
MAIN STORIES THIS WEEK:

- ▣ [Outbreak of *Shigella sonnei* in north London](#)
- ▣ [Outbreaks of pertussis \(whooping cough\) in primary school children](#)
- ▣ [Improving patient care by reducing the risk of hospital acquired infection: a progress report from the National Audit Office](#)
- ▣ [Statutory notification of tuberculosis](#)
- ▣ [Department of Health update on CJD](#)
- ▣ [An integrated approach to human transmissible spongiform encephalopathies \(prion diseases\)](#)

Immunisation:

- ▣ [Laboratory reports of invasive meningococcal infections, England and Wales: weeks 09-12/2004](#)
- ▣ [Laboratory Reports of *Haemophilus influenzae* by age group and serotype, England and Wales: April to June 2004](#)
- ▣ [Laboratory reports of hepatitis A in England and Wales: January to March 2004](#)
- ▣ [Laboratory reports of acute hepatitis B infection by age group and sex, England and Wales: January to March 2004](#)
- ▣ [Laboratory reports of hepatitis C infection by age group and sex, England and Wales: January to March 2004](#)
- ▣ [Serious hazards of transfusion \(SHOT\), 2003 report](#)







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News

Last updated: **22 July 2004**Next update due: **29 July 2004**

-  [Outbreak of *Shigella sonnei* in north London](#)
-  [Outbreaks of pertussis \(whooping cough\) in primary school children](#)
-  [Improving patient care by reducing the risk of hospital acquired infection: a progress report from the National Audit Office](#)
-  [Statutory notification of tuberculosis](#)
-  [Department of Health update on CJD](#)
-  [An integrated approach to human transmissible spongiform encephalopathies \(prion diseases\)](#)

Outbreak of *Shigella sonnei* in north London

There has been a recent increase in the number of cases of watery or bloody diarrhoea, with or without vomiting, among a religious community in north London.

The first confirmed cases of *Shigella sonnei* were seen within families in May and June. This was followed by a cluster of clinical cases in a nursery school in early July, with 20 out of 34 children and five out of seven staff known to be affected. Cases of *Shigella sonnei* have now also been detected in a number of primary schools. The spread of the outbreak suggests significant person-to-person transmission.

To date, there have been 19 culture-confirmed cases. The Health Protection Agency's Laboratory of Enteric Pathogens has received ten isolates and has typing results on seven. All seven *Shigella sonnei* isolates have the same phage type pattern and the same antimicrobial resistance profile. There may be an association with an outbreak in the same religious community in Greater Manchester.

Information about the outbreak and control measures has been sent to local general practitioners, head teachers, and the community.

CCDCs are asked to ensure that specimens are taken from potential cases to confirm the organism responsible as some cases of *E. coli* O157 have also been reported in the same community. Laboratories should send isolates for typing to the Laboratory of Enteric Pathogens at HPA Colindale.

People aware of additional cases that are associated with the north London outbreak should contact Graham Fraser by email: <graham.fraser@hpa.org.uk>. If any other clusters of *Shigella sonnei* infection are detected, the local Health Protection Unit should be contacted, as well as the Environmental and Enteric Diseases Department at the Health Protection Agency's Communicable Disease Surveillance Centre.



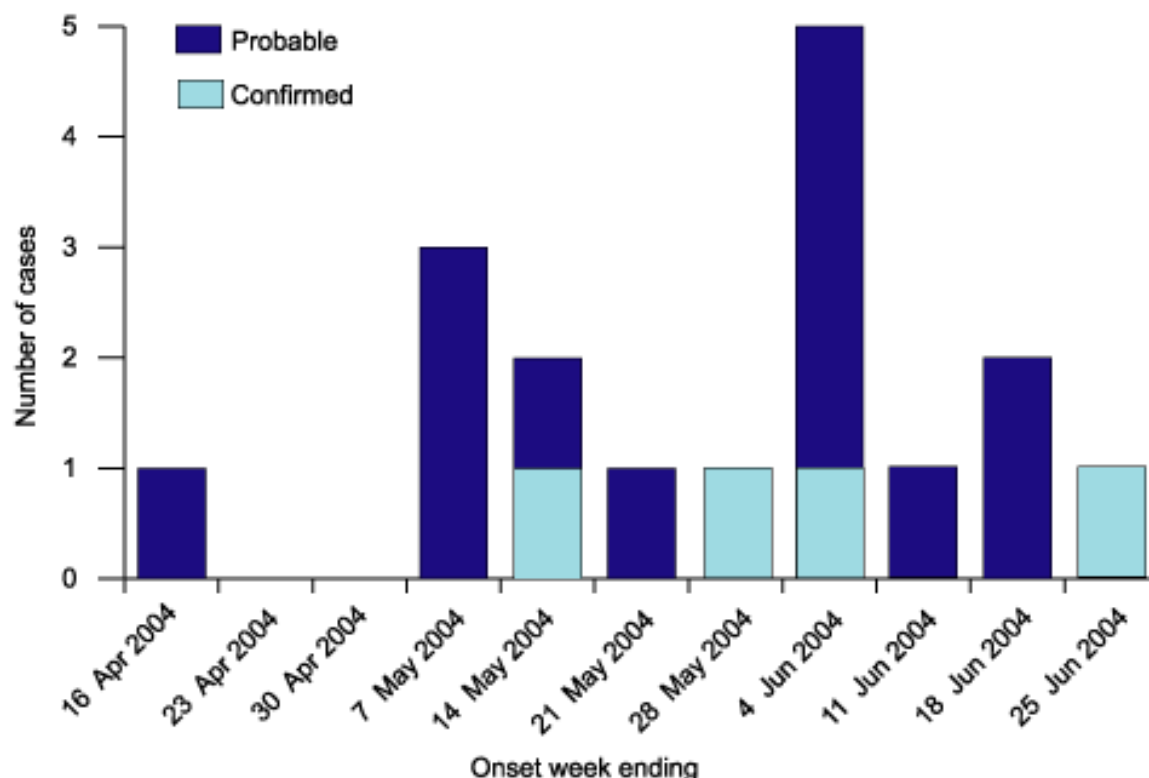
Outbreaks of pertussis (whooping cough) in primary school children

The Health Protection Agency's Health Protection Units (HPUs) have recently reported outbreaks of pertussis in primary schools in different parts of England. These have affected schools in Oxfordshire, and Leicestershire. Many children have presented with only prolonged cough, rather than classical pertussis with paroxysms (a repetitive series/attack of coughing) and whoop. Cases have been confirmed by serology, culture, and/or PCR carried out by local laboratories and the HPA's Respiratory and Systemic Infection Laboratory (RSIL). Cases have been confirmed in both children and adults. Most of the children are aged 8 years or over.

In Oxfordshire, the dates of onset are between 19 May and 29 June 2004. Three of the 12 cases are laboratory confirmed. Three of the cases are males and the mean age of all 12 cases is 9 years (standard deviation = three years). Only one child has a record of having received a fourth dose of pertussis vaccine.

A primary school in Leicester with 190 students aged four to eleven years contacted the local health protection team in June 2004 because of concerns that a large number of students had been coughing for several weeks and one student had been diagnosed with probable whooping cough in hospital. Members of the team visited the school and questioned parents of ill children and obtained specimens from them. Seventeen people (including thirteen students, one member of staff, and three other family members) were reported to have had symptoms of illness lasting two months involving paroxysmal coughing followed by vomiting. One child had been heard to whoop. Over half of the cases were aged between 8 and 12 years with a range from 1 to 32 years, with the earliest date of onset being 16 April (figure). Four cases have been confirmed by serology, including one child who had received diphtheria, tetanus, and acellular pertussis (DTaP) as the pre-school booster. Questioning other schools and general practices in the area did not identify any other cases during the first week.

Figure Epidemic curve, Leicester school pertussis outbreak



Control measures agreed by the outbreak control team included information to parents and general practices in the area to raise awareness and to advise early treatment and exclusion of suspected cases and prophylaxis of vulnerable household contacts. As this outbreak appeared to be associated with this school only, it was agreed to offer booster immunisation with DTaP to all children who had not received this as a pre-school booster.

Twenty-one probable cases have since been identified in five other primary schools (confirmed cases in two) and two secondary schools in the area, with onset dates from early May onwards. Transmission will be interrupted because schools are breaking up for the summer holidays. Pertussis, however, tends to peak seasonally in late summer and early autumn, so clinicians and health protection specialists need to be aware of the possibility of further outbreaks.

A pre-school pertussis booster given to children aged between 3 and 4 years of age was added to the United Kingdom (UK) childhood immunisation programme in November 2001. Most children born from 1998 onwards will have benefited from the booster, and some children born between 1996 and 1998 may have also received a fourth dose of pertussis vaccine. Children born before 1996 (now aged 8 years and over) are unlikely to have received more than three doses of pertussis vaccine in infancy and so are less protected than younger children.

Pertussis remains at historically low levels in the UK, although it is under-notified and under-diagnosed (1,2). National surveillance for whooping cough is managed by Prof Elizabeth Miller in the HPA's Communicable Disease Surveillance centre. In older children and adults, serology is a particularly useful method of diagnosis as patients often present after coughing for some weeks when culture and PCR are usually negative (3). Serology, and PCR are available from the RSIL for clinicians in England and Wales through Tim Harrison, tel: 020 8200 6906 email: <tim.harrison@hpa.org.uk>, or Robert George, tel: 020 8200 4400, email: <robert.george@hpa.org.uk>.

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Improving patient care by reducing the risk of hospital acquired infection: a progress report from the National Audit Office

The second National Audit Office (NAO) study *Improving patient care by reducing the risk of hospital acquired infection: a progress report* has been published (1), and presents its findings as a progress report on the factors influencing the Department of Health's (DH) drive to improve patient care by reducing the risk of hospital acquired infection. This was assessed by determining the extent to which the Committee of Public Accounts (CPA) recommendations based on the first NAO report (2) had been implemented, by measuring any improvements in the management and control of hospital acquired infection in NHS acute trusts, and any discernible changes in patient outcomes. In addition, the CPA had requested that the NAO examine how other countries are addressing these issues.

The first NAO report on the management and control of hospital acquired infection in acute hospitals in England led the CPA to conclude in November 2000 that the lack of control on the extent and costs of hospital acquired infections was impeding NHS trusts in targeting activity and resources to best effect (2). In addition, the Committee said that a 'root and branch' shift towards prevention would be needed at all levels of the NHS if hospital acquired infection were to be kept under control.

The overall conclusion from the second NAO report was that the implementation of the recommendations from the first NAO report and those from the CPA had been patchy. The progress report provides evidence that hospital acquired infection now has a much higher profile and, at the central strategic level, has been accorded a higher priority with the launch of a number of key requirements and that there has been 'notable progress at trust level in putting the systems and processes in place and in strengthening infection control teams. Wider factors, however, continue to impede good infection control practice and there has been limited progress in improving information on the extent and costs of hospital acquired infections'. It points out that progress in preventing and reducing the number of hospital acquired infections is dependent on changing staff behaviour, but change continues to be constrained by the lack of data, limited progress in implementing a national mandatory surveillance programme that meets the needs of the NHS, and a lack of evidence of the impact of different intervention strategies. The report also highlights that:

- at trust level, the pursuit of other key policies and priorities can adversely affect attempts to improve infection control. This is made harder by the emergence of strains of multi-resistant bacteria, increasing antibiotic resistance, and an increase in the number of outbreaks of infection with organisms such as norovirus by the trusts;
- notwithstanding local improvements in information, the NHS still lacks sufficient information on the extent and cost of hospital acquired infection, making it difficult to quantify any changes in NHS Trust infection rates. It argues that feedback of specific local infection rates to clinical staff is vital in engaging them in reviewing and changing their practice, and that the new mandatory national surveillance schemes do not currently enable clinicians to identify and reduce risks within their own specialty. In the absence of such ownership and access to such data, hospital acquired infection is still perceived as a problem for the infection control team to deal with. Consequently, some of the issues identified as barriers to effective infection control practice (as outlined in the first NAO report on the management and control of hospital acquired infection) are in acute NHS Trusts in England (2).
- further action, using a range of approaches, is required to change staff behaviour to reduce the risks of hospital acquired infection. Considerable improvements could still be made in: the coverage of education and training in infection control to all groups of staff, (particularly doctors); compliance with guidance on issues such as hand hygiene, catheter care and aseptic technique; antibiotic prescribing in hospitals; hospital cleanliness; and consultation with the infection control team on wider trust activities such as new build projects. It notes that despite the increasing profile of hospital acquired infection and the publication of guidelines on the measures required to contain the problem, there continues to be a lack of compliance with good infection control practices.
- the study of international comparisons showed that all the countries reviewed had established surveillance programmes. Comparison of national prevalence of hospital acquired infection showed rates between 4% and 10% with the United Kingdom at 9%, but variations in protocols and numbers and frequency of hospital participation make direct comparison unreliable. Data for 2002 from the European Antimicrobial Resistance Surveillance System (EARSS) on levels of MRSA bloodstream infections as a proportion of all *Staphylococcus aureus* bloodstream infections show that the UK is among those with the highest levels in Europe.

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Statutory notification of tuberculosis

Since 1912 it has been a statutory requirement in England and Wales to notify all cases of clinically diagnosed tuberculosis through the notification of infectious diseases (NOIDs) system (1). The primary function of notification is to facilitate the rapid detection of outbreaks and epidemics and, in the case of tuberculosis, to ensure all cases and their contacts are managed appropriately. NOIDs also provides a valuable data set for interpreting historical trends. The Health Protection Agency (HPA) has the responsibility for collating weekly NOIDs data and reporting on local and national trends. As clinical suspicion of a tuberculosis case is all that is required in order to notify, some notified cases are subsequently found not to have tuberculosis. Such cases are not always de-notified. In addition, duplicate reports can also occur. On the other hand, failure to notify some cases of tuberculosis is well recognised to occur. In 1993, however, a review of the notification system for infectious diseases concluded that, for tuberculosis, the overall number of notified cases provided a reasonable estimate of the incidence of the disease in England and Wales (1).

Since 1999 tuberculosis cases have also been reported through the Enhanced Tuberculosis Surveillance system with the aim of adding to the general picture provided by NOIDs by collecting more detailed information on the occurrence of tuberculosis in England and Wales. Enhanced Tuberculosis Surveillance provides a more comprehensive clinical and demographic picture of tuberculosis in England and Wales but the annual data, which have to be collated, checked and de-duplicated, takes longer to finalise than reports to NOIDs.

One consequence of the differences between the two systems (and the extra steps taken to verify reports to the Enhanced Tuberculosis Surveillance system) has been that the final annual totals reported through NOIDs have tended to exceed annual cases reported through Enhanced Tuberculosis Surveillance. In March 2004, however, the HPA reported that total case numbers reported in Enhanced Tuberculosis Surveillance for 2002 exceeded notifications of tuberculosis reported through NOIDs for the first time. Although the Enhanced Tuberculosis Surveillance data for 2003 are still to be finalised, the NOIDs 2003 annual figures are now available and show a fall from the previous year (table 1). For Enhanced Tuberculosis Surveillance the trend has been for a continued rise in cases from 1999 to 2002.

In some areas, the introduction of Enhanced Tuberculosis Surveillance has been associated with a reduction or cessation in NOIDs reporting. Other possible contributing factors to these changes include the consequences of NHS reorganisation and other changes in reporting practice associated with the introduction of Enhanced Tuberculosis Surveillance. As Enhanced Tuberculosis Surveillance, however, involves a number of measures to validate reports, it is likely to better reflect the true incidence of TB.

Caution is therefore advised in the interpretation of recent NOIDs figures for tuberculosis . Discussions are currently underway to review this issue. The statutory requirement to notify all cases of tuberculosis remains in place and is vital for local control measures.

Further information about national tuberculosis surveillance can be obtained from the respiratory department of the HPA Communicable Disease Surveillance Centre at tbsection@hpa.org.uk.

Table 1 Tuberculosis notifications and Enhanced Tuberculosis cases reported for England and Wales: 1999 to 2003

Year	Notifications	Enhanced case reports
1999	6144	5704
2000	6572	6271
2001	6714	6597
2002	6753	6907*
2003	6518	NA

*Provisional figures. NA = Not available.

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Department of Health update on CJD

On 22 July 2004 the Department of Health made two announcements relating to Creutzfeldt-Jakob disease (CJD) in a written ministerial statement.

Following the December 2003 report of a possible transmission of variant CJD by blood transfusion, recipients of blood transfusions since January 1980 were excluded from donating blood with effect from 5 April 2004. Two further groups will be excluded from donating blood with effect from 2 August 2004:

- donors unsure whether they have previously had a blood transfusion
- apheresis donors who have previously had a blood transfusion. (Apheresis donors donate only certain blood components, and the rest of the blood is returned to the donor.)
- This action was postponed until the impact on the blood supply of the previous exclusion had been assessed; this has proved to be small.

The second announcement concerns a second possible transmission of variant CJD via blood transfusion. The patient died of causes unrelated to variant CJD, but a post mortem revealed the presence of variant CJD agent in the patient's spleen. The case is of particular scientific interest as the patient had a different genetic type to patients so far diagnosed with variant CJD.

The Department of Health press release is available at:

<http://www.dh.gov.uk/PublicationsAndStatistics/PressReleases/PressReleasesNotices/fs/en?CONTENT_ID=4086160&chk=9/Ni4w>.



An integrated approach to human transmissible spongiform encephalopathies (prion diseases)

On 9 July, the Chief Medical Officer issued a letter setting out new requirements for the joint reporting of Creutzfeldt-Jakob Disease (CJD) cases to both the National CJD Surveillance Unit <<http://www.cjd.ed.ac.uk>> (as in the past) and the National Prion Clinic <http://www.st-marys.nhs.uk/specialist/prion/index_prion.htm>. This is in addition to the requirement for local reporting by the consultant neurologist (or consultant in another specialty) responsible for the patient to the consultant in communicable disease control for investigation and, if appropriate, referral for advice concerning past medical procedures to the CJD Incidents Panel <http://www.hpa.org.uk/infections/topics_az/cjd/incidents_panel.htm>.

The reason for this change is the need to ensure a good level of referrals to PRION 1, the first United Kingdom clinical trial of potential therapy for CJD, which is due to start shortly. Referral of all new cases to the National Prion Clinic will give all new CJD patients the chance to benefit from participation in the PRION 1 trials and related research programmes. Whether or not patients take part in research trials, they may wish to use the specialist clinical and diagnostic expertise at the National Prion Clinic in addition to the services of the national care package co-ordinator based at the National CJD Surveillance Unit who can facilitate access to local support services.

The CMO letter includes as annexes the National CJD Reporting Form to be faxed by the consultant neurologist to the National CJD Surveillance Unit, National Prion Clinic and the local consultant in communicable disease control and also a patient information leaflet. It can be accessed online at <<http://www.dh.gov.uk/assetRoot/04/08/54/65/04085465.pdf>>.

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Immunisation

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-  [Laboratory reports of invasive meningococcal infections, England and Wales: weeks 09-12/2004](#)
-  [Laboratory Reports of *Haemophilus influenzae* by age group and serotype, England and Wales: April to June 2004](#)
-  [Laboratory reports of hepatitis A in England and Wales: January to March 2004](#)
-  [Laboratory reports of acute hepatitis B infection by age group and sex, England and Wales: January to March 2004](#)
-  [Laboratory reports of hepatitis C infection by age group and sex, England and Wales: January to March 2004](#)
-  [Serious hazards of transfusion \(SHOT\), 2003 report](#)

Laboratory reports of invasive meningococcal infections, England and Wales: weeks 09-12/2004

	Method of diagnosis			Total reports	Cumulative*
	CSF and blood Culture	Non-culture	Other sites	09-12/04	Total to week 12/2004
Group A	–	–	–	–	1
B	48	45	6	99	362
C	1	3	1	5	26
W135	3	1	3	7	16
X	–	–	–	–	–
Y	2	–	–	2	8
Z	–	–	–	–	–
29E	–	–	–	–	–
Ungroupable	–	–	–	–	–
Ungrouped	–	5	–	5	17
Total	54	54	10	118	430

* Combined CDSC data and Meningococcal Reference Unit data latex antigen, microscopy, polymerase chain reaction.

Laboratory Reports of *Haemophilus influenzae* by age group and serotype, England and Wales: April to June 2004

Serotype	Age					Total
	<1 year	1-4 years	5-14 years	≥15 years	Not known	
b	3(9)	2(19)	4(7)	16(25)	–(1)	25(61)
nc	7(6)	2(3)	1(2)	27(47)	3(1)	40(59)
a,e,f	1(1)	2(–)	2(–)	5(14)	–(–)	10(15)
not typed†	4(–)	2(1)	1(–)	15(18)	–(–)	22(19)
Total	15(16)	8(23)	8(9)	63(104)	3(2)	97(154)

* All data are provisional.

†Awaiting further typing data from the Oxford haemophilus reference unit.

Laboratory reports of hepatitis A in England and Wales: January to March 2004

During the first quarter of 2004, 164 laboratory reports of hepatitis A were made to the Health Protection Agency's Communicable Disease Surveillance Centre (CDSC), 42% (117) less than in the equivalent quarter of 2003. The last four quarters have seen a decrease in the number of cases compared to the equivalent quarters in the previous year. Thirty-eight per cent (63) were men aged between 15 and 44 years (table 1) and the majority of cases occurred in the Yorkshire and Humberside region. Three people acquired their infection abroad (Pakistan; South Africa; and Thailand one each) and one infection was reported to be in an injecting drug user (IDU). The overall number of cases of hepatitis A in the first quarter of 2004 decreased by 19% (38), compared to the fourth quarter of 2003. This decrease is similar to the previous quarter and is compatible with the general declining trend seen following the 2002 outbreak year (figure 1). The decrease seen this quarter occurred in males and females aged under 15 years and also in males and females aged between 15 and 44 years, the age group where most increase was seen previously. There was, however, an increase in the number of reports in both males and females aged over 45 years.

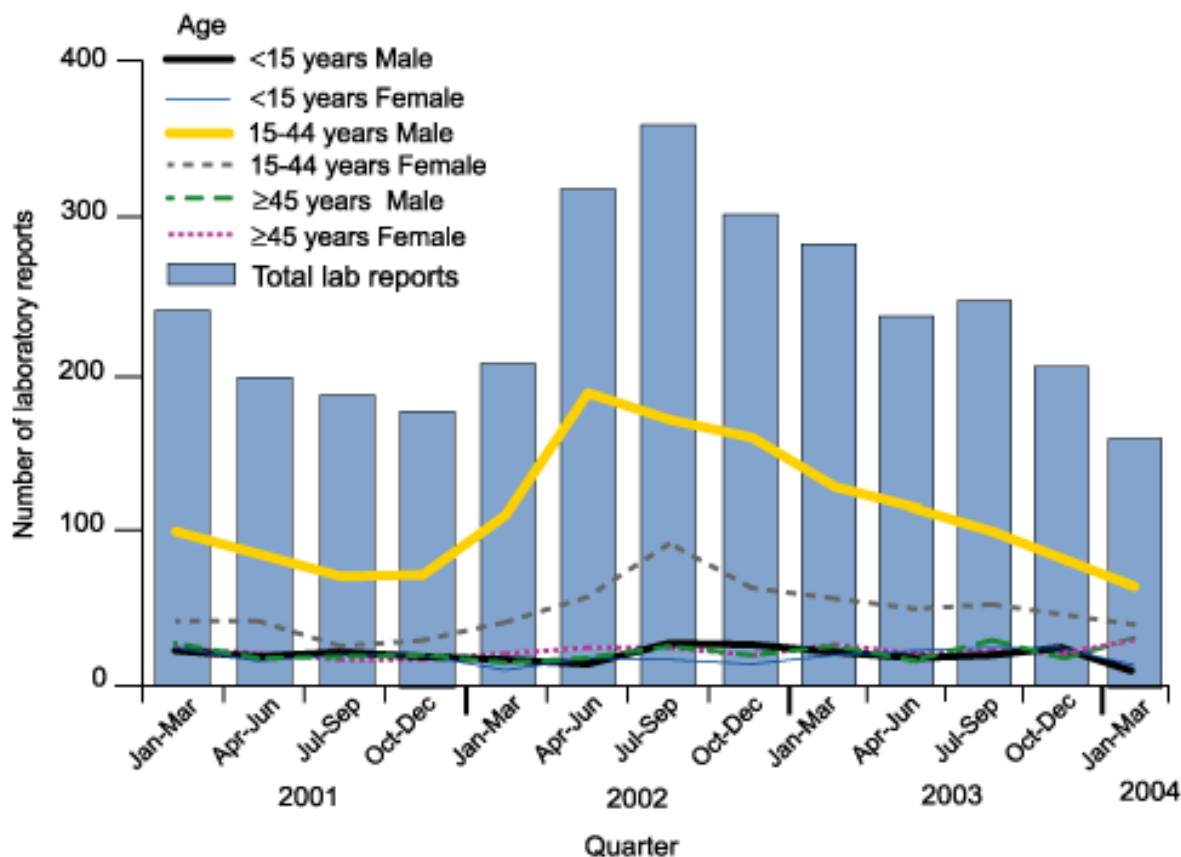
Table 1 Laboratory reports of hepatitis A infection in England and Wales: January to March 2004*

Age group (years)	Male	Female	NK†	Total
1-4	2	1	–	3
5-9	3	3	–	6
10-14	1	5	1	7
15-24	16	18	1	35
25-34	33	8	–	41
35-44	14	6	–	20
45-54	6	4	–	10
55-64	6	7	–	13
≥65	14	15	–	29
Total	95	67	2	164

* All data are provisional.

†NK = Not known.

Figure 1 Number of laboratory reports of hepatitis A by age group and sex: January 2001 to March 2004



Under-reporting and variations in regional reporting continue to present a challenge. A total of 221 cases of hepatitis A were formally notified in the first quarter of 2004, 26% more than laboratory confirmed. The number of notifications exceeded the number of laboratory reports for most regions. Discrepancy between notifications and laboratory reports was high in the London and South East regions. Similar to the fourth quarter of 2003, the largest discrepancy was seen in the North West region, where the number of laboratory reports exceeded the number of notifications. In this region twenty-six laboratory reports were made and only 14 cases were formally notified.

The total number of laboratory reports, as well as the number of notifications, has decreased this quarter compared to last, which may reflect a real decrease in the number of cases of hepatitis A. This suggests that the outbreaks occurring in the IDU community are being controlled.

Priorities for improving control of hepatitis A include enhancing risk-factor reporting by clinicians to laboratories and from laboratories to CDSC, increasing the speed and rates of notification of cases by clinicians to Health Protection Units, obtaining greater participation in laboratory reporting of cases, especially in London, and providing better detection and definition of outbreaks through means such as the application of hepatitis A virus genotyping.

**Laboratory reports of acute hepatitis B infection by age group and sex, England and Wales: January to March 2004**

Eighty-one reports of acute hepatitis B infection were reported in the first quarter of 2004 (*ie*, January to March 2004). The majority of cases (68%) occurred in those aged between 15 and 44 years (table 1).

Table 1 Laboratory reports of acute hepatitis B infection by age group and sex, England and Wales: January to March 2004*

Age group (years)	Male	Female	NK†	Total
<1	–	–	–	–
1-4	–	–	–	–
5-9	–	–	–	–
10-14	–	–	–	–
15-24	10	10	–	20
25-34	10	7	–	17
35-44	22	6	–	28
45-54	8	1	–	9
55-64	3	1	–	4
≥65	3	–	–	3
NK†	–	–	–	–
Total	56	25	–	81

* All data are provisional.

†NK = Not known.

During the first quarter of 2004 injecting drug use was the main risk-factor associated with acute hepatitis B infection, accounting for 17 out of 37 of individuals with known risk-factors (table 2). Acute hepatitis B infection associated with heterosexual exposure accounted for 12 out of 37 of the reported cases, and seven out of 37 men who had sex with men.

Table 2 Laboratory reports of acute hepatitis B infection by exposure category in England and Wales: January to March 2004*

Summary	Total
IVDU†	17
Sex between men & women	12
Sex between men	7
Other identified risk	1
NRI	44
Total	81

* All data are provisional

† IVDU = Intravenous drug user.

**Laboratory reports of hepatitis C infection by age group and sex, England and Wales: January to March 2004**

A total of 1823 reports of hepatitis C infection were reported in the first quarter of 2004 (table 3). Sixty-six per cent (1196/1815) of the cases occurred in those aged between 25 and 44 years. Cases in males exceeded those in females.

Table Laboratory reports of hepatitis C infection in England and Wales: January to March 2004*

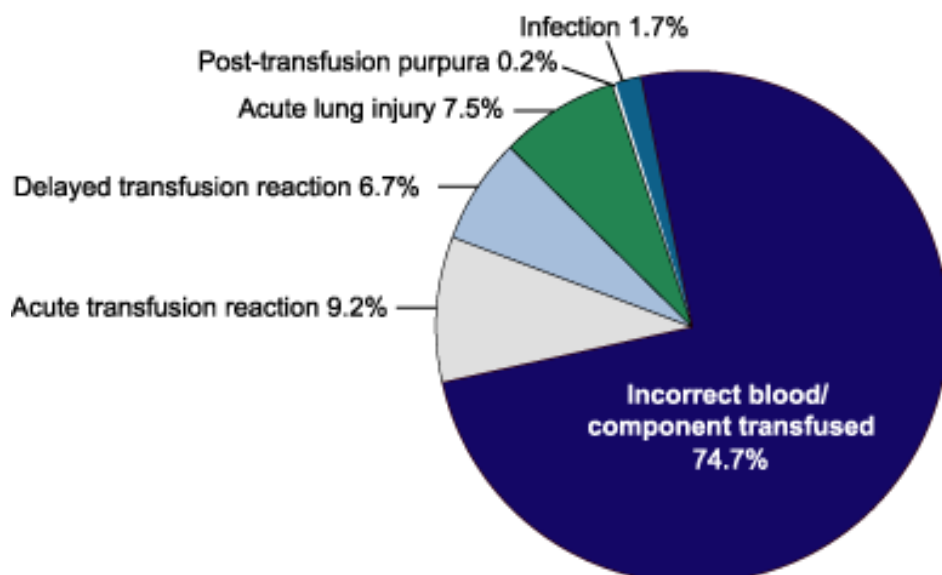
Age group (years)	Male	Female	NK†	Total
<1	1	–	–	1
1-4	9	7	–	16
5-9	1	2	–	3
10-14	1	–	1	2
15-24	112	71	6	189
25-34	421	189	11	621
35-44	418	146	11	575
45-54	191	62	7	260
55-64	51	36	2	89
≥65	33	24	2	59
NK†	5	1	2	8
Total	1243	538	42	1823

* All data are provisional.

†NK = Not known.

Serious hazards of transfusion (SHOT), 2003 report

The surveillance system for serious transfusion complications (serious hazards of transfusion [SHOT]) issued its seventh annual report on 5 July 2004 (1). The report documents incidents of serious complications associated with blood transfusion in the United Kingdom (UK) during 2003 (figure 1). In 2003, the main complication reported was 'incorrect blood/- component transfused' accounting for 74.7% of all case reports and transfusion transmitted infections (TTI's) accounted for 1.7%. There were eight reports of transfusion transmitted infections. Also, the UK's National CJD Surveillance Unit and the National Blood Service (NBS) reported the first possible case of transfusion transmitted vCJD, identified in 2003, following the death of a transfusion recipient (2).

Figure 1 Cases of serious complications associated with blood transfusion reported in the United Kingdom, 2003

In 2003, 351/415 (85%) hospitals reported that they participated in the SHOT scheme (table 1). This is a slight decrease (8%) from the previous period and might have resulted from a reduction in the follow-up of non-responders to SHOT during this year. One hundred and ninety-five (56%) of participating hospitals reported one or more incidents that had occurred during 2003. Forty-four per cent of participants did not make a report, which may indicate that incidents may be unnoticed or unreported.

Transfusion transmitted infections

Blood centres in England, Wales, and Northern Ireland report possible TTIs of which they have been informed to the NBS/Health Protection Agency's Communicable Disease Surveillance Centre (CDSC) Transfusion Transmitted Infection Surveillance scheme, and these are included in the SHOT report. All twelve blood centres reported possible incidents during 2003. Blood centres in Scotland report all incidents to the Microbiology Reference Unit of the Scottish National Blood Transfusion Service, for investigation. Details and findings on each incident are passed to the NBS/HPA CDSC system.

During 2003, a total of 38 reports of possible TTIs in the UK were made to the surveillance scheme. Two are still pending a full investigation. For 24 reports, the infection in the recipient was shown not to be due to transfusion, and classified as not TTIs. For eight reports, transfusion was thought to be the most probable cause of the infection, and these were classified as TTIs. These comprise of two hepatitis B virus (HBV), one HIV, one hepatitis A virus (HAV), one malaria, and three bacterial contaminations.

Despite reports of both transfusion transmitted HIV and HBV infections in 2003, the risk of an HIV or HBV infection donation entering the blood supply still remains low. This is a result of the current routine screening of blood donations, which include highly sensitive combined HIV antibody plus antigen assays, and HBsAg assays. The absence of any reports of transfusion transmitted hepatitis C virus (HCV) infections is consistent with the expected low risk of an HCV infectious donation entering the blood supply in the presence of HCV antibody (anti-HCV) and HCV RNA testing. The low number of reported bacterial contaminations this year may be the result of both improved donor arm cleaning and procedures to divert the first 20 to 30ml of each donation. Transfusion transmitted bacterial infection remains an avoidable cause of death and major morbidity and merits increased efforts to prevent bacterial contamination of components.

Transfusion transmitted infections continue to be rare in the UK. Each year the number of TTIs occurring is small and fluctuations are to be expected. The reporting system is probably biased towards the ascertainment of investigations of infections that cause rapid onset of acute disease such as bacteria. Since reporting began, bacteria have accounted for the majority (62%) of reported transmissions by transfusion and the majority (88%) of known deaths due to TTI's (1).

Table 1 Cumulative total transfusion-transmitted infections reported in the UK: data to 31 December 2003 by year of transfusion. (The number of incidents is shown with the total number of identified recipients in brackets).

Year of transfusion	Pre 1996	1996	1997	1998	1999	2000	2001	2002	2003	Total	Deaths
Infection											
HAV	–	1(1)	–	–	–	1 (1)	–	–	–	2	–
HBV	2(2)	1(1)	1(1)	1(1)	2(3)	1(1)	–	1(1)	1(1)	10	–
HCV	–	1(1)	1(1)	–	–	–	–	–	–	2	–
HIV	–	1(3)	–	–	–	–	–	1(1)	–	2	–
Bacteria	1(1)	1(1)	3(3)	4(4)	4(4)	7(7)	5(5)	1(1)	3(3)	29	7
Malaria	–	–	1(1)	–	–	–	–	–	1(1)	2	1
HTLV I	2(2)*	–	–	–	–	–	–	–	–	2	–
Possible vCJD	–	1(1)	–	–	–	–	–	–	–	1	1
Total*	5(5)	6(8)	6(6)	5(5)	6(6)	9(9)	5(5)	3(3)	5(5)	50	9

*One investigation reported as pending in the sixth SHOT report has since been confirmed transfusion transmitted HTLV infection.

Transfusion transmitted infections reported in 2003

Hepatitis B

The two reports of transfusion transmitted hepatitis B virus infection made in 2003 both involved donations from donors in the early acute phase of infection. In the first report, a repeat donor found to be hepatitis B surface antigen (HBsAg) positive on routine testing. A lookback at previous donations identified a donation made one month earlier that had been HBsAg negative and transfused. The associated archived sample was re-tested and confirmed negative for HBsAg, HBV DNA, and

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Eight of the nine cases of transfusion-transmitted HBV infection made since 1995 were due to acute infections in donors. This is a change from earlier data when between 1991 and 1997 only three of 14 HBV TTI's came from donors with acute infections, with the majority from donors with chronic infection.

HIV

In 2003, the first report of transfusion-transmitted HIV was made since 1996. An anti-HIV positive repeat donor was identified in 2003. The donor had been anti-HIV negative at the time of their most recent previous donation in 2002; the archive of this donation was retrieved for PCR testing and was found to be HIV RNA positive. A single recipient was traced and tested 15 months after transfusion with anti-HIV and RNA positive findings. No source of the donor's infection was identified.

Hepatitis A

Testing for hepatitis A virus is not mandatory in the UK. A report of transfusion-transmitted HAV infection was made in 2003. A repeat donor reported onset of jaundice to the NBS six days after making a donation and was found to be anti-HAV IgM positive. A single recipient of this donation was identified. The recipient was known to be negative for anti-HAV at the time of transfusion, but eight weeks later sero-converted and subsequently developed symptoms of hepatitis A disease with uncomplicated recovery. This case highlights the importance of donors informing the blood centres of any infections noticed in the period following donation, in order that prompt action can be taken to recall any implicated units and test any recipients.

Malaria

Testing for malaria is not mandatory in the UK. A report of transfusion transmitted malaria was made in 2003 following an investigation into low haemoglobin levels in a recipient who had received many transfusions over three months, for treatment of sickle cell disease. Review of a blood film from the recipient identified a low-level *Plasmodium falciparum* parasitaemia despite a lack of travel history outside the UK. The associated archived samples for this recipient were retrieved and tested for malaria antibodies and the donors were contacted for any relevant travel history. This revealed a donor who was positive for malaria antibodies and had lived in West Africa until the age of 21 years, but had not visited the area for seven years prior to the donation. Although malaria antibody testing could have avoided this transmission, in this instance the donor had not visited an endemic area for seven years and thus did not qualify for testing under current guidelines.

Bacterial contamination

During 2003, there were three reports of transfusion-transmitted bacterial contaminations. Two recipients were seriously ill, and one died. All three cases were due to transfusion of contaminated platelets.

One recipient developed rigors and hypotension following transfusion of a two day old unit of apheresis platelets for treatment of leukaemia. Despite intra-venous fluids and antibiotics the patient developed cardiac failure and died 15 hours after the transfusion. *Escherichia coli* was cultured from the recipient's blood and the implicated platelet pack. Extensive investigation failed to reveal a source for the bacterial contamination, but since the venepuncture site of the donor was not swabbed, the donor's arm could not be excluded as a possible source.

One recipient developed fever and diarrhoea following transfusion of a single unit of four day old apheresis platelets during treatment for acute myeloid leukaemia. *Staphylococcus aureus* of the identical strain was cultured from the recipient's blood, the platelet pack and the venepuncture site of the donor. The recipient recovered after antibiotic treatment and was discharged from hospital five days after the transfusion.

One recipient developed hypotension, breathlessness, fever, and rigors following a transfusion of a five day old unit of pooled platelets. *Staphylococcus epidermidis* was cultured from the patient, the pooled unit and (although not same strain) the venepuncture site of one of the donors. The probable source of the contamination was the donor's arm despite the fact that the organism isolated was a different strain from that isolated from the patient and the platelet pack.

Blood centres in England and Wales also report cases of post transfusion reactions (PTRs) suspected to be due to bacteria. In 2003, two PTR reports were concluded to be due to transfusion of bacterial contaminated products (two of the three bacterial TTIs in the UK reported above). A further 38 reports were received where bacterial contamination was initially included in the differential diagnosis, but there was no evidence of bacterial infection in either the recipient or the implicated component that could have caused the reaction.

vCJD

A case of variant Creutzfeldt-Jakob Disease (vCJD) was diagnosed after death in a transfusion recipient (aged 62 years) (2). In 1996, this individual had received blood from a donor who had, three years post donation developed symptoms of vCJD and died from pathologically confirmed vCJD in 2000. The implicated unit was not leucodepleted and had been transfused to the recipient while undergoing surgery. The source of the recipient's infection was concluded to be possibly a vCJD infectious unit of red cells. As a result of the absence of supporting evidence that human prions can be transmitted by transfusion, and because (in this case) other possible sources such as dietary exposure to bovine spongiform encephalopathy (BSE) agent could not be excluded, the source of the recipient's infection could not be confirmed as a transfusion.

References

1. Serious Hazards of Transfusion Organisation. Stainsby D, Cohen H, Jones H, Knowles S, Milkins C, Chapman C, *et al.* *Serious hazards of transfusion annual report 2003*. Manchester: SHOT, 5 July 2004. Available at: <<http://www.shotuk.org>>. Price £15 for private hospitals and commercial organisations but free of charge to NHS staff.
2. Llewelyn CA, Hewitt PE, Knight RS, Amar K, Cousens S, Mackenzie J, *et al.* Possible transmission of variant Creutzfeldt-Jakob disease by blood transfusion. *Lancet* 2004; **363**: 417-21.