

Communicable Disease Report

Health care workers and HIV: surveillance of occupationally acquired infection in the United Kingdom

J Heptonstall, O N Gill, K Porter, M B Black, V L Gilbert

Summary

In the period 1985-1992, 176 significant occupational exposures to HIV were reported to the PHLS Communicable Disease Surveillance Centre. The outcome at three months post exposure was reported for 134 (76%) incidents. Ninety-nine of these involved percutaneous exposure to HIV-infected blood or serum; two resulted in seroconversion, one following the use of zidovudine post exposure. Under-reporting of significant exposures may have been considerable. However, the observed transmission rate, of 2%, is not inconsistent with other estimates.

Two other documented seroconversions after occupational exposure have been reported, making a total of four health care workers known to have acquired HIV infection after occupational exposure in the UK. Another six UK health care workers have possible occupationally acquired HIV infections. Five of these probably became infected while working in adverse conditions in Africa; the other while working with HIV-infected patients in the United States and Europe.

A summary of current good practice of post exposure management is provided. Practitioners providing post exposure care are asked to contribute to the national surveillance scheme. Initial reporting of significant occupational exposures, and of serological outcome at a minimum of six months post exposure, should be regarded as integral to satisfactory post exposure management.

Introduction

The occupational risk of HIV infection became a major issue for health care workers, their physicians and their employers in 1984, when the first case of documented seroconversion after percutaneous exposure to HIV-infected blood at work was reported¹. Countries with well developed health services have since incorporated procedures for monitoring workplace exposure to HIV within established systems for reporting workplace injuries. Local centres may report the details and outcome of each significant exposure to a central registry^{2,3} or may report exposures which result in infection to national AIDS and HIV surveillance centres⁴. Both definite and possible occupationally acquired HIV infections can then be highlighted in the routine information output from the national centre⁵, which may additionally undertake the further investigation of HIV-infected persons without identified risks for HIV infection^{4,6}.

Such surveillance allows the size of the problem and degree of risk to be quantified, and particularly hazardous working practices and procedures may be identified. This information can then be made available to health care workers, occupational health departments, clinicians, employers and manufacturers and, ultimately, can be used to produce a safer working environment.

A national system for the surveillance of UK health care workers who sustained a significant occupational exposure to a known HIV-infected source was set up jointly by the Association of Medical Microbiologists and the PHLS Communicable Disease Surveillance Centre (CDSC) in 1984⁷; results for exposures reported to

Health care workers and HIV: surveillance of occupationally acquired infection in the United Kingdom

J Heptonstall
O N Gill
K Porter
M B Black
V L Gilbert

R147

Tuberculosis in the West Midlands, 1990-1991

I Blair
P Balfour

R154

'COVER' (Cover of vaccination evaluated rapidly): 27

J M White
S Leon
N T Begg

R158

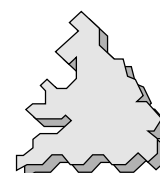


Table 1 Occupational exposure to HIV: points to cover in the incident record

<p>Exposed health care worker:</p> <ul style="list-style-type: none"> – name and code number[†], date of birth, sex – occupation – contact telephone number and address – name, address and telephone number of general practitioner – hepatitis B vaccine history – dates and results of tests for anti-HBs <p>Exposure:</p> <ul style="list-style-type: none"> – date and time of incident – date and time of report – place (eg, accident and emergency, clinic, ward) – brief description of incident (ie, what happened, how, and why) – procedure involved (eg, venepuncture, lumbar puncture) – material involved (blood/other body fluid/laboratory fluid) – exposure (percutaneous/mucocutaneous/other) – if percutaneous: type of sharp object involved (eg, scalpel blade, needle, trochar, teeth) 	<ul style="list-style-type: none"> – if needle: hollow or solid; gauge or size – if mucocutaneous: eyes/nose/mouth/non-intact skin (describe) <p>Source:</p> <ul style="list-style-type: none"> – identifiable/unknown – for identifiable sources: <ul style="list-style-type: none"> name/soundex code, hospital number, date of birth, sex results of tests for HBsAg and HBeAg/anti-HBe – for identifiable sources known or found to be HIV-infected: <ul style="list-style-type: none"> date of first positive anti-HIV test risk factors for HIV infection (exposure category) whether source was known to have AIDS at time of incident, and date of AIDS diagnosis date and result of most recent CD4 count antiretroviral therapy: type and dates of treatment whether exposed health care worker knew at time of incident that the patient was HIV-infected
--	--

[†] see text (this page).

December 1992 are presented here, together with details of definite and possible occupationally acquired HIV infections in UK health care workers derived from voluntary confidential reports of AIDS cases and newly diagnosed HIV infections reported to CDSC by December 1992.

Methods

Prospective surveillance

The surveillance system has evolved as more information about the risks of occupational exposure has become available, and currently operates as follows:

When a health care worker reports an occupational exposure incident, a detailed history is obtained and an incident form is completed by the local centre. Table 1 lists points that might usefully be covered in a local incident record. Standardised documentation facilitates risk assessment and incident management. For significant HIV exposure incidents, a confidential national report form is completed by the practitioner responsible for the health care worker's post exposure care and sent to CDSC. This report summarises the exposure incident, immediate management and the result of the health care worker's baseline test for HIV antibody. The name of the health care worker need not be reported; local codes, or the soundex code⁸, may be used as alternatives. As this code will be the identifier used in any subsequent follow up by CDSC, reporters are asked to ensure that it will be possible for them to link any code they use with their local incident record.

For the purposes of national surveillance, a significant HIV exposure is defined as one in which a health care worker sustains either:

- (i) a *percutaneous exposure to blood or body fluid from a source known or found to be HIV infected*; or
- (ii) a *percutaneous or mucocutaneous exposure to laboratory fluid known to contain live virus*; or
- (iii) a *mucocutaneous exposure to blood from a source known or found to be HIV-infected*.

Types of exposure

A *percutaneous exposure* is one in which the skin is cut or penetrated by a needle or other sharp object (eg, scalpel blade, trochar, tooth, bone spicule).

A *mucocutaneous exposure* is one which involves the eye(s), inside of the nose or mouth, or an area of non-intact skin of the person exposed.

An *HIV-infected source* is one for which a confirmed positive result of a test for HIV antibody or antigen has been documented or which is known to have fulfilled the current relevant World Health Organisation case definition for AIDS.

Current good practice for the management of significant exposure incidents is outlined in table 2. Details of the outcome of significant exposure incidents (including the results of tests for HIV antibody six weeks, three months, and at a minimum of six months after the exposure) are also reported to the surveillance system. Other exposures, which do not satisfy the above definitions, may be deemed by the local centre to present a risk of HIV transmission sufficient to justify follow up, but routine reporting to CDSC is not requested. These might include, for example, percutaneous exposure to the blood of an identifiable source patient who is known to have a major risk factor for HIV infection but whose HIV status remains undetermined, or percutaneous exposure to an unknown or unidentifiable source.

In 1992, twelve selected centres in London were contacted and asked to report details of any exposure incident known to them for which an incident report form had not been returned to the scheme.

Other reports of occupationally acquired HIV infection

The surveillance of AIDS and HIV in the UK has been described in detail previously⁹, as has the follow up of infections initially reported as being without identifiable risk¹⁰. For the small number of infections which might have been occupationally acquired, the following case definitions have been used:

Table 2 Management of significant occupational exposure to HIV

1.	Record details of exposure incident (Table 1)	
2.	Assess significance of exposure	
3a.	For exposures assessed as significant[†]	
	(i) Initial post-exposure management:	
	– provide post-exposure counselling to HCW, covering:	
	• assurance of confidentiality	
	• transmission risk from incident	
	• need for post-exposure zidovudine prophylaxis ²⁷	
	• pre-HIV test counselling	
	• access to further advice and support	
	• need for follow up at 6 weeks, 3 months, and at a minimum of 6 months post exposure	
	• need to report interim illness	
	• avoidance of further possible transmission (protected sexual intercourse; avoidance of pregnancy, blood, organ and semen donation)	
	• reassurance that follow up will not restrict employment	
	• advice about future insurance	
	– obtain blood specimen (5-10ml serum) from HCW exposed	
		and arrange HIV testing and storage of serum
		– if source identifiable and available for testing, obtain blood specimen (5-10ml serum) for HIV testing and storage
		– manage hepatitis B ³⁵ , hepatitis C ³⁶ and other risks appropriately
		– arrange follow up appointment for HCW
		– report details of incident to PHLS CDSC
	(ii) Post exposure follow up:	
	– provide continuing access to advice and support	
	– monitor effects of prophylaxis if given	
	– obtain follow up blood specimens (5-10ml serum) from HCW for anti-HIV test and serum storage at 6 weeks, 3 months, and at a minimum of 6 months post exposure	
	– report management and outcome to PHLS CDSC	
3b.	For other exposures:	
	– provide appropriate reassurance/follow up for HIV, consider other risks (hepatitis B, tetanus etc) and future hepatitis B exposure risk and manage appropriately ³⁶	

[†] see page R148 for definition of significant exposure to HIV.

- (i) *definite occupationally acquired HIV infection*: a health care worker with documented HIV seroconversion after a specific occupational exposure to a known HIV infected source.
- (ii) *possible occupationally acquired HIV infection*: a health care worker found to be HIV infected, without identified non-occupational risks for HIV infection, with a history of occupational percutaneous or mucocutaneous exposure to blood or body fluid from a source known to be HIV infected or with a history of percutaneous or mucocutaneous exposure to blood during employment in a high prevalence area.

Results

Prospective surveillance

In the period 1985-1992, 176 significant exposures were reported to CDSC (Table 3). Of these, 113 (64%) were reported from centres in London. In the same period, 45% of AIDS case reports and 36% of reports of newly diagnosed HIV-infected persons originated in these centres.

The outcome of 42 (24%) of the significant exposures (33 from London centres, 9 from elsewhere) remains unknown, because follow up reports were not received. For the remaining 134 exposures, which involved 134 health care workers, results of tests for HIV antibody at baseline and at three months post exposure were available. For 95 (71%) of these exposures, results of tests for HIV antibody at six months post exposure were also available.

Sixty-six (49%) of the 134 reported exposures occurred in the period 1984-86; 42 (31%) occurred in 1987-89, and 26 (19%) in 1990-92. Most (106, 79%) were percutaneous (Table 3). The occupations of the health care workers who sustained these exposures are shown in table 4. Ninety-nine (93%) of the 106 percutaneous exposures involved exposure to HIV-

infected blood or serum. Two of these exposures resulted in infection; an estimated seroconversion rate of 2% (95% confidence intervals 0.3% to 7.1%^{*}). One of these cases, given zidovudine post exposure, has been described in detail previously^{11,12} (case B, table 5). Further details of the other (case C) are shown in table 5.

Other reports of occupationally acquired HIV infection

Definite occupationally acquired HIV infections

Two other health care workers seroconverted after percutaneous exposure to HIV-infected blood in the UK between 1984 and 1992. One of these cases occurred before the surveillance system had begun, and has been reported previously¹ (case A, table 5). The other (case D, table 5) was reported as a newly diagnosed HIV infection by the physician responsible for the health care worker's subsequent medical care. Thus, four cases of documented seroconversion after occupational exposure to HIV in the UK have now been recorded.

Possible occupationally acquired HIV infections

There are six health care workers (two surgeons and four nurses) with possible occupationally acquired HIV infections. Four have been reported previously^{13,14}; one had possibly received a blood transfusion in Africa. The other five had no major identifiable risks for HIV infection. None of the six had reported a specific exposure incident, but all had jobs which involved occupational exposure to blood and all had cared for HIV-infected patients. Five of the six had worked in adverse conditions in African countries where HIV prevalence was high; the sixth had worked with HIV-infected patients in the United States and Europe and recalled several needlestick injuries.

^{*} confidence limits were estimated assuming a binomial distribution, or approximated to a normal distribution where appropriate.

Table 3 Prospective surveillance of occupational exposure to HIV infection: number of significant exposures initially reported (a) and with known outcome at 3 months post exposure (b)

Type of exposure	Year in which the exposure occurred										Total a b	Sero- conversions
	1984 a b	1985 a b	1986 a b	1987 a b	1988 a b	1989 a b	1990 a b	1991 a b	1992 a b			
Percutaneous												
blood	2 2	26 23	23 20	16 14	12 12	8 7	12 5	20 11	12 5		131 99	2/99
other	– –	3 3	1 1	2 2	– –	– –	– –	1 1	– –		7 7	0/7
Mucocutaneous	1 1	11 11	6 5	6 1	5 5	2 1	– –	6 4	1 –		38 28	0/28

Discussion

By September 1993, 64 documented seroconversions and 118 cases of possible occupationally acquired HIV infection in health care workers (including those reported here) had been recognised and reported worldwide, mainly from developed countries¹⁵ (Table 6). These reports suggest that the main hazard to health care workers is percutaneous exposure to HIV-infected blood, particularly exposure which involves fresh blood and hollow needles. Of the 32 documented seroconversions described in sufficient detail to allow evaluation, three quarters (24) resulted from such exposure¹⁵.

Although the observed seroconversion rate after percutaneous exposure to HIV-infected blood of 2% (95% confidence intervals 0.3% to 7.1%*), or 1 in 50, reported here is high, the sample size is small. The rate, however, is not inconsistent with previously reported estimates from larger (and therefore more powerful) studies, which have ranged from 0.18% (95% confidence intervals 0.02% to 0.64%)⁸ to 0.56% (95% confidence intervals 0.02% to 3.1%)¹⁶ (Figure 1^{2-4, 16-19}).

The estimation of transmission rates may be affected by biological factors, observational bias, and sampling variation. Biological factors include the volume of the inoculum and the concentration – and perhaps the strain – of virus present. Antigenaemia increases as disease progresses, but may be modified by antiretroviral therapy, which may also affect the efficacy of post-exposure prophylaxis. Observational bias may result from differences in thresholds for reporting exposure incidents, definitions of significant exposures, and in surveillance methods for case ascertainment. Exposures are probably ascertained most effectively through surveillance based in a single centre, or on the study of a cohort of workers recruited prospectively and tested for HIV at regular intervals, which perhaps explains the relatively high proportion of mucocutaneous exposures reported in such studies^{16,18}. Exposures will also be ascertained effectively through surveillance based on active collection of data from designated sentinel centres^{2,3}. When

reliance is placed on passive reporting of exposures by local centres which have not been specifically recruited, exposures which result in HIV transmission may be more likely to be reported than those which do not.

The decline in the number of significant exposure incidents reported to CDSC over time and the decline in the proportion with a known outcome at three months post exposure, during a period when the number of clinically diagnosed HIV-infected patients in the UK was increasing, suggest that under-ascertainment of exposure incidents may have occurred. This may explain why the observed transmission rate is relatively high (Figure 1). The seroconversion rate of 5.8% (3/52) observed in a small study in Natal, South Africa¹⁷, in a centre responsible for all local HIV testing, remains unexplained. One hypothesis would be that only exposures in some way more likely to result in seroconversion had been reported by health care workers in Natal.

Although the overall estimate of risk of seroconversion after percutaneous exposure to HIV has been assessed as around 1 in 300¹⁵, it could well be higher for certain subcategories of percutaneous exposure. None of the large studies of HIV transmission after percutaneous occupational exposure has yet sub-categorised seroconversion rates by the type of procedure during which the exposure occurred (eg, venepuncture, intramuscular injection, etc).

The occupationally acquired infections reported here should stimulate efforts to monitor and prevent percutaneous exposure to blood in the workplace. Three of the four UK health care workers in whom seroconversion was documented were known to have been using gloves at the time of exposure in accordance with current guidance²⁰. Two of the four exposures could, however, perhaps have been prevented by alterations in technique: that which occurred when blood was being transferred to an open specimen bottle from a disposable syringe with the needle still attached; and the injury sustained while resheathing a

Table 4 Occupation of workers with a known outcome at 3 months after significant occupational exposure to HIV

Type of exposure	Doctor	Nurse*	Surgeon	Dental staff†	Other	Not reported	Total
Percutaneous	34 (32%)	48 (45%)	6 (6%)	3 (3%)	12 (11%)	3 (3%)	106 (100%)
Mucocutaneous	6 (21%)	18 (64%)	3 (11%)	–	1 (4%)	–	28 (100%)

* includes midwives and nursing auxiliaries. † includes dentists and dental auxiliaries.

Table 5 Documented seroconversions in health care workers after occupational exposure to HIV in the United Kingdom, 1984-1992

	Case A*	Case B [§]	Case C	Case D
Interval between incident and report to CDSC (months)	18	3	4	10
Occupation	Nurse	Nurse	Phlebotomist	Not reported
Exposure[†]				
Elective or emergency	Elective	Emergency	Elective	Elective
Procedure	Arterial blood gases	IV cannula insertion	Venepuncture	Venepuncture
Material involved	Blood	Blood	Blood	Blood
Place	Ward	Ward	Clinic	Ward
Type of exposure	Percutaneous	Percutaneous	Percutaneous	Percutaneous
Site	Finger	Upper arm	Thenar eminence	Finger
Sharp object	Needle	Used introducer	Needle	Needle
Hollow/solid object	Hollow	Hollow	Hollow	Hollow
Gauge of sharp object	Not reported	18 or 20G	23G	21G
HCW wearing gloves	Not reported	Yes	No	Yes
Source patient				
Sex/age	Female adult	Female adult	Female child	Female adult
Exposure category	Heterosexual	Heterosexual	Vertical, heterosexually infected parent	Heterosexual
Time since HIV infection diagnosed	–	20 months	4 years	15 months
AIDS case	Yes	Yes	No	Yes
Time since AIDS diagnosed	Not reported	20 months	–	15 months
CD4 count at time of incident	Not known	<50/μl	Not known	<10/μl
Antiviral therapy	No	On zidovudine	No	Zidovudine in past
HIV status known to exposed HCW	Yes	Yes	No	Yes
Incident management				
Time to beginning care	Not reported	< 1 hour	< 1 hour	< 1 hour
Zidovudine (ZDV) given	No	Yes	No	No
Time to first dose ZDV	–	1 hour	–	–
Dosage	–	250mg qds	–	–
Days	–	42	–	–
Outcome post exposure				
Baseline anti-HIV test	Negative (day 27)	Negative (day 0)	Negative (day 4)	Negative (day 0)
Last negative test	Day 27	Day 43	Day 4	Day 0
First positive test	Day 49	Day 56	3 months	Day 81
Seroconversion illness	Days 13-33	Days 13-28	Pruritus only, 3rd week	6th week

* See reference 1 for further details. [§] See reference 12 for further details. [†] One incident (case A) occurred while resheathing a needle on a syringe containing blood from an arterial line. One of the two previously unreported cases occurred after injury when blood was being transferred to an open specimen bottle from a disposable syringe with the needle still attached; the other followed attempted venepuncture on a restless patient.

needle after arterial blood gas sampling¹.

Measures developed to prevent hollow needle injuries have not yet effected a sustained reduction in the rate at which such injuries occur²¹. Post-disposal injuries may be prevented by the introduction of sharps disposal boxes, provided that the boxes are properly placed and correctly used^{22,23}. Evidence on the value of campaigns to reduce needle recapping is, however, conflicting. In one study in the United States, reported injuries due to needle recapping were reduced by 50%²⁴; another reported a threefold increase in such injuries between 1975-9 and 1987-88, though this was in part attributable to increased reporting and increased potential for exposure²⁵; there may be little difference in injury rates between workers who normally recap needles

and those who normally do not^{21,26}.

There remains a need for effective post exposure prophylaxis. Present UK guidance²⁷ reiterates that of the Centers for Disease Control, Atlanta²⁸; stating that zidovudine cannot be considered a necessary part of post exposure prophylaxis, but requires districts to formulate local policy on its use. One of the UK seroconversions occurred following the use of zidovudine post exposure. The source patient had received zidovudine therapy and the virus subsequently isolated from the health care worker was relatively zidovudine resistant¹¹. Seroconversion following zidovudine prophylaxis has now been reported in eight health care workers (including the one above). Three had been exposed to blood from source patients

who had never received zidovudine; one of these health care workers was shown to have become infected with a strain apparently sensitive to zidovudine¹⁹. Transmission of relatively resistant virus, therefore, cannot provide a complete explanation for the ineffectiveness of zidovudine prophylaxis. Five instances of failure of post-exposure zidovudine prophylaxis in other circumstances (accidental intravenous injection of HIV infected material, self-inoculation, assault, and blood transfusion) have also been reported²⁸⁻³². Experimental prophylactic regimens may be developed as progress in antiviral therapy is made, and will require surveillance. Didanosine has already been used in combination with zidovudine post exposure³³. As the modes of action of the two drugs are similar there is no reason to suppose that such a combination could be more efficacious but every reason to expect increased toxicity.

Surveillance of occupational exposure, especially when complete, should provide the information necessary to fulfil some of the requirements of the Health and Safety at Work Act 1974, the Control of Substances Hazardous to Health Regulations 1988, and the Management of Health and Safety Regulations 1992, which impose upon employers a duty to perform a competent assessment of risk, to inform and train staff, to monitor and review procedures, and to reduce risks as far as practicable³⁴. It is apparent that variations in post exposure management presently exist; current good practice is summarised in table 2^{20,27,35,36}. Incident outcome reporting (at a minimum of six months post exposure) is integral to satisfactory post-exposure management, and might usefully be thought of as the final stage of good post-exposure care. Health care workers deserve to receive post-exposure care of a uniformly high standard. Knowing that this was available and readily accessible might encourage workers to report exposures.

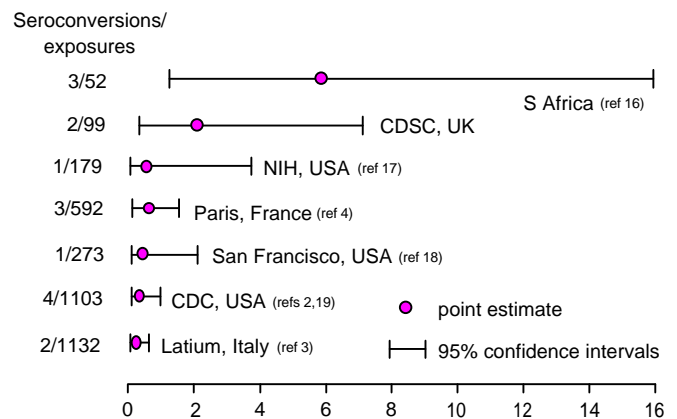
While the further evaluation and refinement of devices and instruments designed with safety in mind are awaited³⁷, we need to discover more about the circumstances in which percutaneous injuries occur, and the practices and procedures with which they are associated. The risks of transmission are low, so it is difficult to study procedure-based transmission risks and the effects of any available prophylaxis, change in practice or technique; it may not, however, be impossible. Indeed, with standardisation of surveillance definitions and data presentation, harmonisation of surveillance systems, more complete incident reporting and follow up, and international collaboration, it should be achievable.

Table 6 Reported occupationally acquired HIV infections in health care workers, to September 1993¹⁵

	USA	UK*	Rest of world	Total
Documented seroconversion after specific occupational exposure	37	4	23	64
Possible occupationally acquired infection	78	6	34	118
Total	115	10	57	182

* See text for details.

Figure 1 Published estimates of the HIV transmission rate after percutaneous occupational exposure



Acknowledgements

We thank those who have reported occupational exposures to CDSC, our counterparts at the Communicable Diseases (Scotland) Unit through whom exposures that occurred in Scotland were reported, and those who have allowed us to report the details of documented seroconversions and possible occupationally acquired infections.

References

1. Anon. Needlestick transmission of HTLV-III from a patient infected in Africa. *Lancet* 1984; **ii**: 1376-7.
2. Marcus R, CDC Cooperative Needlestick Surveillance Group. Surveillance of health care workers exposed to blood from patients infected with the human immunodeficiency virus. *N Engl J Med* 1988; **319**: 1118-23.
3. Ippolito G, Puro V, De Carli G and the Italian Study Group on Occupational Risk of HIV infection. Ninth International Conference on AIDS, Berlin, June 1993: abstract PO-C18-3021.
4. Lot F, Abiteboul D, Bouvet E, Laporte A. Surveillance of occupationally acquired HIV infections in France. Ninth International Conference on AIDS, Berlin, June 1993: abstract PO-C18-3039.
5. Centers for Disease Control and Prevention. Health care workers with documented and possible occupationally acquired AIDS/HIV infections, by occupation, reported through June 1993, United States. *HIV/AIDS Surveillance Report*, 1993; **5**(no. 2):13.
6. Castro KG, Lifson AR, White CR, Bush TJ, Chamberland ME, Lekatsas AM, et al. Investigations of AIDS patients with no previously identified risk factors. *JAMA* 1988; **259**: 1338-42.
7. CDSC. Surveillance of health care staff: HTLV 3. *Communicable Disease Report* 1984; (52): 1.
8. Goehring R. Identification of patients in medical data bases - soundex codes versus match code. *Med Inform* 1985; **10**: 27-34.
9. PHLS AIDS Centre. The surveillance of HIV-1 infection

- and AIDS in England and Wales. *Communicable Disease Report* 1991; **1**: R51-6.
10. Evans BG, Noone A, Mortimer JY, Gilbert VL, Gill ON, Nicoll A, et al. Heterosexually acquired HIV-1 infection: cases reported in England, Wales and Northern Ireland, 1985 to 1991. *Communicable Disease Report* 1992; **2**: R49-55.
 11. Anon. HIV seroconversion after occupational exposure despite early prophylactic zidovudine therapy. *Lancet* 1993; **341**: 1077-8.
 12. Wincelhaus J. HIV seroconversion after occupational exposure despite early prophylactic zidovudine therapy. (Anonymous reply to letter). *Lancet* 1993; **341**: 1537.
 13. Porter JD, Cruickshank JG, Gentle PH, Robinson RG, Gill ON. Management of patients treated by surgeon with HIV infection. *Lancet* 1990; **335**: 113-14.
 14. Fernando R, Terry P, Willmott F. Midwifery and body fluid contamination. *BMJ* 1992; **305**: 713.
 15. Heptonstall J, Porter K, Gill ON. Occupational transmission of HIV. Summary of published reports – September 1993. Internal report of the PHLS, available from PHLS AIDS Centre at CDSC.
 16. Henderson DK, Fahey BJ, Willy M, Schmitt JM, Carey K, Koziol DE, et al. Risk for occupational transmission of human immunodeficiency virus type 1 (HIV-1) associated with clinical exposures: a prospective evaluation. *Ann Intern Med* 1990; **113**: 740-6.
 17. Tait DR, Pudifin DJ, Gathiram V, Windsor IM. HIV seroconversions in health care workers, Natal, South Africa. Eighth International Conference on AIDS, Amsterdam, July 1992: abstract PoC 4141.
 18. Gerberding JL, Littell C, Brown A, Sande M. Long term follow up of health care workers with polymerase chain reaction and antibody testing to detect delayed seroconversion. Sixth International Conference on AIDS, San Francisco, June 1990: abstract Th.C.601.
 19. Tokars JJ, Marcus R, Culver DH, Schable CA, McKibben PS, Banda CI, et al for the CDC Cooperative Needlestick Surveillance Group. Surveillance of HIV infection and zidovudine use among health care workers after occupational exposure to HIV-infected blood. *Ann Intern Med* 1993; **118**: 913-18.
 20. Anon. Needlesticks: preaching to the seroconverted? *Lancet* 1992; **340**: 640-2.
 21. UK Health Departments. *Guidance for clinical health care workers: protection against infection with HIV and hepatitis viruses*. London: HMSO, 1990.
 22. Krasinski K, LaCouture R, Holzman RS. Effect of changing needle disposal systems on needle puncture injuries. *Infect Control* 1987; **8**: 59-62.
 23. Haiduvan DJ, DeMaio TM, Stevens DA. A five-year study of needlestick injuries: significant reduction associated with communication, education, and convenient placement of sharps containers. *Infect Control Hosp Epidemiol* 1992; **13**: 265-71.
 24. Gerberding JL. Does knowledge of human immunodeficiency virus infection decrease the frequency of occupational exposure to blood? *Am J Med* 1991; **91**(suppl 3B): 308S-311S.
 25. McCormick RD, Meisch MG, Ircink FG, Maki MD. Epidemiology of hospital sharps injuries: a 14-year prospective study in the pre-AIDS and AIDS eras. *Am J Med* 1991; **91**(suppl 3B): 301S-307S.
 26. Choudhury RP, Cleator SJ. An examination of needlestick injury rates, hepatitis B vaccination uptake and instruction on "sharps" technique among medical students. *J Hosp Infect* 1992; **22**: 143-8.
 27. UK Health Departments. Occupational exposure to HIV and use of zidovudine. A statement from the Expert Advisory Group on AIDS. May 1992.
 28. Centers for Disease Control. Public Health Service statement on management of occupational exposure to human immunodeficiency virus, including considerations regarding zidovudine post exposure use. *MMWR* 1990; **39**: RR-1.
 29. Lange JMA, Boucher CAB, Hollak CEM, Wiltink EHH, Reiss P, van Royen EA, et al. Failure of zidovudine prophylaxis after accidental exposure to HIV-1. *N Engl J Med* 1990; **322**: 1375-7.
 30. Centers for Disease Control. Patient exposures to HIV during nuclear medicine procedures. *MMWR* 1992; **41**: 575-8.
 31. Durand E, Le Jeunne C, Hugues F-C. Failure of prophylactic zidovudine after suicidal self-inoculation of HIV-infected blood. *N Engl J Med* 1991; **324**: 1062.
 32. Jones PD. HIV transmission by stabbing despite zidovudine prophylaxis. *Lancet* 1991; **338**: 884.
 33. Malcolm JA, Dobson PM, Sutherland DC. Combination chemoprophylaxis after needlestick injury. *Lancet* 1993; **341**: 112-3.
 34. Macdonald EB. Health workers need protection. *BMJ* 1993; **306**: 1202.
 35. PHLS Hepatitis Subcommittee. Exposure to hepatitis B virus: guidance on post-exposure prophylaxis. *Communicable Disease Report* 1992; **9**: R97-101.
 36. PHLS Hepatitis Subcommittee. Hepatitis C virus: guidance on the risks and current management of occupational exposure. *Communicable Disease Report* 1993; **3**: R135-9.
 37. Anon. Needle safety update. *Hosp Infect Control* 1993; **20**: 49-57.

J Heptonstall MRCPATH

Hepatitis Section

O N Gill FFPHM

K Porter MSc

M B Black MFPHM

V L Gilbert RGN

PHLS AIDS Centre

PHLS Communicable Disease Surveillance Centre

Tuberculosis in the West Midlands, 1990-1991

I Blair, P Balfour

Summary

In the late 1980s, notifications of tuberculosis stopped their former steady decline. There has been speculation as to why this should be so, with much interest centred on a possible association with the HIV epidemic. Notification rates are higher in persons of Indian subcontinent ethnic origin compared with the indigenous white population. Changes in the size and structure of the former population subgroup may have contributed to the recent increase in notifications in some areas. The absence of data on ethnic group in routinely collected data has led to the recommendation that special surveys should be conducted to clarify the contribution of ethnic minorities to the occurrence of tuberculosis in the UK. One such survey has been carried out in the West Midlands, where notifications increased by 27% between 1987 and 1989. Notification rates were found to vary widely by age, sex, district of residence and ethnic group; the highest notification rates occurring in older females of Indian subcontinent origin. These differences help to explain the increase in the absolute number of notifications and suggest that certain population subgroups warrant special attention.

Introduction

Notifications of tuberculosis in England and Wales declined until 1987, but have risen every year since then (from 5086 to a provisional total of 5861 in 1992)^{1,2}. It is not certain why this should be so. It has been suggested that this may be due to improved reporting of diagnosed cases¹, particularly as the numbers of deaths from tuberculosis and the number of laboratory reports of *Mycobacterium tuberculosis* to the PHLS Communicable Disease Surveillance Centre (CDSC) have continued to fall. It is possible that the rising number of cases is due to increases in the size of population sub-groups which are at increased risk of tuberculosis. These sub-groups include the elderly, those who are immunocompromised (including those with HIV infection), the homeless, and immigrants who have migrated from countries which have a high incidence of tuberculosis¹.

Increases in tuberculosis notifications have been observed in both sexes; in both younger and older adults, and in several regions of the country (particularly those that have had above average notification rates in the past)³. In the West Midlands, notifications increased from 663 to 843 between 1987 and 1989. Notifications in females aged 20-34 years accounted for 30% of that increase (62 in 1987 to 116 in 1989) while notifications in females aged 45-74 years accounted for 25% of the increase (105 in 1987 to 150 in 1989).

It is difficult to assess the contribution made by different ethnic groups to these recent increases, as notifications do not include data on ethnicity. However, a special survey⁴ has shown that notifications in the West Midlands, for which ethnic group was known, increased from 640 to 791 between 1987 and 1989. Notifications in persons of Indian subcontinent (ISC) origin accounted for 67% of this increase.

It is unlikely that the HIV epidemic is responsible for the

increase in tuberculosis observed in England and Wales in recent years. However, heightened surveillance of tuberculosis has been recommended to assess the impact of the HIV epidemic and the contribution of ethnic minorities to the occurrence of tuberculosis³. A national survey of tuberculosis notifications started this year, co-ordinated from CDSC in collaboration with the British Thoracic Society and the Department of Health⁵. This survey is collecting data on ethnic group, place of birth, type of disease and outcome.

We describe a similar survey of tuberculosis notifications, involving local consultants in communicable disease control, that was carried out in the the West Midlands region in 1992.

Methods

Consultants in communicable disease control (CCDCs) in the West Midlands were asked to provide a detailed dataset (Table 1) on every case of tuberculosis notified to them in 1990 and 1991. To assist with data collection and collation they were provided with a database written in Epi Info software. The software required ethnic group to be classified according to the system devised for use in the 1991 Census. Data for the four Birmingham health districts were supplied by the Director of the Birmingham Chest Clinic.

Data on age, sex and ethnic group distribution of the population at risk were obtained from the 1991 Census. Notification rates were calculated using the mean number of notifications in 1990 and 1991 as the numerator and the 1991 census population as the denominator.

Results

Data were supplied by CCDCs (or their staff) on cases of tuberculosis notified in 1990 and 1991 for 15 of the present 19 district health authorities (DHAs) in the West Midlands region. There was a total of 1662 cases. Census data on the age and ethnic group distribution of the resident population were unavailable for two DHAs and notifications from these DHAs were excluded. Two hundred and forty-seven notifications of persons receiving chemoprophylaxis were also excluded. The data presented here are based on the 1314 remaining notifications from 13 DHAs.

The absolute numbers of notifications and notification rates were highest for urban districts, such as Birmingham,

Table 1 Data collected on notified cases of tuberculosis

Sex	Year of entry
Date of birth	Special risk factors
Age	Previous treatment
Occupation	Previous BCG
Ethnic group	Date of BCG
Postcode	Site of disease
DHA of residence	Organs involved
Contact examination	Sputum smear result
LA of residence	Sputum culture result
Date of notification	Organism
Country of birth	Outcome

DHA – district health authority. LA – local authority.

Table 2 Tuberculosis notifications by sex and district for 1990 and 1991

District	Number of notifications (rate per 100,000 population)				Total
	1990		1991		
	Female	Male	Female	Male	
Birmingham (4 districts)	148 (30)	142 (29)	159 (31)	171 (35)	620
Coventry	32 (21)	38 (25)	46 (30)	40 (27)	156
Dudley	28 (18)	24 (16)	16 (10)	23 (15)	91
Mid-Staffordshire	7 (4)	11 (4)	5 (3)	7 (4)	30
North Staffordshire	18 (8)	24 (11)	21 (9)	20 (9)	83
North-East Warwickshire	4 (3)	8 (6)	8 (6)	12 (9)	32
South Warwickshire	6 (5)	7 (6)	8 (7)	5 (6)	26
Solihull	1 (1)	5 (5)	3 (3)	4 (4)	13
Walsall	20 (15)	29 (22)	37 (28)	27 (21)	113
Wolverhampton	31 (25)	41 (33)	37 (29)	41 (34)	150
Total	295	329	340	350	1314

Cases from two health authorities for which denominator data were unavailable have been excluded. Cases receiving chemoprophylaxis have also been excluded.

Coventry and Wolverhampton, and lowest for more rural areas such as Solihull, South Warwickshire and Mid-Staffordshire (Table 2). Age, sex and ethnic group were known for 1303 notifications (Table 3). Similar numbers of males (673) and females (630) were notified across all ethnic groups. In white ethnic groups, males outnumbered females, particularly in those aged 35 years or more. In those of Indian and Pakistani ethnic origin, females outnumbered males, particularly in the 15-64 year age group. Five hundred and twenty-two notifications (40%) occurred in those aged under 35 years and 125 (10%) in the 0-14 year age group.

Notification rates were low among whites in the younger age groups but increased with age, particularly among males (Table 4). The rates among non-white ethnic groups were higher at all ages compared with notification rates in whites. Notification rates were 7-10 times higher in those aged 0-4

years of Indian ethnic origin than in whites of similar age. Females of Indian ethnic origin aged 5-14 years had a notification rate 21 times higher than white females of similar age. Compared with white females of similar age, notification rates in females of Pakistani and Bangladeshi ethnic origin were 50 times higher in those aged 15-34 years; 68 times higher in those aged 35-64 years, and 89 times higher in those aged over 65 years.

The site of disease and age were known for 1129 notifications (Table 5). Of these, lung (66%) and lymph nodes (17%) were the most commonly reported sites. Site and ethnic group were known for 1124 notifications (Table 6). Pulmonary and genito-urinary disease was more commonly, and lymph node and gastrointestinal disease less commonly, reported in whites than in the other ethnic groups. The exception was genito-urinary tuberculosis; 23 of 30 reports were in whites.

Table 3 Tuberculosis notifications by sex and ethnic group for 13 West Midlands districts

Ethnic group	Age group (years)											
	0-4		5-14		15-34		35-64		≥65		All ages	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
White	12	8	11	10	35	42	103	67	112	65	273	192
Pakistani	2	3	15	5	51	80	56	79	17	13	141	180
Indian	5	6	6	16	59	73	66	75	28	30	164	200
Black African and Black Caribbean	3	3	6	6	22	8	17	3	3	–	51	20
Bangladeshi	–	–	1	–	4	2	7	8	1	1	13	11
Chinese	–	–	–	–	1	1	–	1	1	1	2	3
Other	3	1	3	–	11	8	5	4	7	11	29	24
Total	25	21	42	37	183	214	254	237	169	121	673	630

Table 4 Tuberculosis notification rates (per 100,000 population per year) by age and ethnic group

Ethnic group	Notification rates (95% confidence intervals)											
	0-4 years		5-14 years		15-34 years		35-64 years		≥65 years		All ages	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
White	5.8 (2.5-9.1)	4.1 (1.3-6.9)	2.9 (1.2-4.6)	2.8 (1.1-4.5)	3.9 (2.6-5.2)	4.7 (3.3-6.1)	8.9 (7.2-11)	5.8 (4.4-7.2)	26 (21-31)	10 (7.8-13)	8.9 (7.8-10)	5.9 (5.1-6.7)
Pakistani & Bangladeshi*	14 (0-33)	22 (0-46)	51 (26-76)	17 (2.1-32)	165 (121-209)	237 (186-288)	259 (195-322)	394 (311-477)	692 (373-1010)	921 (441-1401)	145 (122-168)	187 (161-214)
Indian	42 (5.3-79)	53 (11-95)	21 (4.3-39)	60 (31-90)	125 (93-157)	150 (116-185)	163 (124-203)	191 (148-234)	520 (328-713)	601 (386-815)	124 (105-142)	153 (132-174)
Black [§]	31 (0-65)	31 (0-65)	38 (7.6-68)	40 (7.9-71)	72 (42-101)	22 (6.8-37)	69 (36-102)	13 (0-27)	56 (0-120)	–	59 (43-75)	23 (13-32)

* Only one DHA appeared to differentiate between persons of Pakistani and Bangladeshi origin. A combined rate has therefore been calculated.

§ All DHAs appeared to have difficulty in differentiating between persons of black Caribbean, black African and other black ethnic groups. A combined rate has therefore been calculated for this grouping.

Discussion

The data show that districts in the West Midlands region vary greatly in the extent to which the population is affected by tuberculosis. Urban districts have the highest absolute numbers of notifications and also the highest notification rates. Urban districts tend to encompass areas that are socially and economically disadvantaged and have sizeable ethnic minority populations.

Surveys that attempt to estimate ethnic group specific rates of disease face difficulties either in assigning cases to the correct ethnic group or in obtaining accurate denominator data. CCDCs were confronted by the first of these difficulties in this study, particularly because of its retrospective design. Errors due to mis-classification were minimised by aggregating cases into four main ethnic groupings when rates were calculated. Accurate denominator data were available from the 1991 Census. The 1988 National Survey⁶, which used denominator data from the National Labour

Force Survey, concluded that notification rates had declined during the 1980s but that major differences remained between ethnic groups, with rates in persons of Indian subcontinent origin up to 25 times greater than those in the white population.

The present survey was not able to examine trends in notification rates but did permit age and ethnic group specific rates to be calculated. Although some of these rates are calculated from small numbers, and therefore have wide confidence limits, they have confirmed important differences between ethnic groups. For certain age groups these differences are many times greater than the differences highlighted by the 1988 National Survey. Compared with whites, notification rates were higher in non-white ethnic groups for all age groups. Notification rates increased with age in all ethnic groups, with very high rates observed in elderly persons of ISC ethnic origin.

The high notification rates in older females of ISC ethnic origin have implications for local strategies to prevent and

Table 5 Tuberculosis notifications by site of disease and age group

Site	Age group (years)					Total (%)
	0-4	5-14	15-34	35-64	≥65	
Lung	22	49	210	279	186	746 (66)
Lymph nodes	8	18	70	75	17	188 (17)
Bone and joints	2	2	7	14	8	33 (3)
Meningitis, miliary	3	–	6	12	6	27 (2)
Gastrointestinal	–	–	15	17	1	33 (3)
Genito-urinary	–	1	4	15	10	30 (3)
Other	4	2	25	32	9	72 (6)
Total (%)	39 (3)	72 (6)	337 (30)	444 (39)	237 (21)	1129 (100)

Table 6 Tuberculosis notifications by site of disease and ethnic group

Site of disease	Number of notifications (%) [*]							Total (%)
	White	Pakistani	Indian	Black [§]	Bangladeshi	Chinese	Other	
Lung	292 (77)	177 (62)	177 (56)	51	10	4	34	745 (66)
Lymph nodes	32 (8)	68 (24)	66 (21)	6	7	–	7	186 (17)
Bone and joints	10 (3)	8 (3)	10 (3)	4	1	–	–	33 (3)
Meningitis, miliary	7 (2)	4 (1)	14 (4)	–	–	–	2	27 (2)
Gastrointestinal	2 (0.5)	9 (3)	14 (4)	3	3	–	1	32 (3)
Genito-urinary	23 (6)	2 (1)	3 (1)	–	1	–	1	30 (3)
Other	15 (4)	17 (6)	33 (10)	3	1	1	1	71 (6)
Total (%)	381 (100)	285 (100)	317 (100)	67	23	5	46	1124 (100)

^{*} The figures in parentheses are the proportions of notifications for each site of disease. [§] See footnote to table 4.

control tuberculosis. This is a population sub-group where efforts should be made to ensure that cases are diagnosed and treated promptly, contacts are identified and examined, and all cases of tuberculosis are notified to the CCDC so that local and regional surveillance can be maintained. This finding also supports the proposal that the recent increases in tuberculosis in the West Midlands are due in part to increases in the size of population sub-groups with an increased risk of tuberculosis. Continued surveillance will be necessary to monitor trends in notification rates and the contribution of ethnic minorities to the occurrence of tuberculosis in the West Midlands.

The 1988 National Survey⁶ reported variations in the site of disease according to ethnic origin. Among whites, 80% had only respiratory disease, 16% had only non-respiratory disease and 4% had both. The corresponding proportions amongst those of ISC ethnic origin were 53%, 36% and 11%. The study reported here confirms that those of Pakistani and Indian ethnic origin are more likely to have non-pulmonary disease compared with whites and that this usually involves lymph nodes.

Most CCDCs were willing to co-operate with the study and either they or their staff were able to provide the required data. Three of the four DHAs that did not contribute data to the study did not have a CCDC in post at the time of the study. Of these four DHAs, three are relatively affluent, rural districts where the occurrence of tuberculosis is low. Most CCDCs made use of the computer software that was provided. CCDCs have been encouraged to continue to use the software prospectively as a tuberculosis register and this should enable a similar surveillance exercise to be carried out in the future.

In recent years, rising numbers of notifications have signalled the re-emergence of tuberculosis as a public health problem for the 1990s. Tuberculosis is no different from any other infectious disease in that surveillance is an essential precursor of any programme of control measures. Although tuberculosis is a notifiable disease, the data collected often lack important details. This has led to periodic national surveys, one of which is currently underway. One of the shortcomings of national surveys is that they do not develop and sustain local surveillance arrangements.

In the West Midlands, efforts have been made to ensure

that tuberculosis surveillance involves the CCDC at local level. This has been successful and the West Midlands model of tuberculosis surveillance is commended to other regions.

Acknowledgements

We thank Dr J Innes, the CCDCs and their staff for their assistance with this survey.

References

1. CDSC. Tuberculosis in England and Wales. *Communicable Disease Report* 1993; **3**: 85.
2. Watson JM. Tuberculosis in perspective. *Communicable Disease Report* 1991; **1**: R129-32.
3. Watson JM, Fern KJ, Porter JDH, Whitmore SE. Notification of tuberculosis in England and Wales, 1982-1989. *Communicable Disease Report* 1991; **1**: R13-16.
4. Cooper C. Notifications of tuberculosis in the West Midlands 1987-1989. Department of Public Health Medicine, Sandwell Health Authority, West Midlands. Unpublished report 1991.
5. CDSC. National survey of tuberculosis notifications. *Communicable Disease Report* 1993; **3**: 1.
6. Medical Research Council Cardiothoracic Epidemiology Group. National survey of notifications of tuberculosis in England and Wales in 1988. *Thorax* 1992; **47**: 770-5.

I Blair MFPHM

School of Postgraduate Medical Education

University of Warwick

P Balfour MB ChB MSc

Department of Public Health Medicine

Stockport Health Authority

'COVER' (Cover of vaccination evaluated rapidly): 27

The COVER programme, a scheme for the rapid evaluation of vaccine coverage, started in January 1987 with 14 districts contributing data (*Communicable Disease Report* 1987; (12): 3-6). This twenty-seventh quarterly report includes information from 186 districts.

Methods

Data were collected at the beginning of August 1993 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, mumps and rubella (MMR) vaccine. 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 April to June 1992 (for D3 and P3 by 12 months), October to December 1991 (for D3 and P3 by 18 months) and April to June 1991 (for MMR by 24 months).

Results

Altogether, 186 districts participated from 14 English regions, Wales and Northern Ireland. Data were available for every district 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 **94%** for D3 (district range 82-99%) and **91%** for P3 (district range 81-97%). Cover by 18 months was **95%** for D3 (district range 86-100%) and **93%** for P3 (district range 85-98%). For MMR, average cover by 24 months was **93%** (district range 77-98%).

Comment

We report COVER data from 186 of 193 (96%) districts in England, Wales and Northern Ireland. Vaccine coverage for P3 (at 18 months) has improved by 1% since the previous report¹. Coverage for all other sentinel antigens has remained unchanged.

The 95% target was achieved by 104 districts for D3 (at 12 months), by 40 districts for P3 (at 12 months) and by 70 districts for MMR. Two districts (North West Durham and West Cumbria) achieved 100% coverage, and a further thirteen districts (Cambridge, Chichester, Exeter, Hartlepool, Huddersfield, Mid Essex, North Bedfordshire, Northallerton, Norwich, South Cumbria, South West Durham, Southern Health Board and Worthing) recorded 99% coverage for D3 (at 18 and/or 12 months). The lowest reported district coverage was 77% for MMR, 82% for D3 (at 12 months) and 81% for P3 (at 12 months).

This report includes further preliminary data on coverage for Hib 3 in South-East Thames and Wales¹. Coverage for Hib 3 at 11 months in South-East Thames was 2% lower than D3 but only 1% lower than P3 (Table 1); in Wales, it was 1% lower than D3, but 3% greater than P3.

*The regional breakdown of these data is available in request.

Table 1 Diphtheria, pertussis and Hib vaccination coverage at 5, 8 and 11 months (February, May and August 1993)

Region	Age at time of evaluation	Percentage vaccinated by evaluation date		
		D3	P3	Hib 3
SE Thames	5 months	53%	53%	51%
	8 months	85%	83%	82%
	11 months	89%	88%	87%
Wales	5 months	74%	71%	70%
	8 months	89%	85%	87%
	11 months	93%	89%	92%

Study cohort comprised 15,043 children born between 1 August and 30 September 1992.

Developments in future COVER reports

The next COVER report will include the first national evaluation of Hib 3 (at 12 months). The 18 month evaluation of D3 and P3 will be discontinued, bringing COVER data into line with World Health Organisation standards for evaluating coverage at 12 and 24 months².

Changes in district boundaries over the last two years have led to the formation of large districts in some regions and, consequently, some sensitivity in COVER data has been lost. To date, COVER reports have retained old district definitions unless the use of a new district boundary has been specifically requested. From February 1994, new district configurations as at 1 April 1993 will be used routinely for all COVER tables. Coverage data for old district configurations will be available on an *ad hoc* basis where data are supplied in an appropriate format.

The timing of COVER and Department of Health Korner requests (KC51), and the extra work these information requests generate, are under review. The aim is to integrate the two systems by matching evaluation dates, cohort, denominator and numerator definitions. An implementation date for full integration has not yet been agreed.

References

1. White JM, Leon S, Begg NT. 'COVER' (Cover of vaccination evaluated rapidly): 26. *Communicable Disease Report* 1993; **3**: R117-8.
2. Guérin N, Thiers G. *Eurepi - research in methodologies of immunization programmes management in Europe*. Paris: Centre International de l'Enfance, 1992: 40.

J M White BSc

S Leon

N T Begg FFPHM

Immunisation Division

PHLS Communicable Disease Surveillance Centre