly intervals	Vaccine and Schick test toxin/control Wellcome (Tel: 0270 583151) Low dose vaccine for adults Regent Laboratories (Tel: 081 965 3637)
<b>Hepatitis A</b>	All countries	Ingestion of contaminated food especially shellfish or water	1 injection of human normal Ig shortly before departure	Immuno (Tel: 0732 458101) KabiVitrum (Tel: 0628 850300) Public Health Laboratories and CDSC (Tel: 081 200 6868)
<b>Hepatitis B</b>	All countries	Sexual contact Needle stick injuries	2 doses one month apart, 3rd dose 6 months later	Merck Sharp and Dohme Ltd (Tel: 0992 467272) Smith Kline Beecham (Tel: 0707 325111)
<b>Japanese B encephalitis</b>	South-East Asia	Bite of infected mosquito	2 doses 1–2 weeks apart 3rd dose 28 days later	Cambridge Selfcare Diagnostics Ltd (Tel: 091 261 5950)
<b>Meningococcal infection</b>	Meningitis belt of Africa, Nepal, Mecca during pilgrimage, New Delhi	Direct contact with an infected person	1 injection at least a week before travel	Merieux, UK (Tel: 0628 785291) Smith Kline Beecham (Tel: 0707 325111)
<b>Poliomyelitis</b>	All countries, except Europe, North America, Australia and New Zealand	Direct contact with an infected person, contaminated water, shellfish	3 doses at monthly intervals.	Live vaccine Wellcome (Tel: 0270 583151) Smith Kline Beecham (Tel: 0707 325111) Inactivated vaccine Department of Health (Tel: 071 636 6811) Welsh Health Common Service Authority (Tel: 0222 471234)
<b>Rabies</b>	All countries except UK, Scandinavia, Australia, New Zealand	Bite of infected animal	2 doses one month apart 3rd dose 6–12 months later	Vaccine for pre-exposure prophylaxis Merieux UK (Tel: 0628 785291) Vaccine and specific Ig following exposure PHLS Virus Reference Laboratory (Tel: 081 200 4400)
<b>Tetanus</b>	All countries	Penetrating wound	3 doses at monthly intervals	Wellcome (Tel: 0270 583151) Evans Medical (Tel: 0582 608308) Merieux UK (Tel: 0628 785291)
<b>Tick-borne encephalitis</b>	Forested areas of central Europe and parts of the USSR	Bite of infected tick	2 doses 1–3 months apart, 3rd dose 9–12 months later	Immuno (Tel: 0732 458101)
<b>Tuberculosis</b>	Tropical countries under conditions of crowding and poor hygiene	Direct contact with an infected person, ingestion of unpasteurised milk	Intradermal injection for tuberculin negative person	BCG and PPD District health authorities
<b>Typhoid</b>	All countries except N Europe North America, Australia and NZ	Ingestion of contaminated food, water or milk	2 doses 4–6 weeks apart	Wellcome (Tel: 0270 583151)
<b>Yellow fever</b>	Endemic countries in Africa and South America	Bite from infected mosquito	1 injection at least 10 days before departure	Yellow fever vaccination centres (For list see Immunisation against Infectious disease, HMSO 1990)

<sup>§</sup> Modified from reference 7

# Booster injections are covered in reference 2

\* England and Wales only

Ig - Immunoglobulin

# Prevention of flavivirus encephalitides in travellers to endemic areas

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Advice for travellers to endemic areas on the need for vaccination against Japanese B encephalitis and tick-borne encephalitis is requested frequently. Both diseases may pose a risk in certain areas and at certain times of the year. The current vaccines are unlicensed in the United Kingdom and are available only on a named patient basis (see page R63).

## Japanese B encephalitis

Japanese encephalitis is a serious mosquito-borne flavivirus infection endemic in parts of South East Asia and the Far East<sup>1,2</sup>, especially in rice growing areas, and is the principal cause of epidemic viral encephalitis in the world<sup>3</sup>. Its name reflects the country of initial recognition (1871) and the isolation of the virus in 1934, rather than the present area of greatest occurrence. The virus is transmitted from birds and animals by night-biting culicine mosquitos; pigs being the principal amplifier host for man. Most infections (95%) remain subclinical<sup>1</sup> but when symptoms develop, the disease has a case fatality of at least 20% and results in permanent neurological sequelae in another 30%.

The endemic zone extends from India (especially Uttar Pradesh, the north east and southern states) and Nepal (the Terai and parts of the south and east) across South East Asia to Japan and Korea in the Far East. After extensive local vaccination programmes the risk in the latter two countries is now confined to some rural areas. The countries between India and Japan are included, at least in part ie, Bangladesh, Sri Lanka, Thailand, Myanmar (formerly Burma), Malaysia, Indonesia, Cambodia, Laos, Vietnam, China, the Eastern USSR, Taiwan and the Philippines. Some of the Polynesian islands of Melanesia and Micronesia may be affected.

In much of the region there is a hotter and wetter (monsoon) season from June or July to September/October/November and this is the time of highest risk. However, some of the more tropical areas have less seasonal variation in both climate and disease pattern, and outbreaks occur sporadically.

The risk to an individual traveller is difficult to assess, for whilst there are large areas where the disease is a public health problem (parts of India and Nepal, Thailand, China, Korea and some rural parts of Japan), there have been only isolated cases in civilian travellers and only one recognised case has been imported into the United Kingdom. A 35 year old British born female resident of Hong Kong flew back to the United Kingdom while symptoms developed and died of serologically proven Japanese encephalitis<sup>4</sup>. There have also been American and Australian military cases reported following the Korean and Vietnamese wars and postings to South East Asia.

However, adventurous travel is presently on the increase and a vaccine is now available in the United Kingdom against this disease. Advice for prophylaxis includes avoidance of mosquito bites by use of repellents and cover up clothes, especially around dusk but also through the

night to dawn. Rural travellers should sleep indoors and use mosquito nets wherever possible. Vaccine should be considered, in addition, for those going on trips lasting more than 2-3 weeks to rural endemic areas, although there may also be a risk in some towns in the Far East where pig keeping is prevalent and the paddy field breeding grounds of the culicine mosquitoes are close by.

Inactivated vaccines are manufactured in Japan, Korea and China. The last is not an internationally recognised vaccine. The Japanese and Korean vaccines, despite being mouse brain preparations, are widely used in their countries of origin without reports of serious side effects; and Hoke et al working in Thailand with Japanese vaccines concluded that they were safe and effective<sup>3</sup>. The limited experience with the unlicensed vaccine in the United Kingdom (the Japanese version since about 1984) and by the US military, is similar. The vaccine is not yet available to civilians in the USA who are recommended to obtain it (for high risk destinations) by contacting US consulate facilities<sup>2</sup>, and some request immunisation while passing through the United Kingdom on their way eastwards.

Studies on US Army personnel suggest that the initial two doses provide adequate antibody levels for up to three months and that a third dose will maintain the levels for longer trips<sup>5</sup>. A booster can be given at a year if the traveller is still at risk.

A possible additional benefit of the vaccine for travellers to Asia concerns the interesting speculation by Hoke<sup>3</sup> that the vaccine may provide some protection against dengue haemorrhagic fever which is caused by a related flavivirus.

## Tick-borne encephalitis

Tick-borne encephalitis is a flavivirus infection which is transmitted to man by the bite of an infected tick. The principal vector is the common wood tick *Ixodes ricinus* which inhabits the undergrowth of forested areas in Central, Eastern and Northern Europe<sup>6</sup>. The disease is particularly common in Austria, where it is known as fruhsummer (early summer) meningoencephalitis, and transmission occurs most frequently in late spring and summer.

After an incubation period of 7-14 days a flu-like illness develops, which lasts for up to a week. Approximately 30% of patients develop neurological complications which include meningitis, encephalitis and occasionally muscular paralysis. There is no specific treatment, although a specific immunoglobulin is available for pre and post-exposure prophylaxis.

A purified killed vaccine is available which has been widely used in Austria, resulting in a 10-fold reduction in disease incidence<sup>7</sup>. The vaccine should be considered for travellers to endemic areas who plan to walk through, or camp in, dense woodland terrain. A basic immunisation series consists of two doses 1-3 months apart and gives protection for one year. A third dose 9-12 months later provides longer lasting protection. Reactions are rare, although there may be some swelling and redness at the injection site. Additional advice for travellers to high risk areas is to:

1. use woodland paths free of grass and shrub when walking in the forest
2. wear stout shoes or boots and thick socks or stockings
3. apply insect repellants
4. boil unpasteurised milk as this may occasionally be a vehicle of infection.

### Other flavivirus encephalitides

Many other flaviviruses cause encephalitis in man. They are transmitted from animal reservoirs by mosquitos (St. Louis encephalitis, Murray Valley encephalitis, Rocio encephalitis) or ticks (Kyanasur Forest disease, Louping ill, Powassan encephalitis). While vaccines are not available for these diseases (yellow fever is an exception - see page R63), general measures should be taken to prevent mosquito and tick bites in infected areas (see above). In some flavivirus infections (eg Rio Bravo encephalitis) the method of transmission is not known.

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## Legionnaires' disease surveillance: England and Wales, 1990

In 1990 there were 188 cases of legionnaires' disease in residents of England and Wales reported to the national surveillance scheme for legionnaires' disease at CDSC. This compares with 238 in 1989 and 278 in 1988.

One hundred and forty-seven cases (78%) were male, ranging in age from 22 to 85 years (average of 55 years) (Figure 1). The forty-one female cases were aged between 31 and 76 years (average of 59 years). Twenty-three patients (12%) died (19 males and 4 females). Five cases of Pontiac fever were also reported (3 males and 2 females).

### Figure 1 Distribution of age and sex

Isolates were obtained from 37 (20%) of cases of legionnaires' disease; 36 were identified as *Legionella pneumophila* (the species for the remaining case was not stated). Twenty-four of the *L. pneumophila* isolates were serogroup 1, one was serogroup 2, one was serogroup 3, five were serogroup 10 and 1 was serogroup 12. The serogroup

of the remaining four was not specified. A further 90 (48%) cases were confirmed by seroconversion (four-fold or greater rise in antibody titre). The remaining 61 cases (32%) were diagnosed presumptively on the basis of a single high antibody titre only. Three Health Regions reported more than 10% of the cases: Trent - 30 cases (16%); West Midlands - 26 cases (15%) and North Western - 18 cases (10%).

There were four nosocomial infections, two of which occurred in a hospital in the West Midlands. In addition, three other cases of legionnaires' disease were possibly nosocomial: one was associated with a hospital in the East Midlands where cases had been reported in 1988 and 1989.

There were five outbreaks (two or more cases associated in time and place) of legionellosis in England and Wales during 1990. The largest of these occurred in November and was centred on an industrial estate in the North West of England where four cases of legionnaires' disease and one case of Pontiac fever occurred. Two further outbreaks of legionnaires' disease occurred in the North West during the year: two cases in July were associated with an industrial complex, and a further two cases were reported from an outbreak that occurred in September/October in a town centre where a further nine suspected cases were also ascertained. Two cases of legionnaires' disease occurred among the staff of a manufacturing plant in South West London in August and October. In none of these outbreaks was it possible to identify the source of infection. The fifth outbreak consisted of the two nosocomial cases referred to above.

Ninety-two cases of legionnaires' disease were associated with travel abroad in the two weeks before the onset of illness. Of these, 39 (42%) had been to Spain or the Balearic Islands. Most of the travel cases occurred between May and

October, the peak holiday period (Figure 2). Clusters involving two or more cases were ascertained from 5 hotels abroad: three cases from Pineda, Spain and three cases from Alcudia, Majorca (which were associated with cases in other foreign nationals); two cases each from Rimini (Italy) and Kephallonia (Greece), and two cases from the Dominican Republic. A further thirteen cases had travelled only within the United Kingdom.

Sixty seven cases of legionnaires' disease (35%) were

#### Figure 2 Cases by month of onset and travel

sporadic (not known to be associated with an outbreak or travel and not hospital-acquired). Of these 67 cases, 54 were male and 13 were female.

#### Comment

Regular and effective maintenance of water systems is the most important step in preventing outbreaks of legionellosis. Although no major outbreaks occurred in England and Wales during 1990, investigation of some of the small outbreaks revealed many water systems in a poor condition despite the wealth of publicity about this problem and published guidance<sup>1-3</sup>.

Nearly 50% of the reported cases occurred in residents

who had recently travelled abroad, most often on holiday to countries bordering the Mediterranean. A history of recent travel should be elicited from all cases of community-acquired pneumonia and an appropriate antibiotic given if the diagnosis is suspected.

In only 20% of cases was the diagnosis confirmed by culture. Isolation of the organism, where available, may not only provide the diagnosis more rapidly than serology, but is also valuable in linking clinical cases with suspected environmental sources.

Just over a third of the cases were sporadic and little is known about their environmental source(s). Bhopal et al<sup>4</sup> have recently reported an association between some of these cases and proximity to wet cooling systems (cooling towers) in city centres. How important this source is as a cause of sporadic cases throughout the country is not known.

#### Acknowledgements

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#### Citation of articles published in the *Communicable Disease Report*

Articles published *before* the *Communicable Disease Report* became a formal publication at the beginning of 1991 should be cited with the weekly issue number in parentheses eg:

Newman CPS, Peel RH, Jones DM. Meningococcal meningitis in a military establishment. *Communicable Disease Report* 1990;(33):3-4. Internal publication of the Public Health Laboratory Service, London.

Citations for articles published *after* the beginning of this year should use the appropriate volume and page numbers as follows:

Maguire HC, Begg NT, Handford SG. Meningoencephalitis associated with MMR vaccine. *Communicable Disease Report* 1991;1:R60-1.

Page numbers for the four-weekly review require the prefix 'R' in contrast to the simple pagination of the weekly bulletin.

## Health risks from exposure to algae

R. Philipp, M G M Rowland, P J Baxter, C McKenzie, R H Bell

Algal blooms affecting inland waters were unusually pronounced in 1989 when reports of animal deaths attracted considerable media coverage. Although there was less publicity in 1990, algal blooms recurred and public awareness has been raised by further reports of animal deaths and of human illness following contact with blooms. It is possible that the problem will recur this summer. This brief report is intended to help those people who may be approached for advice regarding hazards and risk reduction.

Blue-green algae (cyanobacteria) commonly occur in fresh and brackish waters. Massive annual growths occur during favourable climatic conditions and are probably compounded by nutrient enrichment from natural waters, agricultural fertiliser run-off, or domestic/industrial effluents<sup>1</sup>. During calm weather algae can rise to the water surface to form a scum which may look like blue-green paint or jelly. The scum can be blown around the water surface and thus appear at different places at different times. It can also disappear and reappear on subsequent days. Although algal scums are not always harmful, it is sensible to regard them as such. Even in the absence of scums, algae may produce toxins which if ingested can cause fatal poisoning of agricultural livestock, wild animals, birds and fish<sup>1</sup>. Although deaths have not been reported in humans, ingestion of toxic algae or body immersion in scum-containing water has been associated with dizziness, headaches, muscle cramps, nausea, vomiting, gastroenteritis, liver damage and pneumonia<sup>1,2</sup>, and skin contact has been associated with irritation and contact dermatitis<sup>1</sup>.

In April 1990 the National Rivers Authority (NRA) established a routine monitoring programme for such algae at 500 inland water sites in England and Wales. The NRA has determined a trigger concentration level for "a potential to cause harm". This is defined as six countable cyanobacteria units (be they colonies or single cells) per 0.5ml water, when examined microscopically by appropriate methods. When this level is reached or exceeded the water owner, local chief environmental health officer, CCDC or MOEH, and the area office of the Ministry of Agriculture, Fisheries and Food, are immediately informed. Where appropriate, and to comply with NRA guidance, notices are then placed by water owners to warn recreational water users that exposure may have adverse human or animal health effects.

The Institution of Environmental Health Officers (IEHO) recently drew attention to the potential health risks<sup>3</sup>, and further noted that "although the hazards are known, the likelihood of health effects from different patterns of occupational and recreational exposure is not clearly understood"<sup>4</sup>. CCDCs or MOEHs may be contacted about the risk assessment studies that were established to try and clarify these points<sup>4,5</sup>.

The IEHO, Local Authority Unit of the Health and Safety Executive and some Public Health and Occupational Physicians are already being asked for practical suggestions to help reduce the potential for harm and for advice about risk assessment. It may help CCDCs and MOEHs to be aware of existing information and guidelines on the subject:

1. The National Rivers Authority has published a review of blue-green algae and their toxins; factors affecting the incidence of their blooms; approaches to their control; the impact on animals and humans, and recommendations for eutrophication control, long term monitoring, research and development<sup>6</sup>.
2. The PHLS *Communicable Disease Report* has previously outlined a health and safety policy for recreational water exposure<sup>7</sup>. The general points in it, and those for Weil's disease, apply equally where algal blooms have been identified.
3. The Royal Yachting Association has prepared a code of practice to minimise the risks for dinghy sailors on affected inland waters. The points in it have been brought to the attention of IEHO members and are also relevant for other water users with different exposure patterns<sup>8</sup>.
4. Rotherham Metropolitan Borough Council has found it prudent to establish visual inspection criteria that determine if recreational waters may be open to the public and to train staff in their use.
5. Risk assessment studies during 1990 amongst 609 dinghy sailors in Hampshire and Avon did not identify morbidity patterns that could be causally associated with exposure to algal blooms<sup>9</sup>.
6. Studies amongst 246 recreational fisherman in Grantham and 67 windsurfers in North Norfolk and Rotherham did not identify any morbidity patterns that could be causally associated with exposure to algal blooms. (Unpublished observations).
7. The local authority unit of the Health and Safety Executive is proposing guidance to help local authorities as enforcers of the Health and Safety at Work Act.
8. The World Health Organisation is publishing the final report of a WHO Working Group on the health impact of human exposure to fresh and saline recreational waters as one of their Environment and Health Series and as part of their interest in blue-green algal blooms and red algal tides<sup>10</sup>.

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### Comment from Dr J V Dadswell, Chairman of PHLS Microbiology of Water Committee

Blue-green algae are receiving increasing attention, both as indicators of environmental change and for their potential to cause adverse health effects. Although previously recognised as widespread in relatively brackish fresh and estuarine surface waters throughout warm climates, it is only recently that they have become more prevalent in temperate climates including the United Kingdom (UK). The increasing use of surface waters in the UK for recreational purposes has also brought an awareness of health risks associated with such practices, which now include possible exposure to the toxins of blue-green algae. There is also a remoter risk of the contamination of water supplies with algal toxins.

There are many questions that require answering before the health significance of algal toxins can be accurately quantified. Relatively few species appear to produce them and it is difficult to ascertain the factors involved - toxin production seems to be very variable, even among those algae known to be capable of producing them, and little is known about dose-related effects in man. At present, the National Rivers Authority's approach of identifying the

presence of blue-green algae in relatively large numbers as an indication of the likely presence of toxins seems the most practical, although it may overestimate the possible health risks. Hitherto, the detection of toxins has largely depended upon the use of test animals but tissue culture and serological methods are being developed and should enable simpler, more rapid tests to be done.

Algal overgrowth is dependent upon many factors. These include raised temperature and the presence of suitable nutrients. To what extent global warming can be retarded by human action is uncertain. Changes in agricultural practices, and in the purification of sewage effluents, seem more likely to produce less favourable conditions for algal growth in the more immediate future but at some cost.

A PHLS symposium on blue-green algae, to be held on 14 June, will address some of these questions and hopefully point the way to finding answers. In the meantime, the procedures outlined above provide a sensible way of minimising the possible health risks to recreational users of surface waters.

### **Submission of manuscripts to the *Communicable Disease Report***

Authors are invited to submit papers on all aspects of surveillance, prevention and control of communicable diseases to be considered for publication in the *Communicable Disease Report*.

Manuscripts and their references should conform to the "Uniform requirements for manuscripts submitted to biomedical journals" (ie, the Vancouver style)<sup>1</sup>.

1. International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1991;302:338-41.