

Acute hepatitis B infection in England and Wales: 1985–96

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SUMMARY

Confirmed acute hepatitis B infections are reported to the Public Health Laboratory Service Communicable Disease Surveillance Centre by laboratories in England and Wales. These reports have been used to monitor trends in the incidence of hepatitis B virus (HBV) infection over time, and between exposure categories and age groups. Between 1985 and 1996 a total of 9252 cases of acute HBV infection were reported; the number of reports fell from 1761 in 1985 to 581 in 1996. Most infections were reported in adults aged 15–44 years [$n = 7365$ (80%)], and infections were more commonly reported in males [$n = 6490$ (70%)] than females [$n = 2658$ (29%)]. The probable means of acquisition was known for just over half of all adult cases [$4827/8956$ (54%)]. Injecting drug use was the most common exposure [$n = 1901$ (21%)], followed by sex between men and women [$n = 1140$ (13%)] and sex between men [$n = 1025$ (11%)]. The number of infections in injecting drug users fell in the late 1980s, but increased again from 1991 onwards. In children aged under 15 years, infections acquired by mother to baby transmission accounted for 35/170 (21%) of the total. Surveillance indicates that the incidence of acute hepatitis B infection fell in the late 1980s, probably reflecting changed behaviour in injecting drug users. An increase in the number of infections in injecting drug users since 1993 may indicate ongoing transmission that has not been contained by the introduction of needle exchange schemes or by selective vaccination.

INTRODUCTION

Hepatitis B virus (HBV) infection can lead to chronic hepatitis, cirrhosis and hepatocellular carcinoma [1]. The United Kingdom (UK) is a relatively low prevalence country for hepatitis B infection with an estimated carriage rate of less than 1% [2]. Infection is mainly acquired in adulthood, usually by sexual intercourse or injecting drug use. Acute HBV infection may be asymptomatic or cause non-specific symptoms; about one-third of adult infections are symptomatic. Acute HBV infections confirmed in

laboratories in England and Wales can therefore be used to estimate the incidence of HBV infection, and to monitor the trends in that incidence. This paper summarizes the epidemiology of acute infections reported from laboratories in England and Wales between 1985 and 1996.

METHODS

Acute hepatitis B (HBV) infections confirmed by laboratories in England and Wales are reported to the Public Health Laboratory Service Communicable Disease Surveillance Centre (PHLS CDSC). Lab-

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oratory confirmation of acute HBV infection includes a positive result of a test for hepatitis B anti-core IgM (anti-HBc IgM), or a positive result of a test for hepatitis B surface antigen (HBsAg) together with symptoms compatible with acute HBV and, if available, a negative result of a test for IgM antibody to hepatitis A virus (HAV). Additional cases ascertained by contact tracing or the investigation of clusters are included in the surveillance database if they have evidence of recent infection (anti-HBc IgM positive or seroconversion to anti-HBc IgG) even in the absence of a clinical illness. Children infected by perinatal transmission and identified during the follow up of known high-risk mothers are also included. Surveillance reports include clinical and demographic details and information about risk exposure(s) in the previous 6 months. These details are supplied by laboratory personnel on the basis of information available at the time of test request or upon discussion of the results with the requesting doctor. Reports with missing exposure information are followed up by writing to the laboratory for further details. By this time, further epidemiological investigation and control measures may have been undertaken and additional information ascertained on exposure history. For all cases reported during 1993, additional information on possible exposures was obtained from clinicians and patients and has been used to augment routine data [3]. In 1994, routine reporting methods from laboratories were resumed. In cases where more than one risk exposure is reported a hierarchical system for coding is employed. Injecting drug use is considered to be the most likely risk factor, followed by sex between men and then sex between men and women. The rate of laboratory reported acute hepatitis B infections in adults were estimated using mid-year age-specific population estimates from the Office for National Statistics (ONS) [4]. Prior to 1994, population estimates for the current regional configurations were approximated by amalgamating estimates for the old regional configurations.

RESULTS

Between 1985 and 1996 a total of 9252 cases of acute HBV were reported to PHLS CDSC. Of these, the vast majority [9187 (99%)] were identified because of symptomatic disease; 6786 (73%) were associated with jaundice. The majority of cases were sporadic; only 295 (3.2%) cases were associated with clusters and 65 (22%) of these were identified during investi-

gation of cases associated with surgical or medical treatment in hospital. Fifty-five (0.6%) cases were known to have died; in 45 HBV infection was thought to be the cause or a contributory factor in the death.

The total number of cases reported each year showed a threefold decline from a total of 1761 cases in 1985 to 583 in 1989 and has remained at similar levels since, with 581 reports received in 1996. A total of 8956 (97%) of cases were reported in adults (over the age of 15 years) (Table 1) with only 170 (1.8%) in children (in 126 cases the age was not stated). Most cases were in the 15–24 and 25–44 years age groups and the decline in the rate of laboratory reported acute HBV infections has been most marked in these age groups (Fig. 1). The average estimated annual rate of laboratory reported hepatitis B infection in adults within each region varied from 1.27 to 2.81 per 100 000 population. The highest rates were reported from the North-West, Northern and Yorkshire, and South Thames regions but rates fell in all regions over the period (Table 2).

Of all adult acute HBV infections reported, men accounted for 6303 (70%) and women for 2560 (29%) (in 93 cases the sex was not stated). Risk exposures were reported in 4827 (54%) – 3395 men and 1391 women (in 41 cases the sex was not stated) (Table 1). Amongst male cases, injecting drug use was the most common risk factor, followed by sex between men and sex between men and women. Amongst female cases, sex between men and women was the most commonly reported exposure followed by injecting drug use. Amongst both sexes the numbers of cases associated with injecting drug use fell in the late 1980s, but has been increasing again since 1991. Two hundred and seventy-four cases [260 men (95%); 14 women (5%)] were reported to have been diagnosed in prison of which 186 (68%) were associated with injecting drug use. The number of cases acquired through sexual exposure between men and women has shown a similar trend as cases acquired by injecting drug use, but the changes have been less marked. The numbers of cases acquired through sex between men have remained fairly stable over the whole period, but the proportion of all cases acquired by this route increased (Table 1).

The proportion of adult cases with no known risk exposure was lower in the final period of surveillance (1993–6) than in 1985–8 and 1989–92 (Table 1). For cases reported in 1993, additional risk factor information was obtained from clinicians and patients and only 195/613 (32%) of infections had no

Table 1. Exposure categories in laboratory reported acute hepatitis B infections in persons aged 15 years and over, England and Wales, 1985–96

Exposure category	Gender	1985–8		1989–92		1993–6		1985–96	
		No.	%	No.	%	No.	%	No.	%
Injecting drug use	All	1092	25.0	274	12.4	535	22.4	1901	21.2
	Male	846		191		406		1443	
	Female	246		83		129		458	
Sex between men and women	All	457	10.5	283	12.8	400	16.8	1140	12.7
	Male	126		119		182		427	
	Female	331		164		218		713	
Sex between men	Male	346	7.9	313	14.0	366	15.0	1025	11.0
Sexual contact (unspecified)	All	80	1.8	83	3.8	45	1.9	208	2.3
Other risks	All	252	5.8	116	5.3	185	7.8	553	6.2
All known risks	All	2227	51.0	1069	48.5	1531	64.2	4827	53.9
Risk not identified	All	2142	49.0	1134	51.5	853	35.8	4129	46.1
Total	All	4369		2203		2384		8956	

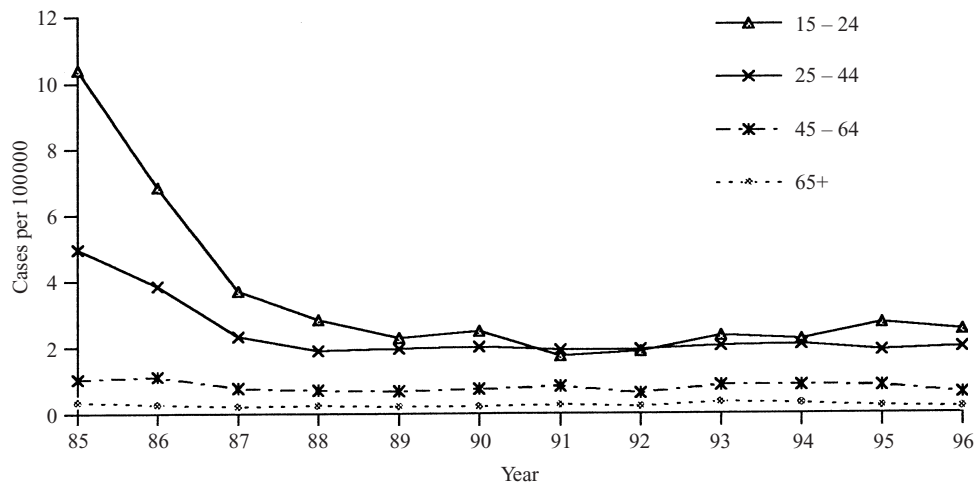


Fig. 1. Estimated annual age-specific rates of laboratory-reported acute symptomatic hepatitis B.

Table 2. Laboratory reported acute hepatitis B infections in persons aged 15 years and over by region: England and Wales 1985–96

Region	1985–8		1989–92		1993–6		1985–96	
	No.	Mean annual rate per 100000	No.	Mean annual rate per 100000	No.	Mean annual rate per 100000	No.	Mean annual rate per 100000
Northern and Yorkshire	714	3.31	218	1.00	391	1.84	1323	2.05
Trent	354	2.35	123	0.80	173	1.09	650	1.42
Anglia and Oxford	328	2.30	86	0.53	179	0.99	593	1.27
North Thames	546	2.30	394	1.65	310	1.36	1250	1.77
South Thames	576	2.65	407	1.85	356	1.61	1339	2.04
South and West	466	2.33	259	1.26	228	1.07	953	1.55
West Midlands	351	2.10	198	1.09	156	0.92	705	1.37
North West	845	4.11	443	2.15	454	2.16	1742	2.81
Wales	189	2.07	75	0.81	137	1.46	401	1.44
Total	4369	2.68	2203	1.31	2384	1.41	8956	1.80

Table 3. *Acute hepatitis B laboratory reports in persons under 15 years: England and Wales 1985–96*

Exposure category	Probable place of exposure		Total	Percentage
	In UK	Overseas		
Injecting drug use	3	0	3	1.8
Sexual exposure	3	0	3	1.8
Medical treatment	6	11	17	10.0
Others	7	1	8	4.7
Mother to baby	31	4	35	20.6
Family/household	22	7	29	17.1
Not reported	64	11	75	44.1
Total	136	34	170	100.0

identified risk exposure. After resuming routine reporting of risk factors by laboratories in 1994, the proportion of cases with no identified risk exposure remained low [658/1771 (37%)].

Of the 4827 cases with known risk exposures, 468 (9.7%) were reported to have been acquired abroad (353 in countries with a high or intermediate endemicity). For this subgroup, sex between men and women was the most commonly reported exposure [$n = 207$ (44%)] followed by medical, dental or surgical treatment [$n = 101$ (21%)].

Of the 2953 adult cases with a known risk exposure in the UK, only 376 (13%) were associated with exposures other than injecting drug use or sexual exposure. This included 213 with surgical or medical treatment in hospital, 2 with dental treatment, 62 with household contact (22 with another acute case, 13 with a known carrier, 2 with a high risk individual and 25 with unspecified contact), 32 with occupational exposure, 11 in residential care, 14 who had been tattooed, 5 who had received acupuncture and 37 others.

Of the 213 cases associated with medical or surgical treatment in UK hospitals, 69 were associated with transfusion, 119 with surgery, and 25 with medical procedures. Results of the blood service investigations of cases associated with transfusion since 1990 have been summarised elsewhere [5]. Those cases associated with surgery have also been described elsewhere [6–11], and, in 1993, resulted in strict guidance on the screening and vaccination of health care workers who perform exposure prone procedures [12, 13]. Since 1993 only 11 infections were acquired by surgery in England and Wales: 5 of these cases were associated with procedures performed by HBeAg negative

surgeons [7, 11]. Of the 25 cases associated with medical procedures, 8 had received clotting factors (6 prior to heat treatment, 2 following a treatment failure), 4 were associated with transmission in a drug trials unit [14], 5 occurred during bone marrow transplantation [15], 2 during dialysis, 2 following renal transplants, 2 occurred in insulin-dependent diabetics admitted to the same ward and 2 were sporadic cases for which no clear exposure was documented.

Occupational exposure in the UK was reported in 32 cases of which 26 (81%) were health care workers [16]. In the period 1985–8 there were 16 cases amongst UK health care workers. This fell to 8 in the period 1989–92 and declined further to only 2 cases in 1993–6. The remaining cases include 2 social workers (exposed to a single carrier in residential care), 3 policemen exposed during the course of their work (1 was in a fight with a known HBsAg carrier and 1 had a documented blood exposure, but prophylaxis with immunoglobulin alone failed) and 1 homehealth-care provider.

One hundred and seventy children under 15 years of age with acute or recently acquired HBV infection were reported to CDSC between 1985 and 1996. Of these children, 75 (44%) had no known risk exposure reported, although 11 of these children were known to have recently travelled to an endemic area (Table 3). For childhood infections with a reported exposure, the most common route of acquisition was by vertical transmission. Of the 35 cases acquired by vertical transmission, 28 were detected by investigation of children born to high-risk mothers, the remaining 7 cases were detected during investigation of symptomatic disease in the child. Thirty-one infections

have occurred in UK born children, 8 occurred before the availability of vaccine, 6 were vaccination failures and in 1 child vaccine was given late – the remaining cases were not known to have been vaccinated. Of the 22 infections reported to have been acquired by family contact in the UK, in 9 a HBsAg carrier was identified in the household and in 5 contact occurred with a case of acute hepatitis (details were not available for the remaining 8). The infections acquired by medical treatment in the UK included 2 associated with transfusion, 2 with receipt of factor VIII prior to heat treatment and 2 with surgery. Of 7 cases infected by other means in the UK, 2 had contact with a HBsAg carrier at a playgroup [17], 3 were thought to have been exposed by ear piercing, 1 during karate and 1 whilst a family member received home dialysis. All six childhood cases infected by sexual contact or injecting drug use occurred in children aged 14 years.

DISCUSSION

Surveillance of acute hepatitis B infections by laboratory reports in England and Wales has been shown to give a reasonable estimate of the incidence of symptomatic infection [18]. A recent audit in three regions suggested that 82% of all laboratory confirmed acute infections are reported to CDSC [19], but because at least two thirds of adult HBV infections are asymptomatic, laboratory surveillance cannot ascertain all acute HBV infections. As the proportion of asymptomatic infections in adults, however, is expected to be fairly constant over time, and because the number of asymptomatic cases detected is small, laboratory surveillance is probably a valid method for monitoring trends in the incidence of hepatitis B in adults.

Surveillance indicates that the incidence of acute hepatitis B infection fell markedly in the late 1980s and has remained stable during the early 1990s. The highest rates of reported acute hepatitis B were observed in the 15–24 and 25–44 years age groups – the groups in which the major risk exposures were most common. Over two-thirds of acute adult HBV infections were reported in males; this is explained by cases amongst men who have sex with men and by the predominance of males amongst injecting drug users [20]. The rate of symptomatic infections reported fell in the 15- to 44-year-old age group in the late 1980s, whilst rates in older adults were stable.

During the early 1980s the annual number of reports of acute hepatitis B almost doubled to reach

1939 in 1984 [21]. This increase was mainly attributable to infections in drug users and cases in this group fell dramatically in the late 1980s. As for injecting drug users, and in contrast to cases acquired by other routes, cases with no reported/identified risk factor fell dramatically in the late 1980s. This fall may partly reflect improved ascertainment of other risk factors, but suggests that some cases with no identified risk were infected by injecting drug use. An on-going cross-sectional survey amongst injecting drug users has also demonstrated a fall in the proportions with evidence of past infection between 1990 and 1995 [22]. The decline in numbers of cases and prevalence may reflect a change in injecting drug user behaviour. Needle exchange programmes, designed to prevent the transmission of HIV, were introduced in England and Wales in the mid-1980s and may have contributed to a fall in HBV incidence and prevalence amongst injecting drug users. In 1995, however, 1 in 5 injecting drug users reported sharing of equipment [22], and the increase in cases in injecting drug users in the mid-1990s may herald the start of another epidemic. Future transmission will be facilitated by the failure to vaccinate the susceptible injecting drug user population [23]. Prevention of a future epidemic requires offering vaccine to this group at highest risk of HBV infection at all opportunities, including within prisons, and through specialist and non-specialist services [24, 25].

Vaccination is currently recommended for other groups, such as health-care workers, genito-urinary medicine (GUM) clinic attenders, and ‘high-risk’ travellers [25]. The decline in cases amongst health-care workers may be attributable to vaccination [16], but the numbers of cases in other groups has shown little decline. Many adult cases are acquired overseas, and such cases might be prevented by vaccination prior to travel. Travellers, however, may not present for vaccination and may not be perceived by their doctor to be at risk [3]. Although infections associated with exposures other than injecting drug use or sexual exposure are uncommon, there is potential for further reduction in incidence by better implementation of selective vaccination in certain settings [3].

Another factor that may have contributed to the fall in cases in the late 1980s is the adoption of safer sexual practices. In contrast to those cases reported amongst injecting drug users, however, the number of cases acquired by sexual exposure has shown less dramatic changes. The promotion of safer sexual practices has been largely directed at men who have

sex with men. Cases in this exposure category, however, have not fallen. An increase in cases with the probable exposure reported as sex between men and women has been observed since 1993, but this increase corresponded to a fall in cases with no identified risk. Enhanced surveillance has suggested that obtaining more complete data would attribute a higher proportion of cases to sexual exposure [3], and therefore this increase may represent better recognition and reporting of this route of transmission. A survey amongst homosexuals in clinic and community settings found that 17% had evidence of past HBV infection [26], whereas the prevalence of past infection was low in heterosexuals attending a GUM clinic outside of London [27]. National coverage of selective vaccination has not been determined but *ad-hoc* surveys suggest that coverage in GUM clinics is low [23, 28, 29]. Although the proportion of the population who attend GUM clinics is small [20], this setting does provide an opportunity to protect some groups at higher risk of HBV infection.

Because up to 90% of acute HBV infections acquired in childhood are asymptomatic, symptomatic cases reported in children considerably underestimate the true incidence of infection. Asymptomatic childhood infections, however, are often identified by the investigation of children at risk and reported to CDSC. Despite this, cases amongst children are rare and, amongst cases with known exposures, the most common route of transmission is from mother to baby. Perinatal transmission may lead to chronic carriage and to long term complications, but can be largely prevented by antenatal testing to identify carrier mothers and by active immunization of the baby [25]. It is therefore disappointing that cases of perinatal transmission have occurred in unvaccinated infants since the licensure of hepatitis B vaccine. This may reflect a failure of selective antenatal testing policies, and universal testing is now recommended [30]. The World Health Organisation (WHO) recommended that all countries integrate HBV vaccine into national immunization programmes by 1997 [31]. It has been suggested, however, that in the presence of universal antenatal testing, few additional childhood infections would be preventable by universal infant immunization in the UK [32]. In addition, vaccination in infancy would take many years to impact upon the incidence in adulthood and so an alternative may be universal adolescent immunization. With either mass approach, in the short to medium term, improved implementation of the current selective vaccination

policy is required. Laboratory surveillance of acute hepatitis B infection can be used, in conjunction with other data, to target future control programmes more effectively and to evaluate the impact of current and future vaccination policy.

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REFERENCES

1. Wright TL, Lau JYN. Clinical aspects of hepatitis B virus infection. *Lancet* 1993; **342**: 1340–4.
2. Boxall EH, Flewett TH. Prevalence of HBsAg in UK population. *BMJ* 1987; **294**: 57.
3. Mangtani P, Heptonstall J, Hall AJ. Enhanced surveillance of acute symptomatic hepatitis B in England and Wales. *Commun Dis Public Health* 1998; **1**: 114–20.
4. Office for National Statistics: Mid-year population estimates PP1 series, 1985–1996.
5. Soldan K, Ramsay M, Collins M. Reports of acute HBV infection associated with blood transfusion in England and Wales, 1991–1997. *BMJ* 1998 (in press).
6. Heptonstall J, Collins M, Botto B, Gill ON. Restricting practice of HBeAg positive surgeons. Lessons from hepatitis B outbreaks in England, Wales and Northern Ireland 1984–1995. IX Triennial International Symposium on Viral Hepatitis and Liver Disease, Rome, 1996.
7. Sundkvist T, Hamilton GR, Rimmer D, Evans BG, Teo CG. Fatal outcome of transmission of hepatitis B from an e antigen negative surgeon. *Commun Dis Public Health* 1998; **1**: 48–50.
8. Heptonstall J. Outbreaks of hepatitis B virus infection associated with infected surgical staff. *CDR Rev* 1991; **1**: R81–5.
9. The incident control team and others. Lessons from two linked clusters of acute hepatitis B in cardiothoracic surgery patients. *CDR Rev* 1996; **6**: R119–25.
10. Mukerjee AK, Westmoreland D, Rees HG. Response to the discovery of two practising surgeons infected with hepatitis B. *CDR Rev* 1996; **6**: R126–8.
11. The incident investigation teams and others. Transmission of hepatitis B to patients from four infected surgeons without hepatitis B e antigen. *N Eng J Med* 1997; **336**: 178–84.
12. Anonymous. Protecting health care workers and patients from hepatitis B: Recommendations of the

- Advisory Group on Hepatitis. UK Health Departments, 1993.
13. Anonymous. Protecting health care workers and patients from hepatitis B: Addendum to HSG (93)40. NHS Executive, 1996.
 14. Vickers J, Painter MJ, Heptonstall J, Yusof JHM, Craske J. Hepatitis B in a drug trials unit: investigation and recommendations. *CDR Rev* 1994; **4**: R1–5.
 15. Tedder RS, Zuckerman MA, Goldstone AH, et al. Hepatitis B transmission from contaminated cryopreservation tank. *Lancet* 1995; **346**: 137–40.
 16. Collins M, Heptonstall J. Occupational acquisition of acute hepatitis B infection by health care workers in England and Wales, 1985–93. *CDR Rev* 1994; **4**: R153–5.
 17. Ngui SL, Andrews NJ, Underhill GS, Heptonstall J, Teo CG. Failed postnatal immunoprophylaxis against hepatitis B: characteristics of maternal hepatitis B virus as risk factors. *Clin Infect Dis* 1998; **27**: 100–6.
 18. Polakoff S, Tillett H. Routine laboratory reports of patients with acute hepatitis B as indicators of incidence of the disease. *J Infect* 1984; **8**: 44–8.
 19. Ramsay M, Gay N, Balogun K, Collins M. Control of hepatitis B in the UK. *Vaccine* 1998 (in press).
 20. Wadsworth J, Hickman M, Johnson Am, Wellings K, Field J. Geographic variation in sexual behaviour in Britain: implications for sexually transmitted disease epidemiology and sexual health promotion. *AIDS* 1996; **10**: 193–9.
 21. Polakoff S. Acute viral hepatitis B, reported to the Public Health Laboratory Service. *J Infect* 1990; **20**: 163–8.
 22. Unlinked Anonymous HIV Seroprevalence Monitoring Programme in England and Wales: Report from the Unlinked Anonymous Surveys Steering Group. Data to the end of 1995. Department of Health, 1996.
 23. Mangtani P, Kovats S, Hall A. Hepatitis B vaccination policy in drug treatment services. *BMJ* 1996; **312**: 1500.
 24. Gill ON, Noone A, Heptonstall J. Imprisonment, injecting drug use, and bloodborne viruses. *BMJ* 1995; **310**: 1264.
 25. UK Health Departments. Immunisation against Infectious Disease, 1996. London: HMSO, 1996.
 26. Bhatti N, Gilson RJC, Beecham M, et al. Failure to deliver hepatitis B vaccine: confessions from a genitourinary medicine clinic. *BMJ* 1991; **303**: 97–101.
 27. El-Dalil AA, Jayaweera DT, Walzman M, et al. Hepatitis B markers in heterosexual patients attending two genitourinary medicine clinics in the West Midlands. *Genitourin Med* 1997; **73**: 127–30.
 28. Nandwani R, Barton S. Medical audit of hepatitis B immunization policy in a genitourinary medicine clinic. *Int J STD AIDS* 1993; **4**: 424.
 29. Hart GJ, Dawson J, Fitzpatrick RM, et al. Risk behaviour, anti-HIV and anti-hepatitis B core prevalence in clinic and non-clinic samples of gay men in England, 1991–1992. *AIDS* 1993; **7**: 863–9.
 30. NHS Executive. Screening of pregnant women for hepatitis B and immunisation of babies at risk. Department of Health, 1998. (HSC 1998/127).
 31. Kane M. Global programme for control of hepatitis B infection. *Vaccine* 1995; **13** (Suppl 1): S47–9.
 32. Hesketh LM, Rowlatt JD, Gay NJ, Morgan-Capner P, Miller E. Childhood infection with hepatitis A and B viruses in England and Wales. *CDR Rev* 1997; **7**: R60–3.