

Communicable Disease Report

The notification of infectious diseases in England and Wales

A McCormick

Summary

For nearly a hundred years, it has been a statutory requirement for doctors to notify a Proper Officer of the local authority (now, usually, the Consultant in Communicable Disease Control) of cases of certain infectious diseases. This prompts local investigation and appropriate action to control the disease. These data are also used for the analysis of local and national trends. This article describes how the notification system has evolved, its legal basis, and some of its strengths and weaknesses, and suggests how improvements could be achieved. Trends in notification data are presented for several diseases and compared with data derived from other sources.

Historical introduction

Notification of specified infectious diseases is a legal requirement under the Public Health (Control of Disease) Act 1984¹ and the Public Health (Infectious Diseases) Regulations 1988².

During the nineteenth century, various Acts of Parliament provided powers aimed at preventing the spread of cholera and "epidemic, endemic or contagious" diseases^{3,4}, culminating in the Public Health Act 1875⁵ which consolidated and amended previous Acts relating to public health in England. However, it was not until 1889 that provision was made enabling any urban, rural or port sanitary district to make certain infectious diseases notifiable⁶. These powers were made mandatory in London in 1891⁷ and throughout the remainder of England and Wales in 1899⁸. The responsibility to notify fell on both the head of the family (or nearest relative, or person in charge of or in attendance on the patient, or the occupier of the building) and the "medical officer attending on or called in to visit the patient". The head of the family was relieved of this responsibility in 1968, which then became solely the concern of the attending medical practitioner⁹. Notification by the head of the family and the medical practitioner had to be sent to the medical officer of health of the district. The additional requirement to notify on suspicion of the diagnosis was introduced in 1968⁹.

The form of certificate was prescribed by the Local Government Board, which was set up in 1871 and became the Ministry of Health in 1919¹⁰. The version currently in use is that set out in the Public Health (Infectious Diseases) Regulations 1988, or a form "substantially to the like effect"². A fee of 2s 6d (12.5 p) was to be paid by the local authority to the medical practitioner if the case occurred in his private practice and of 1s (5p) if in his practice as medical officer of any public body or institution. In 1984, the District Health Authority became responsible for paying the fee instead of the local authority¹. This is currently £2.20. Failure to notify carries a fine.

The 1889 Act did not apply to any building, ship, vessel, boat, tent, van, shed or similar structure belonging to Her Majesty the Queen, or to any inmate thereof⁶. However, under the Act currently in force (Section 73)¹, the authority, for example a Government department controlling Crown property, may agree with the county council or district local authority in which the property is situated that any relevant provision can apply to the property.

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A McCormick

R19

Malaria imported into the United Kingdom during 1991

D J Bradley
D C Warhurst

R25

The clinical features of imported malaria

M G Brook
B A Bannister

R28

The resurgence of scabies

N J Barrett
D L Morse

R32

'COVER' (Cover of vaccination evaluated rapidly): 24

T J Connellan
S Leon
N T Begg

R34

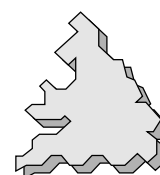


Table 1 Notifiable diseases in England and Wales
(with the date each was made notifiable under
current or similar nomenclature – see text)

Infection	When made notifiable
Under the Public Health (Control of Disease) Act 1984	
Cholera	1889
Food poisoning	1949
Plague	1900
Relapsing fever	1889
Smallpox	1889
Typhus	1889
Under the Public Health (Infectious Diseases) Regulations 1988	
Acute encephalitis	1918
Acute poliomyelitis	1912
Anthrax	1960
Diphtheria	1889
Dysentery (amoebic or bacillary)	1919
Leprosy	1951
Leptospirosis	1968
Malaria	1919
Measles	1940
Meningitis	1968
Meningococcal septicaemia (without meningitis)	1988
Mumps	1988
Ophthalmia neonatorum	1914
Paratyphoid fever	1889
Rabies	1976
Rubella	1988
Scarlet fever	1889
Tetanus	1968
Tuberculosis	1912
Typhoid fever	1889
Viral haemorrhagic fever	1976
Viral hepatitis	1968
Whooping cough	1940
Yellow fever	1968

The nineteenth century Acts restricted the activities of persons with “any dangerous infectious disorder” and provided other legal powers to prevent spread⁵. Some of these powers were restricted to notifiable diseases in 1936^{11,12}, and vary from disease to disease, as specified in Schedule 1 of the regulations currently in force².

Specific conditions

The 1889 Act⁶ listed the diseases to be notified as “smallpox, cholera, diphtheria, membranous croup, erysipelas, the disease known as scarlatina or scarlet fever, and the fevers known by any of the following names, typhus, typhoid, enteric, relapsing, continued, or puerperal”. Local authorities were given the power, upon approval by the Local Government Board in London, to extend the definition of diseases to be notified either temporarily or permanently within their district. These powers have been used, for

example, for the local surveillance of anthrax, glanders in man, hydrophobia, chickenpox, German measles, acute rheumatism in children, pemphigus neonatorum and scabies.

Under the 1875 Act (Section 130)⁵, the Local Government Board and later government institutions have used the power “to make, alter and revoke regulations” to prevent the spread of infectious disease by adding to and subtracting from the list of diseases to be notified either locally or nationally. The 30 diseases or infections currently to be notified throughout England and Wales are listed in table 1. Five diseases (cholera, plague, relapsing fever, smallpox and typhus, and food poisoning) are notifiable under the Act¹, notification of the remaining 24 being required under the Regulations². Although AIDS is not a notifiable disease, certain legal powers can be applied to people with AIDS under regulations issued in 1985¹³. Doctors are urged instead to report HIV infections, AIDS cases, and those who are HIV positive and die without developing an AIDS indicator disease to the voluntary confidential surveillance schemes at CDSC¹⁴.

Under separate arrangements, the number of people attending with sexually transmitted diseases are reported by genito-urinary medicine clinics to the Department of Health¹⁵.

To complement notification by medical practitioners, workers in certain food and milk production premises are required, under food legislation, to inform the manager if they suffer from specified infectious conditions; the manager must notify the proper officer¹⁶.

Purposes of notification

Notification was introduced in 1889 as a means of identifying and preventing the spread of infectious diseases and providing legal powers as mentioned above⁶. The urgency of notification of some diseases for this purpose was recognised in 1968⁹ when doctors were required to notify before the diagnosis was confirmed, enabling local health workers to make appropriate investigation and take action at the earliest opportunity.

As a by-product of notification, local and national statistics are produced and used for surveillance purposes. Reports of notifications were collected centrally for London in 1891⁷ and throughout England and Wales in 1910¹⁷. Since 1928, reports of notifications have been sent by medical officers of health and, more recently, by proper officers (usually the Medical Officer for Environmental Health or the Consultant in Communicable Disease Control) to the General Register Office which became the Office of Population Censuses and Surveys (OPCS) in 1970. These data have been published in the Registrar General's Weekly Return since 1922.

The present situation

The current law^{1,2} states that “If a registered medical practitioner becomes aware, or suspects, that a person whom he is attending within the district of a local authority is suffering from a notifiable disease or from food poisoning, he shall, unless he believes, and has reasonable grounds for believing, that some other registered medical practitioner has complied with this sub-section with respect to the patient, forthwith send to the proper officer of the local authority for that district a certificate stating –

- (a) the name, age and sex of the patient and the address of the premises where the patient is,

- (b) the disease or, as the case may be, particulars of the poisoning from which the patient is, or is suspected to be, suffering and the date, or approximate date, of its onset, and
- (c) if the premises are a hospital, the day on which the patient was admitted, the address of the premises from which he came there and whether or not, in the opinion of the person giving the certificate, the disease or poisoning from which the patient is, or is suspected to be, suffering was contracted in hospital.” (Section 11(1) of the 1984 Act).

Key points about notification are set out in table 2.

Local arrangements

The officer who receives the certificate has to send a copy within 48 hours to the district health authority to which the address specified on the certificate relates. If the certificate relates to a patient in a hospital who came there from premises outside the local authority district in which the hospital is situated, and the certificate states that the patient did not contract the disease or poisoning in the hospital, the officer has to send a copy of the certificate to the proper officer of the local authority and to the district health authority for the district within which the premises from which the patient came are situated (S.11(3)).

The local authority has, upon application, to supply forms of certificate free of charge to any registered medical practitioner practising within its district (S.11(2)). The district health authority has to pay the current fee to a registered medical practitioner for each certificate sent by him (S.12(1)). The Secretary of State may issue regulations specifying which diseases are to be notified or make other modifications (S.13). In addition, a local authority may direct that other diseases become notifiable within their district, subject to approval by the Secretary of State (S.16). The current Act includes a number of restrictions on the movements or work of people with a notifiable disease, the disposal of infected articles, and the use of infected premises and public conveyances.

Table 2 Key points about notification	
By whom?	the doctor attending the patient when diagnosed
What?	any clinically manifested disease listed in table 1
When?	on suspicion or diagnosis of clinical disease
How?	on a form available from the Proper Officer of the local authority, usually the Consultant in Communicable Disease Control (CCDC); also by telephone or fax if urgent action is likely to be required
To whom?	the Proper Officer/CCDC of the local authority district where the patient is when diagnosed
Why?	so that the Proper Officer/CCDC can take appropriate action to investigate and control spread, and to provide data for local and national surveillance

Table 3 Diseases to be reported to the Chief Medical Officer and the Director of CDSC
Diagnosed or suspected diseases or incidents to be reported immediately
Cholera
Plague
Smallpox
Yellow fever
Any serious outbreak of any disease (including food poisoning)
Diseases for which a copy of the certificate should be sent immediately
Cholera
Plague
Smallpox
Yellow fever
Leprosy
Malaria or rabies contracted in Great Britain
A viral haemorrhagic fever

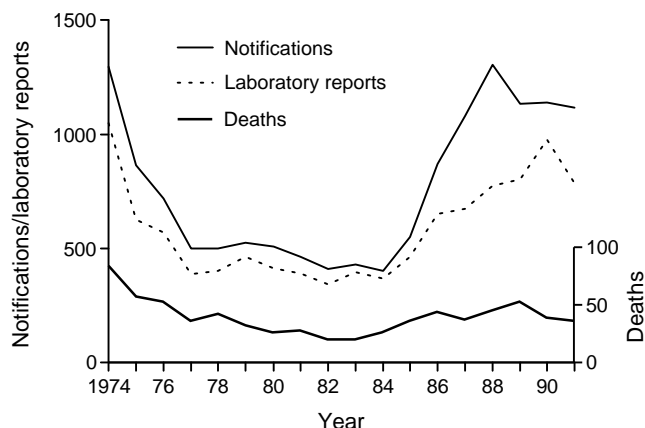
More importantly, legal powers provide for compulsory medical examination (S.35), removal to (S.37) and detention in hospital (S.38), and precautions to be taken when people with a notifiable disease die in hospital and the disposal of the body (S.43,44). A proper officer of a local authority for any district may, by notice in writing, request any person to discontinue his work with a view to preventing the spread of a notifiable disease or food poisoning (S.20(1)). The local authority has to compensate a person who has suffered any loss in complying with this request (S.20(2)). Section 12 of the 1988 regulations² states that confidentiality of notification documents must be ensured.

National surveillance

Under current law², each proper officer is required to send to the Registrar General at OPCS a return of the number of cases of each disease notified during the week ended on the preceding Friday night, to arrive by post on the morning of Tuesday at the latest. If a certificate is sent by the proper officer of one district to the proper officer of another district under the conditions mentioned above, the case should be reported to OPCS by the second district only. The proper officer is required to send a copy of the return to the appropriate medical officer of the appropriate district health authority. The proper officer is also required to send to the Registrar General, by post every three months (and not later than three weeks after the end of the period covered), any notifications that have been corrected subsequently by the notifying medical practitioner, or the practitioner in charge of the patient, and a copy of this return is sent to the appropriate medical officer of the appropriate district health authority.

Copies of a reporting form are sent each November by OPCS to the proper officer of each local authority with instructions for their completion during the following year. All notifiable diseases should be reported on this form with the exception of leprosy; copies of leprosy notifications are

Figure 1 Notifications of, and deaths from, meningococcal meningitis (reported to OPCS) and laboratory isolates of *Neisseria meningitidis* associated with meningitis (reported to CDSC)



sent direct (marked confidential) to the Director of the PHLS Communicable Disease Surveillance Centre. In addition to cases of notifiable diseases, proper officers are also asked to report to OPCS (on the same form) people who have been ascertained as having food poisoning by means other than formal notification, for example by reports of food poisoning from a laboratory or additional cases identified by an environmental health officer in the course of his investigations. On receipt at OPCS, the data are checked and processed promptly on a mainframe computer. The results are faxed to the Department of Health and to CDSC.

In addition to the requirement for proper officers to report notifications to OPCS they are also required, under Section 6 of the 1988 Regulations², to "immediately inform the Chief Medical Officer for England...or...the Chief Medical Officer for Wales and the Director of CDSC¹⁸ of any case or suspected case of" the diseases listed in table 3. Contact telephone numbers in England are: 071 972 1058 for food and water-borne disease and 071 972 3357 for other communicable diseases (outside normal office hours for all diseases - 071 210 5368/5371). The contact number at the Welsh Office is 0222 823468 (outside normal office hours - 0222 825111) and at CDSC is 081 200 6868.

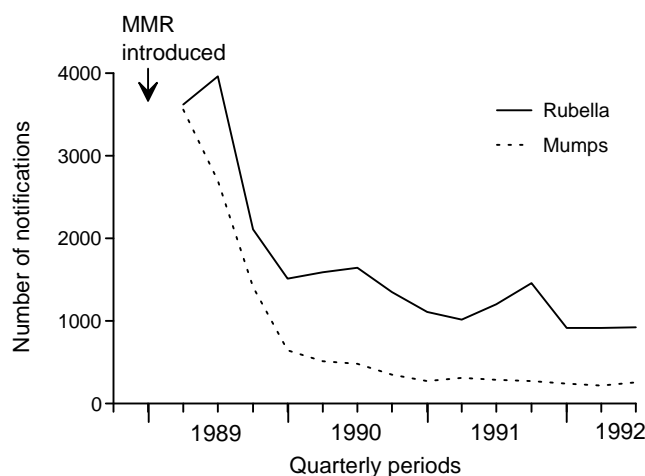
Distribution of information

The numbers of notifications reported to OPCS are published in the Registrar General's Weekly Return, which is despatched on the Monday following receipt of reports on the previous Tuesday. Notification data are also published in the quarterly OPCS *Infectious Disease Monitors*. Single copies of both these publications are sent free to each health and local authority; additional copies are available for a modest charge (Information Branch, OPCS, 10 Kingsway, WC2B 6JP). An annual volume, *Communicable Disease Statistics*, is obtainable from HMSO bookshops. In addition, information on notifications is disseminated by CDSC through the *Communicable Disease Report* and via Network PHLS (Epinet). OPCS is able to provide unpublished statistics, for which a charge may be made if further processing is necessary.

Analysis of trends

Changes in incidence and severity, and the effectiveness of intervention measures, can be estimated when notifications

Figure 2 Mumps and rubella notifications for children aged under five years



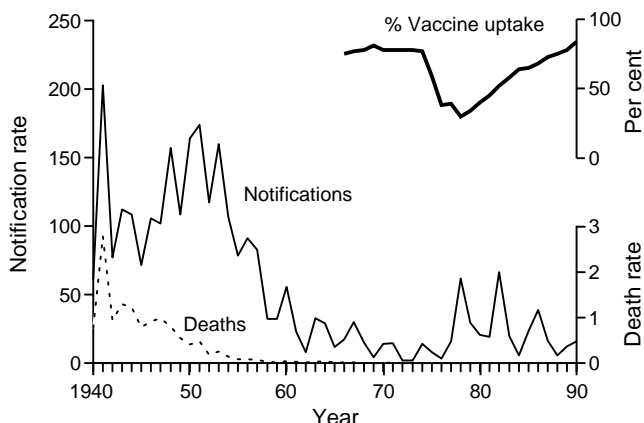
are combined with other data such as laboratory reports, immunisation uptake and mortality statistics (Figures 1-3). For these purposes, it is not essential that every case is notified and changes in notification policy may introduce bias.

Notification data complement the laboratory reporting system, the former being based on clinical diagnoses and the latter on proven infections. Notifications provide information on the incidence of some diseases which are not always diagnosed in the laboratory, for example measles and whooping cough (Figure 3), or for which specimens submitted to a laboratory, for example rubella, are from a biased population, women and their partners being most likely to be tested in their child-bearing years (Figure 4). On the other hand, some conditions such as legionnaires' disease can only be diagnosed reliably in a laboratory and notification, if this were by clinicians on suspicion, could identify large numbers of false-positives suffering from other, more common, types of chest infection.

Under-notification

The notification system provides surveillance data going back many decades for some diseases, but it has been criticised for its many faults. The first is incompleteness. The degree of under-reporting varies for different diseases and has been estimated for many. For tuberculosis, the data collected through the notification system is generally accurate¹⁹. Variations in notification habits between doctors have been reported in Hull²⁰, and between those who do and do not report new episodes weekly to the Royal College of General Practitioners (RCGP) Research Unit in Birmingham^{21,22}. The proportions of cases of meningococcal meningitis that were notified during outbreaks in Mid-Glamorgan²³ and Gloucestershire²⁴ were 65% and 57%, respectively. In Mid-Glamorgan, the proportion of cases of other meningitides notified was even lower²³. It has been estimated that only 40-60% of cases of measles and 5-25% of cases of whooping cough are notified²¹ (assuming that all children have been immunised or develop clinical measles and pertussis by the age of 15 years). Only 79% of all known cases of typhoid fever, 25% of leptospirosis cases and 33% of tetanus cases were notified in 1983²⁵. More recently, attention has been focused on the under-reporting of hepatitis^{26,27}, ascribed by the authors in part to the high proportion of asymptomatic

Figure 3 Whooping cough notifications and deaths (all ages), expressed as rates per 10,000 population (aged under 15 years), and percentage vaccine uptake



cases, which the notification system is not designed to cover.

It is possible that doctors change their notification habits under different circumstances. Opinions vary about whether doctors are more likely to notify during an epidemic. During the peak of whooping cough notifications in 1982 (Figure 5), the estimated proportion of cases notified was thought to be similar to that in inter-epidemic years²⁸. Several studies^{29,30} have compared the ratio of notification rates with rates of cases reported to the RCGP Research Unit. These suggest an increase in the proportion of cases notified during an epidemic, although this could be due to higher immunisation uptake among children on the lists of doctors reporting to the RCGP scheme, resulting in a lower incidence of the disease. Another factor which could influence the number of cases notified is the size of the fee paid. However, when this was increased from 25p to £1.15 in 1984 there was no evidence to suggest any

Figure 4 Distribution of rubella notifications (OPCS) and laboratory reports (CDSC) by age and sex (for 1990)

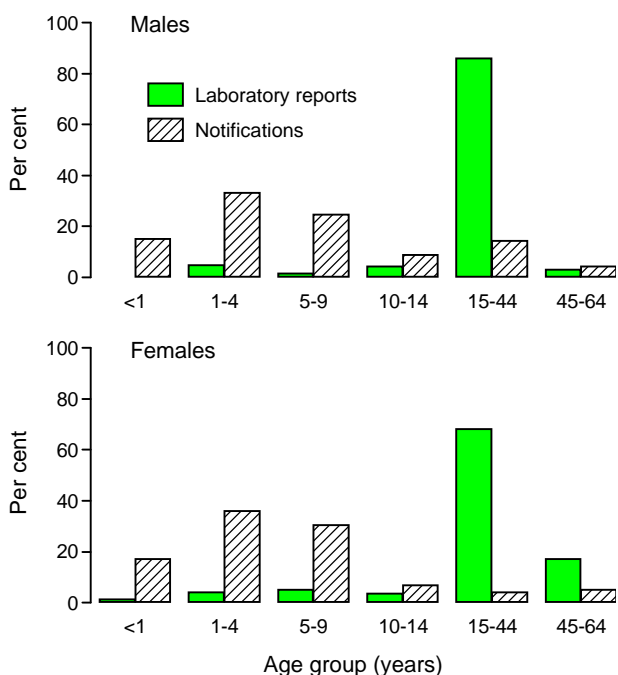
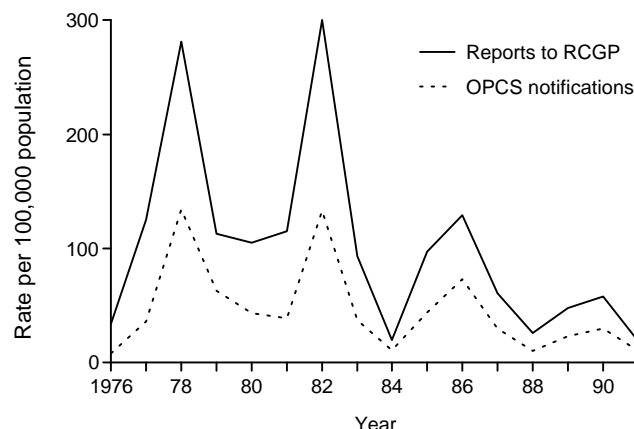


Figure 5 Notifications (OPCS) and new episodes of whooping cough (RGCP) expressed as rates (for 1976 to 1991)



effect on the proportion of cases notified³¹.

Over-notification

For some conditions the number of cases reported is an overestimate. For example, it is correct for a case of cholera to be notified on suspicion so that appropriate action can be taken as early as possible. However, laboratory examination frequently indicates that the illness is caused by a non-cholera vibrio which is not notifiable and the notification should therefore be cancelled but often is not. Another example is diphtheria, a suspected case of which should be notified immediately to the proper officer, but may eventually be identified as non-toxicogenic in the laboratory. The diagnosis may remain unproven, and could be incorrect, for other infections notified on clinical diagnosis or suspicion.

Speed of notification

Speed of notification of some infections is essential for effective control but considerable delays can occur between the onset of illness and receipt of the notification certificate by the proper officer³². This may have several causes, but the delay should be minimal in most cases. While there is not usually any urgency for the notification of diseases such as measles and rubella, others such as meningococcal infection, hepatitis, typhoid, cholera and food poisoning may require urgent action and are more appropriately notified by telephone, fax or other electronic means, with subsequent confirmation by a certificate for which the fee will be paid. Some local authorities list diseases to be notified on the front of certificate pads, indicating those which should be reported immediately by telephone.

Training for notifiers

Deficiencies in notification are largely due to ignorance of the legal requirements by hospital doctors³³ and general practitioners³⁴, and to a lack of understanding of the purposes of notification³⁵. They reflect a defect in the education of young doctors at an appropriate stage in their training, eg, at the start of their first house officer appointment and during postgraduate training for general practice. There is a need for proper officers to encourage notification. This can be achieved by simple methods such as ensuring that pads of certificates are prominently displayed in hospital wards

and are readily available to GPs, and by paying fees promptly. Some laboratories put stickers on reports of identifications of organisms that cause a notifiable disease, to remind the clinician to notify. Some proper officers remind clinicians to notify individual cases that have been identified. Regular feedback to doctors is essential to stimulate communication and interest. In some areas, information is circulated to doctors on the numbers of cases notified locally and nationally and the opportunity is taken to draw attention to individual 'success' stories. The adoption of computing systems by an increasing number of general practitioners introduces the possibility of automatic notification when the diagnosis of a notifiable disease is recorded.

Disease definitions

The statutes only rarely offer guidance on the definitions of diseases to be notified. For example, ophthalmia neonatorum is defined as "a purulent discharge from the eyes of an infant, commencing within 21 days from the date of birth"². Viral haemorrhagic fever is defined as "Argentine haemorrhagic fever (Junin), Bolivian haemorrhagic fever (Machupo), Chikungunya haemorrhagic fever, Congo/Crimean haemorrhagic fever, Dengue fever, Ebola virus disease, haemorrhagic fever with renal syndrome (Hantaan), Kyasanur forest disease, Lassa fever, Marburg disease, Omsk haemorrhagic fever and Rift Valley disease"². The Secretary of State for Health has recently accepted the recommendation of the Advisory Committee on the Microbiological Safety of Food that the following definition of 'food poisoning' should be adopted in England and Wales: "any disease of an infectious or toxic nature caused by or thought to be caused by consumption of food or water"³⁶. The same definition has been in use in Scotland for some years and has also been adopted by the World Health Organisation. Diseases such as typhoid and viral hepatitis, which would normally be covered by this definition, will continue to be notifiable as distinct diseases.

Changes to nomenclature

The use of notification data for long term trends has limitations. While data for some diseases are available and comparable over many decades, other diseases have not been notifiable continuously. Measles and German measles were notifiable between 1915 and 1919; measles rejoined the list in 1940 and rubella in 1988. The nomenclature has been changed for some diseases, and further information is required to derive meaningful trends. For instance, cerebrospinal fever was used to describe meningococcal meningitis in 1912; this was altered to meningococcal infection (which included infection of sites other than the meninges) in 1950. This was changed again in 1968 to be included under the heading of acute meningitis, when notifiers were asked to state the causal organism if known. In 1988 the term was changed to meningitis, with the cause to be specified, and meningococcal septicaemia (without meningitis) was added as a separate entity. Infective jaundice became notifiable in 1968 and was changed to viral hepatitis in 1988. Paratyphoid fever was made notifiable as enteric fever in 1889 when typhoid fever was notifiable separately; paratyphoid fever became notifiable as a single disease in 1968. Acute encephalitis was made notifiable in 1918 as acute encephalitis lethargica, and was changed to the current term in

1950. Acute poliomyelitis and acute polioencephalitis, notifiable since 1912, became known as acute poliomyelitis in 1950.

The future

The legal requirements for notification have changed little since the first Act of Parliament in 1889⁶. Many of the provisions of the 1984 Act¹ and the 1988 Regulations² would benefit from revision. The Department of Health published a consultation document on the review of the law on infectious disease control³⁷ in 1989. It is hoped that this opportunity will lead to the promulgation of clear guidelines for the collection of data for the effective surveillance and control of infectious disease in the twenty-first century. It is also important to consider how progress can be made towards standardisation of terms and practices throughout Europe.

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Malaria imported into the United Kingdom during 1991

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Summary

Two thousand, three hundred and thirty-two cases of human malaria were imported into the United Kingdom in 1991. There were twelve deaths; eleven of which were due to *Plasmodium falciparum* infection contracted in Kenya or West Africa. The annual total of cases of *P. falciparum* infection has increased throughout the last decade, reaching 1268 cases in 1991: over 90% of these were contracted in sub-Saharan Africa, whereas over 80% of *P. vivax* cases were contracted in South Asia. The largest category of infected travellers consisted of settled immigrants visiting friends and relatives in their country of origin.

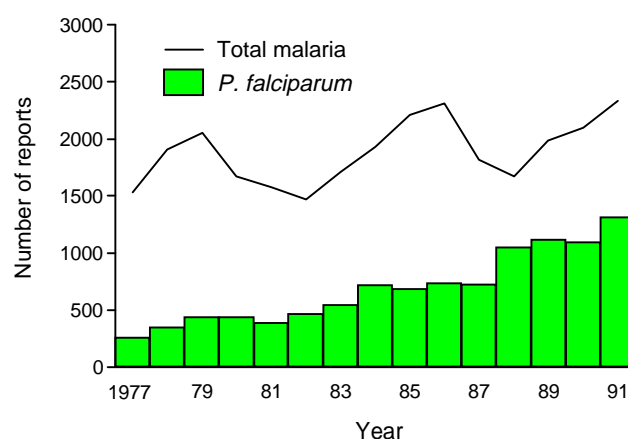
How many cases were there?

The number of imported cases of malaria reported in the United Kingdom each year has fluctuated around 2000 since 1977, with a tendency to rise rather than fall, and the total of 2332 cases recorded in 1991 is the highest since 1945. Of greater concern is the number of cases of *Plasmodium falciparum* infection which reached 1268, an increase of 20% on the previous year, reflecting a steady trend since 1981 of approximately 80 additional cases each year (Figure 1). *P. falciparum* alone comprises 54% of all malarias reported and, with the addition of 46 multiple infections, this rises to 56%. *P. falciparum* has become established as the predominant form of imported malaria in the last four years. It is the principal life-threatening form of malaria and is, therefore, a medical emergency requiring immediate treatment. This trend underlines the need for awareness of malaria by all doctors, especially those in primary care. Of the total of 2332 cases, 71 were imported into Scotland, three into Northern Ireland and 20 into Wales. The proportionately smaller number of cases reported from these parts of the UK is probably due to different patterns of travel and, possibly, a degree of under-reporting of cases.

Where do travellers catch malaria?

The source of infection in cases of imported malaria varies markedly with the species of parasite (Table 1). Fifty-nine per cent of cases were infected in Africa, 34% in Asia, and 1% each in Oceania and Latin America. Of the 1385 infected in Africa, 85% had falciparum malaria alone and another 2% had mixed infections, usually with *P. ovale*. By contrast, 95% of the 754 cases that contracted infection in South Asia had vivax malaria alone and only 5% had falciparum malaria (including mixed infections). Of the 36 cases with *P. falciparum* from South Asia, 13 were also infected with *P. vivax*. Although these comprise only 1.8% of the vivax cases from the region, it underlines the need to examine slides carefully for malaria, even when one species has been found, since the second species may determine the optimal therapy. In all, 2% of reported patients (47 cases) had mixed infections. Of these, 19 had *P. falciparum* and *P. vivax* combined, including 14 from Asia and two from Africa. All the other mixed infections recorded were from Africa. There were seven cases of *P. malariae* with *P. falciparum* and 20 of *P. ovale* with *P. falciparum*.

Figure 1 Malaria cases imported into the UK



One case had *P. vivax* and *P. ovale*. Seventeen of the 25 cases imported from Oceania had travelled to Papua New Guinea.

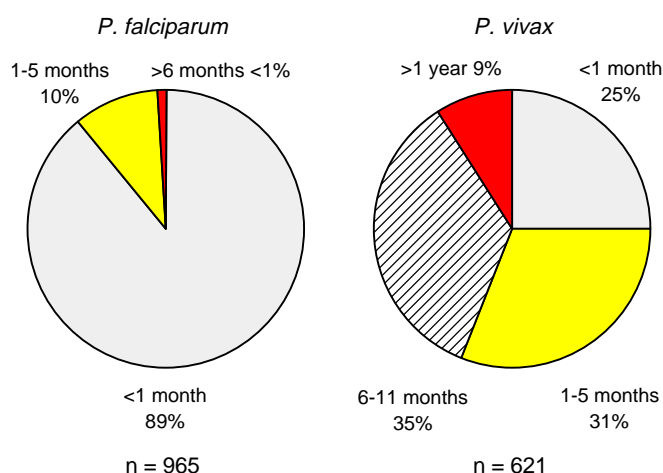
Ninety-one per cent of all falciparum malaria infections (falciparum alone or mixed with other species) were contracted in Africa. Of the 1170 infections with *P. falciparum* alone, 738 were contracted in West Africa (of which 639 [87%] were in Ghana and Nigeria) and 248 in East Africa (of which 155 [63%] were in Kenya). Six of the fatal infections were acquired in West Africa and five in Kenya. It is clear that Kenya, especially the Kenyan coast, and Nigeria and Ghana in West Africa, are particularly hazardous for travellers from the UK (Table 2).

What sorts of travellers get malaria?

Information about types of travel is important for planning preventive measures. Patterns of travel have changed substantially over the last decade, with a decrease in malaria in new immigrants and an increase in cases among settled immigrants and their children, who often contract malaria while visiting friends and relatives in their country of origin. For *P. falciparum* and *P. vivax*, just under 500 cases of each occurred in those visiting friends and relatives, compared with less than 100 cases of each in new entrants to this country (Table 3). The other large group of vivax cases occurred in foreign visitors to the UK.

Falciparum malaria is a substantial hazard for British holiday travellers and business travellers, as well as the much smaller number of UK citizens who live abroad but occasionally visit the UK (Table 3). All are accessible to health education in the UK, although the latter group is less frequently contactable. Among the cases of falciparum malaria reported from sub-Saharan Africa, 87% of new immigrants and foreign visitors to the UK had taken no prophylaxis; 44% of those visiting friends and relatives reported taking prophylaxis, whereas 74% of business travellers and over 85% of tourists claimed to have done so. The 542 cases of falciparum malaria among past and present immigrants indicate the need for special efforts to draw the

Figure 2 Interval between arrival in the UK and diagnosis of malaria



attention of ethnic minorities to the risks of malaria. The 147 cases of malaria in foreign visitors suggest that these groups require attention to health education in their countries of origin and on the incoming aircraft.

Information was available on the date of arrival in the UK, and on the date on which malaria was diagnosed, for 1688 cases with single species malaria infection (Figure 2). Eighty-nine per cent of falciparum malaria was apparent in the first month after reaching the UK and 99% within six months of arrival. By contrast, only a quarter of vivax cases were first diagnosed less than one month after reaching, or returning to, the UK.

Which travellers are most at risk of dying of malaria in the UK?

Twelve people died in Britain during 1991 from malaria contracted overseas. One death was due to rupture of the spleen following infection with *P. ovale* contracted in Ghana. This is a rare but known complication of *P. ovale* infection¹. The patient had an eight day history of fever but the diagnosis

Table 1 Imported malaria by parasite species and area where malaria was contracted

	<i>P. falciparum</i>	<i>P. vivax</i>	<i>P. malariae</i>	<i>P. ovale</i>	Mixed infections	Total
Africa	1170	43	19	123	30	1385
Central	29	3	–	3	1	36
East	248	21	3	22	6	300
Southern	73	7	–	7	1	88
West	738	9	16	78	16	857
Unspecified	82	3	–	13	6	104
Middle East	–	3	–	–	–	3
Asia	37	750	–	1	14	802
South and unspecified	23	717	–	1	13	754
South-East and Far East	14	33	–	–	1	48
Latin America	2	15	–	–	1	18
Oceania	3	21	–	–	1	25
Not known	56	31	2	9	1	99
Total	1268	863	21	133	47	2332

Table 2 Countries where imported malaria was contracted

Country*	Number of cases (%)	
<i>P. falciparum</i>		
Nigeria	383	(30)
Ghana	257	(20)
Kenya	155	(12)
Sierra Leone	44	(3)
Uganda	43	(3)
Tanzania	27	(2)
Malawi	25	(2)
Zambia	20	(2)
India	19	(2)
Zimbabwe	15	(1)
Zaire	14	(1)
Other/not known	266	(21)
Total	1268	(100)
<i>P. vivax</i>		
India	497	(58)
Pakistan	208	(24)
Papua New Guinea	14	(2)
Sri Lanka	11	(1)
Indonesia	10	(1)
Other/not known	123	(14)
Total	863	(100)

* Countries providing more than one per cent of cases.

of malaria was made at post-mortem examination. The remaining 11 deaths were due to *P. falciparum* and are considered in more detail below and in table 4.

Type of traveller

Most of the imported falciparum cases occurred in long-term UK residents of overseas origin who had visited relatives in areas endemic for malaria. They had a lower case fatality rate (2/462) than either visitors to the UK from overseas (2/268) or British tourists on holiday (3/204). Professional or business travellers had the highest case-fatality rate (4/100)

Table 3 Reasons for travel, listed by parasite species and selected countries

	Total number of cases	Species		Country of infection			
		<i>P. falciparum</i>	<i>P. vivax</i>	India/Pakistan	Ghana/Nigeria/Sierra Leone	Uganda	Kenya
Holiday travel	271	204	45	17	38	1	95
Business travel	142	100	35	7	38	4	13
Visiting friends and relatives in country of origin	1032	462	498	505	414	20	26
New immigrant	207	80	94	84	84	16	—
UK citizens living abroad	86	69	12	2	30	3	7
Child visiting parents abroad	7	7	—	—	2	—	—
Foreign student in UK	45	29	7	5	23	1	2
Foreign visitor to UK	247	147	80	69	113	10	13
Aircrew and armed services	23	14	6	—	2	—	11
Not stated	272	156	86	52	48	2	8
Total	2332	1268	863	741	792	57	175

although the overall numbers are small. One of the four fatalities in this group was a seaman who had a prolonged illness before being diagnosed in the UK.

Country, age and sex

The deaths associated with West Africa reflect the overall incidence of imported *P. falciparum* infections (58% compared with 55% of deaths) whereas, for East Africa, 20% of the cases contributed 5 of the fatalities (46%), all of whom acquired their malaria in Kenya. The 16-19 and the over 45 year age groups were over-represented among the fatal cases and there was an excess of males.

Prophylaxis

Forty-two per cent of the imported cases had taken no prophylaxis; the information was unknown in 22%. Sixty-four per cent of the fatal cases (7 of the 11) had taken no prophylaxis. Of the four that had taken prophylaxis, one was stated to have taken chloroquine with proguanil regularly, one took chloroquine alone, one took chloroquine with proguanil while overseas but stopped on returning to the UK a week before she became ill, and the drug used was not known in a fourth case. Chloroquine and proguanil had been taken by 17% of the total cases with falciparum malaria.

Delay in diagnosis

Information about the onset of infection may be unreliable or absent in fatal cases, while in non-fatal cases it may be more reliable, but absent almost as often. The data, although incomplete, do not tend to support the suggestion that delay in diagnosis is the major factor in determining a fatal outcome. Thirty-seven per cent of imported falciparum malaria cases were diagnosed within five days of onset of symptomatic disease compared with 7 (64%) of the fatal infections.

Other factors

One of the fatal cases (a teenage male) had recently undergone renal transplantation and was still on immunosuppressants in Nigeria, without prophylaxis, when he developed malarial infection. *Klebsiellapneumoniae* was isolated from his blood.

Table 4 Deaths from falciparum malaria

Country of infection	Sex	Age group (years)	Nationality	Occupation or reason for visit	Prophylaxis	Chronology (days)		
						Arrival to illness	Illness to diagnosis	Diagnosis to death
Ghana	M	50-59	Ghanaian/British	Visiting family	Proguanil/ chloroquine	<1	<1	6
Ghana	F	50-59	Ghanaian	Visiting family	Nil	<1	<1	<1
Kenya	M	10-19	Kenyan/Asian	On visit to UK	Nil	Not known	4	11
Kenya	M	20-29	British	Holiday visit to relatives	Nil	7	3	<1
Kenya	M	40-49	British	Working intermittently in Kenya	Drug not known	Not known	6	1
Kenya	M	50-59	British	On holiday	Nil	5	3	8
Kenya	F	50-59	British	On holiday	Proguanil/ chloroquine	7	6	<1
Nigeria	M	10-19	Nigerian	With renal transplant	Nil	<1	<1	2
Nigeria	M	30-39	British	Working in Nigeria/ visit to family	Chloroquine	Not known	Not known	3
Togo	M	50-59	Swiss/British	On business visit	Nil	1	1	1
West Africa	M	30-39	British	Merchant seaman	Nil	Not known	35	<1

A male in the 50-59 year age group, who was reported to have taken chloroquine and proguanil compliantly, developed a septicæmia associated with bronchopneumonia, diabetic ketoacidosis, cerebral infarction and severe atherosclerosis. He died despite exchange transfusion.

It is possible that chloroquine prophylaxis has become less effective with the spread of chloroquine-resistance, although the increase in cases of imported falciparum malaria from Kenya has not been as steep as the rise in number of UK travellers to that country.

The number of imported infections with *P. falciparum* has risen steadily since the 1970s but the case-fatality rate has fallen, from nearly 3% in 1977, 1978 and 1982, to the current level of less than 1%. The decline in the mortality rate for imported falciparum malaria is undoubtedly due to increased awareness of the importance of malaria on the part of the travelling public and medical practitioners, leading

to improved prophylaxis and more rapid diagnosis.

Acknowledgement

The quality of the data recorded owes much to Mrs M Blaze and Mrs V Smith of the Malaria Reference Laboratory, and the accuracy of the diagnosis to the technical staff of the laboratory.

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The clinical features of imported malaria

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Summary

Fifty-one patients with malaria were admitted to the Coppetts Wood Unit of the Royal Free Hospital in 1991. The majority had taken either no prophylaxis or a suboptimal regimen. This was especially evident among patients from ethnic minorities. The most common clinical feature of malaria is fever, which may present months and occasionally years after exposure which can lead to failure or delay in the diagnosis. Doctors should stress the need for travellers to endemic areas to take prophylaxis as well as mosquito avoidance measures. All patients for whom a diagnosis of malaria is considered should be referred for a same-day diagnostic test, preferably to a centre with appropriate expertise in tropical medicine.

Introduction

It is frequently not appreciated that malaria can present in a variety of ways, which may lead to misdiagnosis, delayed treatment and avoidable morbidity and mortality. We describe here the presenting features of the 51 cases of malaria referred to the Regional Department of Infectious and Tropical Diseases at the Coppetts Wood Unit of the Royal Free Hospital during 1991 and feature three cases that emphasise the diverse manifestations of this disease.

Case reports

Case 1

A nurse, who was born in Sierra Leone and lived in the UK, was admitted ten days after a trip to the Gambia, Sierra Leone and Senegal. She had taken only chloroquine as antimalarial prophylaxis. On the return flight she developed

vomiting, diarrhoea, abdominal pain, fever and cough. Her general practitioner diagnosed gastroenteritis and prescribed oral rehydration salts. On the sixth day of illness she was advised to return to work but continued to feel ill and on the tenth day saw an emergency doctor who diagnosed hepatitis and arranged admission to our unit.

The patient was jaundiced, drowsy and had a high fever (39.8°C) on admission. A blood film revealed that 8% of the red cells contained *Plasmodium falciparum* associated with low platelets ($109 \times 10^9/l$), uraemia (10 mmol/l) and deranged liver function (aspartate aminotransferase (AST) 84 IU/l, bilirubin 35 $\mu\text{mol/l}$). She slowly recovered after therapy with quinine, initially given intravenously, and Fansidar. Her course was complicated by severe anaemia, gastrointestinal haemorrhage and drowsiness requiring intensive monitoring and blood transfusion.

Case 2

A teenage male from the United States travelled to rural Kenya for a six week visit without vaccination or antimalarial prophylaxis. On his return he complained of fever, diarrhoea and malaise. After eight days of illness he was given ampicillin and chloroquine but failed to improve and was referred to our unit on the twelfth day. He was unwell, with rigors and fever (up to 40.5°C). A blood film showed 2.5% falciparum parasitaemia and a low platelet count ($68 \times 10^9/l$). He was given a standard five day course of oral quinine, with Fansidar as a single dose on the fifth day, following which he recovered uneventfully.

Case 3

During a two week holiday in Mombasa, Kenya, a female UK resident spent a few days on safari. She took proguanil and chloroquine prophylaxis for the correct period and was especially careful about insect avoidance measures. Three months later she developed fevers, fatigue, myalgia, headache and anorexia. These symptoms were severe and, at first,

Table 1 Malaria cases by country of infection

Country where infection was acquired	Species			Total
	<i>P. falciparum</i>	<i>P. vivax</i>	<i>P. ovale</i>	
Angola	1	–	–	1
Ghana	8	–	–	8
India	–	5	–	5
Kenya	2	–	–	2
Liberia	1	–	–	1
Nigeria	17	–	1	18
Pakistan	–	1	–	1
Papua New Guinea	–	1	–	1
Sierra Leone	2	–	–	2
South America (extensive travel)	–	–	1	1
Sri Lanka	–	1	1	2
Thailand	–	1	–	1
Uganda	2	–	–	2
Zaire	6	–	–	6
Total	39	9	3	51

Table 2 Antimalarial prophylaxis*

Ethnic background	Prophylaxis			Total
	Adequate [†] Number (%)	Inadequate Number (%)	None taken Number (%)	
Foreign nationals	–	2 (9)	20 (91)	22
UK residents:				
ethnic minorities	1 (5)	3 (15)	16 (80)	20
Caucasian	5 (63)	3 (27)	–	8
Total	6 (12)	8 (16)	36 (72)	50

* Recorded for 50 of the 51 cases.

[†] See text.

continuous, but later appeared at intervals of two to three days. She had been ill for five weeks before referral to this unit.

Initial examination revealed hepatosplenomegaly, anaemia and no fever, although the axillary temperature subsequently rose to 37.5°C. The haemoglobin was 8.8 g/dl, falling later to 7 g/dl; the platelet count was $36 \times 10^9/l$ and 2% of the red cells contained *P. vivax* trophozoites. She recovered slowly after therapy with chloroquine for three days followed by primaquine for two weeks, and a two unit blood transfusion.

Malaria admissions in 1991

Fifty-one cases (32 male, 19 female) of malaria were confirmed in patients whose ages ranged from 6-64 years (mean 32 years). Twenty-two patients were foreign nationals newly arrived in the UK. Of the 29 UK citizens, 20 were from ethnic minorities and had acquired malaria after a visit to their country of origin; the remainder were Caucasians who had been infected during travel abroad. Table 1 shows the parasite species and the country where infection was acquired for the 51 cases. No infections due to *P. malariae* were seen in 1991. The prophylaxis taken by each patient is described in table 2. Adequate prophylaxis was defined as that recommended by the 1989 meeting of malaria experts¹ – ie, in adults, chloroquine 300mg weekly with either proguanil 200mg daily or Maloprim one weekly, or mefloquine 250mg weekly for visits less than three weeks, all continued for four weeks after return (3 weeks for mefloquine).

Nine patients (18%) experienced a delay in referral of one to five days after the first consultation. The diagnosis was not initially considered in four of these cases, three of whom presented within three weeks of arrival from an endemic area. Three of these patients were treated with antibiotics for incorrect clinical diagnoses. In five of eight cases for whom a malaria film was requested by the general practitioner, the result was delayed by 1-2 days. By the time of referral to this unit the diagnosis was correctly considered by 48 of the referring doctors – the remaining three diagnoses being gastroenteritis, appendicitis and fever of unknown cause (with no travel history obtained).

Clinical features

Thirty-five of the patients with falciparum malaria presented within two weeks of arrival from the endemic area (median 5 days) – in the remaining four cases there was a further delay of between one and 90 days. *Ovale* and *vivax* malarias

Table 3 Frequency of presenting symptoms

Symptom	Number of patients	(%)
Fever	49	(96)
Rigors/shivering	23	(45)
Headache	23	(45)
Vomiting	18	(35)
Arthralgia/myalgia	12	(24)
Sweats	9	(18)
Diarrhoea	8	(16)
Cough	7	(14)
Malaise	5	(10)
Abdominal pain	5	(10)
Periodic fever*	4	(8)
Sore throat	1	(2)
Loss of consciousness	1	(2)
No symptoms [†]	2	(4)

* Periodic (tertian) fever was only found in patients with benign malarias (3 vivax, 1 ovale). [†] 2 asymptomatic children had positive blood films when investigated after a sibling was admitted with symptomatic malaria.

presented much later (median 60 days, range 20-310). Illness had lasted less than three days in 15 patients (30%), 3-5 days in 18 (36%) and more than five days in 17 (34%). The duration of illness was not recorded in one case. Three of the four cases of severe falciparum malaria (parasitaemia >5%²), and all the cases of benign malaria, had been ill for 5 days or more.

The presenting symptoms are listed in table 3. Apart from a raised temperature and obvious malaise, physical signs were uncommon but included splenomegaly in six cases (12%), jaundice in one (2%) and loss of consciousness in one case (2%). Severe falciparum malaria was diagnosed in four patients. One had cerebral malaria, renal failure, pneumonitis and 35% parasitaemia and has been reported previously³; the other three cases had parasite levels of 5-10% but without complications other than anaemia and thrombocytopenia. The remaining patients with falciparum malaria had parasitaemias of 1-4% in seven cases (18%) and less than 1% in 30 cases (75%). One patient with a parasite count of less than 1% suffered transient hypoxia after aspiration of vomit. Other common abnormalities included a low haemoglobin and platelet count, raised serum bilirubin and increased serum aminotransferase levels (Table 4).

Treatment

Five patients with falciparum malaria had taken antimalarial drugs as self-treatment prior to admission. After admission, falciparum malaria was treated with a five-day course of quinine (10 mg/kg up to 600 mg per dose, 2-3 times per day). The twice-daily course was used if the higher dose caused unacceptable side effects after two days of therapy. The intravenous route was used initially if parasite levels were 5% or more or where there was significant vomiting, diarrhoea or complications. Pyrimethamine/sulphadoxine (Fansidar) was given (3 tablets in adults) with the final quinine dose. This was omitted and replaced by a further 5-9 days of quinine in pregnant women or individuals intolerant to Fansidar (including those with G6PD deficiency). Benign malarias were treated with chloroquine in standard doses⁴. Primaquine was administered to prevent relapse (15 mg/day for two weeks) in the absence of G6PD deficiency. There

were no treatment failures or relapses in 1991 although a relapse was detected in the previous year in a man with falciparum malaria acquired in Guinea. He responded to quinine and tetracycline. All patients admitted in 1991 recovered completely apart from a man with cerebral malaria who was left with permanent brain damage³.

Discussion

Malaria causes considerable morbidity and mortality, much of which is avoidable. Over the last ten years, around 2000 imported cases have been reported each year in the UK. There has been a steady rise in reports of the falciparum type, which now accounts for more than 50% of all isolates (see accompanying article⁵). There were 68 reported deaths during this period with 12 occurring in 1991 and five in the first half of 1992⁶. A review of the 12 deaths in 1991 indicates that at least nine patients had taken either inappropriate or no prophylaxis and three were diagnosed too late⁵.

Prophylaxis

Prophylactic antimalarial drugs reduce the incidence of malaria, as demonstrated by the fact that only six (12%) of our patients had taken adequate prophylaxis. The disease is also likely to be less severe in these cases⁷. However, currently recommended prophylactic drugs are only active against the asexual erythrocytic malarial trophozoites and have no appreciable action against the dormant (hypnozoite) liver stage of vivax and ovale malarias. Travellers should therefore be warned that benign malarias can arise many months after prophylaxis is ended (eg, case 3) and should be instructed in mosquito avoidance to reduce the risk of infection.

Our finding that 80% of patients who were UK residents from the ethnic minorities had taken no antimalarial prophylaxis is cause for concern. Most of these individuals had been visiting their country of origin and discussions suggest that many of them felt they still had some immunity. They may also have perceived malaria to be a benign disease having seen it treated at outpatient clinics in endemic areas. The need for antimalarial prophylaxis needs to be emphasised to this section of the UK population.

Diagnostic problems

Some relatively common presentations of malaria may be misleading. The 35% of cases with vomiting and 16% with diarrhoea (including cases 1 and 2) may be thought to have gastroenteritis. Viral infection, especially influenza, is also a common misdiagnosis². This is unsurprising considering the significant numbers reporting arthralgia or myalgia (24%), cough (14%) and sore throat (2%). The physical sign most likely to cause confusion is jaundice, found in 2% of our cases, which may be interpreted as signifying viral hepatitis (case 1).

Malaria may also be excluded inappropriately on the assumption that certain features must be present. Splenomegaly (12%) and periodic fever (8%) were relatively uncommon findings in this series despite the prominence they have sometimes been given in the past. The only assumption that should be made is that the feverish patient who has been to endemic areas any time within the past five years may have malaria, particularly if the travel was within the previous 12 months, irrespective of prophylaxis or accompanying symptoms.

Eighteen per cent of cases, including the majority of the

Table 4 Common laboratory abnormalities in malaria

	Number of cases	Number showing abnormality (%)			
		Low platelets*	Low haemoglobin*	Raised bilirubin*	Raised AST/ALT*
<i>P. falciparum</i>	39	19 (49)	9 (23)	10 (26)	8 (21)
<1%	28	9 (32)	4 (14)	6 (21)	5 (18)
≥1%	11	10 (91) [†]	5 (45)	4 (36)	3 (27)
<i>P. vivax</i>	9	5 (56)	2 (22)	4 (44)	–
<i>P. ovale</i>	3	3(100)	1 (33)	2 (67)	–
Total	51	27 (53)	12 (24)	16 (31)	8 (16)

* Normal values in our laboratory are: platelets 150-450 x 10⁹/l; haemoglobin, males 13-18 and females 12-16 g/dl; bilirubin 5-17 µmol/l; AST and ALT <40 IU/l. [†] A low platelet count was found significantly more often with *P. falciparum* parasitaemias of ≥ 1% than with those <1% (p<0.001 on Chi square test).

seriously ill patients, experienced a delay in diagnosis. In some, this was due to 'misleading' symptoms. The presentation of disease up to three months after travel in a small number of falciparum cases and up to ten months later in benign cases was also a possible explanation (eg, case 3). However, the inappropriate use of routine diagnostic blood films, noted in 8% of patients, is an avoidable cause of delay. Patients with falciparum malaria can deteriorate rapidly² and a diagnostic film should only be requested if the result can be guaranteed the same day. Delays may occur during transport of the specimen, requests may be made late in the day or at weekends, or there may be communication problems. Research at this unit has shown that low grade parasitaemia may be missed if a film is made more than 24 hours after the blood sample is taken⁸. Most infectious and tropical disease units will offer a same-day diagnostic service for malaria on request, and at least one unit offers day-care facilities⁹. Malaria may also be missed if only one blood film is examined. Four of our patients had previously had a negative film. This may be due to an initial low parasitaemia, but can also be due to inexperience or delay in preparing the film⁸, or poor staining. A new diagnostic technique using acridine orange and fluorescence microscopy (QBC system) may improve diagnostic sensitivity but is still less sensitive than a thick film technique, does not speciate the malaria, and requires special equipment^{10,11}.

Many patients had no abnormal blood findings other than a positive film. Thrombocytopenia proved to be a valuable laboratory correlate with the more severe cases of falciparum malaria, and also the benign malarias, and when found should lead to greater efforts in pursuing a diagnosis of malaria. The same applies to a lesser extent to the findings of anaemia, bilirubinaemia and raised serum amino-transferase levels. However, the latter abnormalities emphasise the potential for this disease to mimic viral hepatitis, as in case 1.

Despite the potential problems and delays in reaching a diagnosis, the majority of patients responded well to therapy and only one patient suffered long term sequelae. The malaria mortality rate in this country is currently about 1 in 100 for falciparum malaria⁵. Greater efforts are required to emphasise to travellers the need for prophylaxis and mosquito avoidance, and to encourage early referral of feverish cases to centres with appropriate expertise. These measures would reduce the avoidable incidence of death

and severe illness, which often affects relatively young and active people.

Acknowledgement

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The resurgence of scabies

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Summary

The prevalence of scabies infection shows a cyclical pattern with a periodicity of 10-30 years. Various sources indicate that a major increase has been underway since 1991 and CDSC has received several reports of outbreaks in schools, hospitals and nursing homes in recent months. This brief review outlines the clinical features, diagnosis, epidemiology and management of this time-honoured infection.

Introduction

Human scabies is a parasitic disease of the skin caused by infection with the mite *Sarcoptes scabiei* and the ensuing allergic response. It has been documented to affect man for at least 3000 years. The microscopic mites penetrate the epidermis causing tiny, characteristic, linear burrows that may be seen in the skin. Eggs are laid in the epidermis and hatch after 3-4 days. The emerging larvae then appear on the surface of the skin before excavating new tunnels. Transmission from one person to another usually occurs during close and prolonged skin contact which includes holding hands. Outbreaks of scabies have been reported in hospitals, particularly on geriatric, psychiatric and long-stay wards, in AIDS units, and in residential homes for the elderly, where patients and staff may be affected¹⁻³.

Clinical features

Classical scabies

The clinical picture in healthy individuals is the appearance of raised burrows, or small red, slightly elevated, papules or vesicles, particularly on the wrists, back of the hands and between the fingers. Further spread is usually confined to the elbows, armpits, beneath the breasts, waist, groin, genitalia, buttocks, knees and ankles. The incubation period is 2-6 weeks before the onset of itching in those infected for the first time but symptoms may occur 1-4 days after re-exposure. Symptoms are due to an allergic reaction to the burrowing mite and include itching, particularly at night, although the rash is almost invisible. The local effects of the parasite may be aggravated by trauma from scratching which can result in secondary bacterial infection.

Atypical and crusted scabies

Immunocompromised persons (eg, those with AIDS and patients receiving immunosuppressive therapy) and the very young or elderly, may present with an **atypical** form with minimal signs or, rarely, a severe **crusted** form (sometimes referred to as 'Norwegian' scabies)⁴⁻⁶. When the immune response is impaired, between thousands and millions of mites may be present compared with only a few (10-20) when healthy persons become infected^{5,6}. Patients with these atypical forms are highly infectious but may not itch, and mites may be present anywhere on the body, including the head⁷. Delay in diagnosis may lead to widespread dissemination including staff and family contacts⁸. Recent reports of scabies in patients with HIV infection have demonstrated the potential for development of the severe crusted form; associated nosocomial outbreaks, and life-threatening secondary infection^{2,3,6,9}.

Diagnosis

The diagnosis of classical scabies is usually based on the history and clinical appearance but may be confirmed by identification of the mites, or their faecal pellets, in skin scrapings of burrows or papules. The eggs are usually present in the deeper parts of the burrows. Identification involves placing a drop of sterile mineral oil on the lesion and on a sterile scalpel blade. The lesion is scraped until tiny flecks of blood appear in the oil. The sample is then placed on a slide or cover-slip and examined under a microscope for the presence of mites, eggs or faecal pellets. If skin scrapings fail to establish the diagnosis, or cannot be performed, the presence of burrows, complaints about itching which is worse at night, and clustering of cases, are highly suggestive of scabies infection.

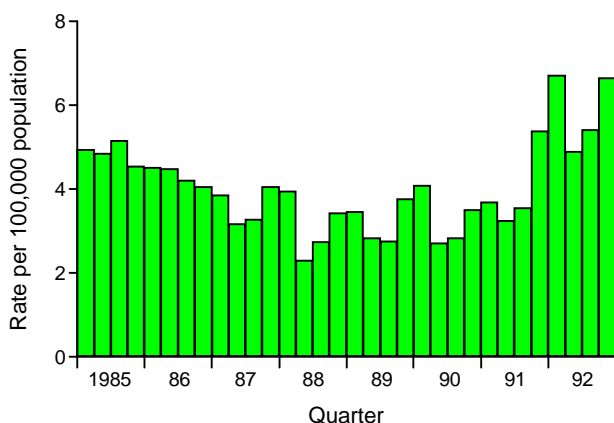
Epidemiology

Scabies has a worldwide distribution and is endemic in many developing countries where it mostly affects children and young adults, and is associated with poor hygiene and overcrowding. The incidence of scabies in a population fluctuates over time with epidemics occurring in 10-30 year cycles⁵. In England and Wales, the incidence of scabies in the early 1970s was about twice that seen in recent years but there is evidence from recent reports that the incidence is increasing again⁷. The number of patients with scabies referred to dermatology clinics in Cumbria in 1990-91 was similar to that seen in the previous epidemic years of 1944-46 and 1964-66¹⁰ and the Medical Entomology Centre in Cambridge reports a ten-fold increase in enquiries in recent months.

Scabies is not a notifiable disease in England and Wales but data from the sentinel practice scheme of the Royal College of General Practitioners show an increase in the number of episodes of scabies infection reported throughout 1992 (Figure 1). About one third of cases diagnosed in general practice occur in children (aged 0-14 years) and over half are in the age group 15-44 years¹¹. The number of scabies diagnoses made in genito-urinary medicine clinics and recorded by the Department of Health gradually declined up to 1988 but diagnoses of pediculosis pubis and scabies have been aggregated into a single category since then.

CDSC has received reports of outbreaks of scabies in elderly residential homes, mental institutions, schools and hospitals in the United Kingdom and Ireland during the last few months. One district health authority has recently investigated three institutional scabies outbreaks. Two of these occurred in residential homes for the elderly (20 and 10 confirmed cases, respectively) and the third involved at least five cases in a mental institution. Cases were scattered throughout the institutions in each of these outbreaks and it was necessary to treat all residents and exposed family members to control transmission (approximately 300 persons in total). The North East Thames region investigated a scabies outbreak at a junior school involving 10 cases which was successfully controlled by targeting treatment to close contacts and family members via general practitioners. Hospitals have also been affected as demonstrated by reports from one public health laboratory of scabies outbreaks at three hospitals involving at least 37 cases (17 in patients and 20 in staff) which required the treatment of all patients and

Figure 1 Mean weekly incidence (by quarter) of clinical episodes of scabies reported by the RCGP sentinel practice scheme



staff on the affected wards. In one of these hospital outbreaks, six patients developed secondary infection with group A haemolytic streptococci.

Treatment^{4,5,12,13}

Suitable treatment regimens have been reviewed recently^{4,12}. Several scabicides are available and the choice depends on factors such as the age and medical condition of the patient. Permethrin, malathion and lindane are used most commonly. Permethrin is relatively new, has very low toxicity, high cure rates, short treatment time and has gained increased acceptance as a first line treatment. Malathion is pleasant to apply and effective with few contraindications for its use. It has been the treatment of choice for infants, and during pregnancy. Lindane is still widely used and effective but relatively toxic and should be avoided during pregnancy or breast feeding, in young children, and in patients with low body-weight or a history of epilepsy.

Aqueous lotions give better coverage than creams and are less irritating than alcoholic preparations. The scabicide lotions should be applied to cool dry skin over the entire body and left on for several hours (8-24 hours, depending on the preparation). In classical scabies the head need not be treated, as mites are not normally found there in otherwise healthy people. However, for crusted or atypical cases, those in children less than two years of age, or cases in which previous treatment has failed, every inch of skin should be covered, with special attention to the fingernails and ears. If hands or other skin areas are washed during the treatment period the treatment should be reapplied.

It is important that close contacts and family members are treated at the same time as they are often infected but may still be in the incubation phase. A single treatment is often effective, especially for contacts, but a second treatment in 7-10 days is often used for symptomatic cases and those with severe disease, as eggs are relatively more resistant than mites. Itching persists for several days after the infection has been eliminated and does not, necessarily, indicate treatment failure. Antipruritic treatment such as calamine may be helpful.

Management of outbreaks

In institutional outbreaks, patients and long-term residents should be examined in an attempt to identify the index case, and adequate barrier nursing methods should be employed.

Every effort should be made to confirm the diagnosis by identification of the mite in a sample of affected individuals. Once confirmed, it is important to provide accurate information for staff, patients and families. All individuals on an infected ward or in a residential home, all medical and nursing staff, and the families of symptomatic members of staff, should be treated with a scabicide. Particular attention should be paid to the nails as mites may persist subungually. Bedding, clothing and towels are at risk of indirect transfer of mites only when contaminated immediately beforehand, and should be laundered normally using hot water and a dryer.

Patients seen by dermatologists are probably only a minority of the cases present within the community, and successful intervention may require concerted control methods^{14,15}. It is important to educate the public, as well as the medical and nursing professions, about the life history of the scabies mite, modes of transmission, and methods of diagnosis and treatment. Effective control is dependent on early diagnosis, adequate treatment of infected patients and contacts, and the prevention of further spread.

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'COVER' (Cover of vaccination evaluated rapidly): 24

Methods

Data were collected at the beginning of November 1992 for quarterly cohorts whose youngest member had reached the target ages for completion of immunisation: 18 months for the third dose of diphtheria (D3) and pertussis (P3) vaccines and 24 months for measles. For D3 and P3, data were also requested for quarterly cohorts whose youngest member had reached a lower target age of 12 months. The cohorts studied were those born in July to September 1991 (for D3 and P3 by 12 months), January to March 1991 (for D3 and P3 by 18 months) and July to September 1990 (for measles by 24 months).

Results

Data were received from 189 (95%) of the 198 districts in England, Wales and Northern Ireland (Table). All districts contributed data in nine English regions, Wales and Northern Ireland. In all other regions (except Mersey) at least 80% of districts participated. The average cover by 12 months was 93% for D3 (district range 79-99%) and 90% for P3 (district

range 77-97%). Cover by 18 months was 95% for D3 (district range 84-99%) and 91% for P3 (district range 81-96%). For measles, average cover was 93% (district range 74-98%).

Comment

Vaccine coverage for D3 and P3 (at both 12 and 18 months) is unchanged since the previous report. Coverage for measles has improved by 1%, despite the adverse publicity about recent changes in the supply of vaccine and reports of vaccine shortages. However, the impact of these problems is most likely to be on cohorts of children evaluated in the next two COVER reports. The 95% target was achieved by 131 districts for D3 (at 18 months), by 31 districts for P3 (at 18 months) and by 67 districts for measles. All but one district achieved 80% or greater coverage for all the sentinel antigens.

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Table Diphtheria and pertussis vaccination at 12 and 18 months, and measles vaccination at 24 months: November 1992

Region	Number of participating districts (total)	% coverage at 12 months*		% coverage at 18 months*		% coverage at 24 months*
		D3 by evaluation date	P3 by evaluation date	D3 by evaluation date	P3 by evaluation date	Measles by evaluation date
England:						
Northern	16 (16)	95 (90-99)	92 (88-97)	96 (93-99)	92 (88-95)	94 (91-98)
Yorkshire	14 (16)	93 (89-99)	90 (86-96)	95 (93-99)	91 (86-95)	93 (91-96)
Trent	10 (12)	93 (89-96)	91 (87-93)	94 (91-98)	91 (89-95)	92 (88-96)
E Anglia	7 (8)	96 (93-99)	94 (90-97)	97 (94-99)	94 (91-96)	95 (91-98)
NW Thames	13 (13)	92 (86-98)	90 (84-95)	94 (86-98)	91 (86-96)	92 (85-97)
NE Thames	15 (15)	92 (86-98)	90 (83-96)	95 (90-99)	92 (88-98)	92 (86-98)
SE Thames	15 (15)	92 (79-97)	90 (77-95)	94 (84-98)	91 (82-95)	91 (74-97)
SW Thames	13 (13)	92 (87-98)	90 (86-96)	94 (89-98)	92 (86-96)	90 (83-97)
Wessex	10 (10)	96 (91-98)	93 (86-95)	96 (92-99)	94 (89-96)	95 (93-98)
Oxford	7 (8)	95 (91-97)	93 (89-95)	96 (95-97)	93 (92-94)	94 (93-97)
S Western	9 (9)	95 (93-98)	93 (92-94)	96 (94-99)	93 (92-96)	95 (92-98)
W Midlands	21 (21)	93 (82-97)	90 (80-95)	95 (84-98)	91 (83-96)	93 (87-96)
Mersey	7 (10)	90 (86-97)	86 (82-94)	92 (87-97)	88 (82-94)	89 (83-96)
N Western	19 (19)	93 (85-97)	90 (82-93)	95 (87-98)	90 (81-95)	92 (83-97)
Wales	9 (9)	95 (93-96)	89 (87-93)	96 (93-98)	88 (87-92)	92 (89-97)
N Ireland	4 (4)	94 (92-96)	90 (89-93)	96 (95-98)	91 (89-94)	93 (90-96)
Total	189 (198)	93 (79-99)	90 (77-97)	95 (84-99)	91 (81-96)	93 (74-98)

* The district range is given in brackets

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