PostScript

LETTERS

Perceived transmissibility of STIs: lack of differentiation between HIV and chlamydia

Sexually transmitted infections (STIs), such as HIV and chlamydia, differ widely in their transmissibility. The estimated probability of HIV transmission from an infected heterosexual man to a woman in one act of unprotected vaginal intercourse is 0.1%,¹ whereas the same probability for chlamydia is 35%.² This research examines college students' knowledge about the per act transmission probabilities for HIV and chlamydia.

Previous studies reported median perceived transmission probabilities of 50%³ and 33.4%⁴ for HIV for one act of unprotected receptive vaginal intercourse with an infected man. These findings were interpreted as demonstrating "badly overestimated per act transmission probabilities" (Pinkerton *et al.* p 19). However, the distributions of the estimates were not provided. If estimates are widely dispersed across the entire probability range from 0% to 100%, interpretations of averages are meaningless and interpreting the data as indicating a systematic overestimation of the transmission probability would be unfounded. We studied this possibility in a sample of college students.

In all, 234 undergraduate university students (145 women, 85 men, mean age 21.14 years, SD 2.82, four did not report their age and sex) enrolled in a variety of academic programmes were randomly selected and individually approached after classes. Aside from their age and sex, participants were asked in two separate questions: "What do you think is the probability, in percentages, of a woman becoming infected with HIV (chlamydia) from one unprotected act of vaginal intercourse with an infected man?" The order in which people were asked the two questions was counterbalanced.

Figure 1 presents the distribution of the estimates, showing that they are widely and quite equally dispersed across the entire range from 0% to 100%, and that distributions do not differ between the two infections (Kolmogorov-Smirnov $Z = -0.73$, $p = 0.46$).

No age or sex differences were found. Only 3.9% and 5.6% of the estimates for HIV and chlamydia, respectively, come close to the correct probabilities if "correct" is defined as smaller than 0.5% for HIV and between 30% and 40% for chlamydia. In all, 34.8% of the participants falsely estimate that chlamydia has a lower transmission probability than HIV, 39.5% correctly estimate that chlamydia has a higher transmission probability than HIV, and 25.8% provide exactly the same percentage estimate for both STIs.

The data show that a large majority of college students clearly lacks knowledge of the transmission probabilities of HIV and chlamydia and does not know that chlamydia is more infectious than HIV. Previous reports of statistical averages of the perceived transmissibility^{3,4} and their interpretation as indicating a systematic overestimation bias may be unfounded. The results highlight the importance of inspecting response distributions and restraining from reporting statistical averages when distributions are widely dispersed. Furthermore, they highlight that information about transmission probabilities should be incorporated into sexual health programmes in order to make people more aware of STIs that are considerably easier to contract than HIV.

Acknowledgements

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Contributors

The study was jointly conceptualised and designed by BK and RK; data were collected by RK, with the assistance of Surkhraj Cheema; BK analysed the data and led the writing; both authors jointly interpreted the findings, reviewed drafts of the manuscript, and approved the final version.

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Figure 1 Distribution of estimates for HIV and chlamydia for one act of unprotected vaginal intercourse with an infected man.

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References

- 1 Mastro TD, de Vincenzi I. Probabilities of sexual HIV-1 transmission. *AIDS* 1996;10(Suppl A):S75-82.
- 2 Katz BP, Caine, Jones RB. Estimation of transmission probabilities for chlamydial infection. In: *International symposium on human chlamydial infections. Chlamydial infections proceeds of the seventh international symposium on human chlamydial infections*. New York: Cambridge University Press, 1990:567-70.
- 3 Linville PW, Fischer GW, *et al.* AIDS risk perceptions and decision biases. In: Pryor JB, Reeder GD, eds. *The social psychology of HIV infection*. Hillsdale, NJ: Erlbaum, 1993:5-38.
- 4 Pinkerton SD, Wagner-Raphael U, Craun CA, *et al.* A quantitative study of the accuracy of college students' HIV risk estimates. *J Appl BioBehav Res* 2000;5:1-25.

A new method for extended trichomonad storage

With the introduction of the InPouch test for *Trichomonas vaginalis*,¹ *T. gallinae*, and *T. foetus*, it was desirable to have a procedure available for maintaining extended culture viability. The three trichomonads are viable after 8 days by subculture in the InPouch at 33°C. Extended viable storage of these three trichomonads is the subject of this letter.

We have evaluated various procedures involving freezing 24 hour InPouch cultures at -70°C. We now report a procedure that has demonstrated storage of viable trichomonad cultures for more than 2 years.

The freshly subcultured trichomonads are incubated at 35°C for 24 hours, which should produce a viable count of approximately 1.0×10^3 /ml. It is important to note that subsequent subculture will require an adequate nutrient available for growth in the pouch. Then 0.1 ml of pure sterile glycerol is added to the medium in the pouch and thoroughly mixed employing the "shoe-shine" technique. It is important to immediately place the pouch in a -70°C freezer.

After freezing most of the trichomonads in the pouch are non-viable, but successful subculture is routinely achieved upon thawing. When the pouch is removed from the freezer, it should be immediately placed in an incubator at 35-37°C. After 3 days a few viable trichomonads will be observed, and after 4 days it may be subcultured.

This procedure has been effective for *T. vaginalis*, *T. gallinae*, and *T. foetus*.

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Reference

- 1 Borchardt KA, Hernandez V, Miller S, et al. A clinical evaluation of trichomoniasis in San Jose, Costa Rica using the InPouch TV test. *Genitourin Med* 1992;68:328–30.

"Water can" penis caused by tuberculosis

Tuberculosis of the penis is a very rare condition, clinically manifesting as primary or secondary tuberculosis or tuberculide.¹ Penile involvement secondary to urethral tuberculosis is rare and its presentation with periurethral fistulas leading to "water can" penis is unknown. We report this rather intriguing condition in a patient.

A 40 year old male agricultural labourer presented with a 1 year history of purulent discharge per urethra with multiple discharging sinuses on the tip of the penis. The patient was asymptomatic about a year ago, when he developed multiple nodules on the glans penis that ulcerated to discharge purulent material. These nodules became persistent sinuses and discharged pus. Within a few weeks, he started passing urine through these sinuses in the glans penis. He also experienced difficulty in micturition but it was not associated with pain or strangury. The patient had no systemic complaints. He was married with two children and had no history of extramarital contact or genital ulcers.

On physical examination, the penis shape was like a saxophone. The prepuce and glans penis were oedematous and indurated. The glans penis had multiple sinuses around the urethral meatus (fig 1). On squeezing the penis, pus was expressed from the meatus and the sinuses. The glans penis also showed areas of depigmentation (vitiligo). The distal part of the shaft of the penis showed induration involving corpora cavernosa whereas the proximal part was devoid of any lesion. The testes, bilateral epididymis, and scrotum were normal. The vas deferens was normal on

Figure 2 Forearm showing positive Mantoux reaction.

palpation. The prostate was normal on rectal examination.

The routine haemogram revealed an elevated erythrocyte sedimentation rate of 100 mm in the first hour. His liver and renal functions were normal. The discharge smear stained with Gram stain and Zeihl-Neelsen stain. The Gram stained smear revealed numerous pus cells and acid fast stain showed abundant acid fast bacilli. Culture for *Mycobacterium tuberculosis* grew contaminants. A roentgenogram of the chest and penis was unremarkable. An intravenous pyelogram was normal. Voiding cystourethrography revealed glandular urethral stricture with urethrocutaneous fistulas. Ultrasonography of abdomen and prostate was normal. Mantoux skin test was strongly positive (30×30 mm) (fig 2). His venereal disease research laboratory test (VDRL) and HIV serology was non-reactive.

Based on these clinical features, positive Mantoux test and acid fast bacilli in the urethral smear, the diagnosis of urethral tuberculosis with urethrocutaneous fistula was made. The patient was started on anti-tuberculous treatment comprising isoniazid 300 mg, rifampicin 600 mg, pyrazinamide 1500 mg, and ethambutol 800 mg per day. The patient showed marked improvement after 4 weeks of treatment. The sinuses closed and discharge ceased. Patient was referred to urology for management of stricture, which was planned after the anti-tuberculous treatment. The patient tolerated antituberculous treatment and completed 9 months of treatment with remarkable recovery in the swelling of the penis.

Genital involvement occurs in 50% of male patients with urogenital tuberculosis. Penile tuberculosis is rare with less than 1% of patients having penile involvement.² Tuberculosis of the penis usually presents as ulcers, tubercular cavernositis, or nodules. In most cases, the lesion appears as a superficial, solitary, painless ulcer on the glans penis. It can be clinically indistinguishable from malignant disease.³ Rarely, lesions may persist as solid nodule or cavernositis with ulceration.^{4,5} Papulonecrotic tuberculide may also present as an ulcer on the penis.¹ Penile

involvement may occur secondary to co-existing urinary tract tuberculosis. The transmission occurs secondary to bacilluria in these patients. Infection of the penis may occur by direct contact at the time of intercourse with a partner having urogenital tuberculosis.²

Tuberculosis of male urethra is an uncommon condition and presents as urethral strictures, periurethral abscesses, or fistula formation. Fistulas can occur in the perineum leading on to "water can" perineum.⁶ Similar occurrence of fistulas in penis can aptly be designated as "water can" penis. In our case, penile involvement occurred secondary to urethral tuberculosis. Such involvement of the penis by tuberculosis is unique and not reported in the literature. "Water can perineum" is also known to occur with gonorrhoea but our patient had a negative urethral smear for Gram negative diplococci and had features suggestive of urethral tuberculosis. Further, the strictures, fistulas, and lymphoedema had led to "saxophone" deformity of the penis. Such deformity is well known with lymphogranuloma venereum, but is unknown in tuberculosis.

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References

- 1 Vijai Kumar M, Thappa DM, Kaviarasan PK. Papulonecrotic tuberculide of the glans penis (correspondence). *Sex Transm Infect* 2001;77:147.
- 2 Elkin M. Urogenital tuberculosis. In: Pollack HM, eds. *Clinical urography: an atlas and textbook of urological imaging*. Philadelphia: WB Saunders, 1990:1030–52.
- 3 Gow JG. Genitourinary tuberculosis. In: Walsh PC, Ritik AB, Stamey TA, et al, eds. *Campbell's urology*, Vol 1. 6th ed. Philadelphia: WB Saunders, 1992:951–81.
- 4 Venkataramaiah NR, van Raalte JA, Dutta SN. Tuberculous ulcer of penis. *Postgrad Med J* 1982;58:59–60.
- 5 Ramesh V, Vasanthi R. Tuberculous cavernositis of the penis. *Genitourin Med* 1989;65:58–9.
- 6 Symes JM, Blandy JP. Tuberculosis of male urethra. *Br J Urol* 1973;45:432–6.

South Asians with HIV in London: is it time to rethink sexual health service delivery to meet the needs of heterosexual ethnic minorities?

Recent conservative estimates suggest that at the end of 2002, 4.8 million people were living with HIV/AIDS in south Asia including 4.58 million in India.¹ In the United Kingdom there are estimated to be 1.5 million people of south Asian ethnicity. While the National Strategy for Sexual Health aims to improve health care in those who have HIV through earlier diagnosis,² studies have shown that that other ethnic minority groups present with advanced disease and not through routine genitourinary medicine (GUM) screening.^{3,4} We studied the case notes of all adults self defining as of Indian, Pakistani, Bangladeshi, or Sri Lankan ethnicity diagnosed HIV positive from

Figure 1 Saxophone penis with multiple sinus openings over the glans penis.

Table 1 Characteristics of presentation of study population at time of HIV diagnosis (n = 117)

	Heterosexual men (n = 45)	Homosexual men (n = 36)	Heterosexual women (n = 27)	Other risk groups (n = 9)
AIDS illness at presentation	16 (36%)	6 (16%)	2 (7%)	1 (11%)
Median CD4 cell count $\times 10^6/l$ (range)	178 (3–1,023)	381 (4–810)	377 (10–1,104)	151 (50–795)
Median HIV viral load copies/ml (range)	24 500 (50–1 000 000)	24 636 (425–3 000 000)	7822 (173–489 184)	12 870 (6676–57 530)
Reasons for HIV test				
AIDS/symptomatic	27 (60%)	11 (31%)	7 (26%)	2 (22%)
Known HIV+ sexual partner	3 (7%)	4 (11%)	12 (44%)	0
Routine screen for sexually transmitted infections	1 (2%)	16 (44%)	1 (4%)	0
Patient request	7 (15%)	3 (8%)	2 (8%)	2 (22%)
Child positive	3 (7%)	0	3 (11%)	0
Insurance/visa purposes	3 (7%)	1 (3%)	0	2 (22%)
Antenatal screening	0	0	2 (7%)	0
Other	1 (2%)	1 (3%)	0	3 (34%)

January 1985 to December 2002 attending four HIV treatment centres in London. Information was collected on demography, mode of first presentation, and clinical stage of HIV infection.

In all, 117 patients were identified, 30 women and 87 men. The number of new diagnoses among south Asians increased by more than threefold over the period 1996 to 2002 compared to earlier years (25 diagnoses before 1996, 90 diagnosed from 1996–2002).

The median age at diagnosis was 38 years (range 19–64 years) for men and 28 years (range 20–55 years) for women. Forty five patients (38%) had originated from Africa, 28 (24%) from India, and 18 (15%) from the United Kingdom. The majority were of Indian ethnicity (95/117; 81%) with the next largest ethnic group being Sri Lankan (12/117; 10%).

The primary mode of transmission was heterosexual sex (72/117; 62%) with transmission through sex between men accounting for a further 31% (36/117) of cases. Four infections were acquired through blood transfusion, two through injecting drug use, one from a needle stick injury, and in two cases risk behaviour could not be identified. The majority (39%, 45/117) of patients identified Africa as the probable place of infection with 28% and 15% probably infected in the United Kingdom and India, respectively.

There were substantial differences in the reasons for testing between individuals in the main risk groups. In particular, heterosexual men and women were both significantly less likely than homosexual men to be diagnosed via routine attendance at a GUM clinic (2% and 4%, compared to 44%, respectively, $p < 0.001$, Fisher's exact test). Among heterosexuals, the main reason for testing in men was symptomatic HIV infection/AIDS (60% of men but only 26% of women), whereas women were more likely to be tested through partner notification of a known HIV+ sexual contact (44% v 7% in males) (table 1).

The median CD4 count at presentation overall was 300 (range 3–1104) cells $\times 10^6/l$. However, male heterosexuals presented with significantly lower CD4 counts (median 178,

range 3–1023 cells $\times 10^6/l$) than either homosexual men (median 381, range 4–810 cells $\times 10^6/l$; $p = 0.01$) or heterosexual women (median 377, range 10–1104; $p = 0.02$).

While there are methodological limitations with retrospective case note reviews and differing reporting categories used for Asian ethnicity, our data confirm national surveillance reports of increasing HIV infection among Britain's south Asian communities.⁵ The four centres taking part in this study reported 90 cases from 1996–2002 representing one in three of all HIV positive south Asians reported in this time period. Despite the fact that the majority of these were not diagnosed through routine GUM screening the median CD4 count at presentation of heterosexual and homosexual men was consistent with national trends.⁶ Indeed, south Asian women presented higher CD4 counts than seen nationally, primarily attributable to effective partner notification. While south Asians still represent less than 5% of all reported HIV positive diagnoses in UK ethnic minority groups⁵ (Asians 334; black Africans 8848; black Caribbeans 844) numbers are likely to continue to increase in the future and methods for encouraging early presentation need to be developed in response to this.

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References

- 1 UNAIDS. AIDS epidemic update, 2002.
- 2 Department of Health. *National strategy for sexual health and HIV*. London: DoH, 2001.
- 3 Burns FM, Fakoya AO, Copas AJ, et al. Africans in London continue to present with advanced HIV disease in the era of highly active antiretroviral therapy. *AIDS* 2001;15:2453–5.
- 4 Del Amo J, Petrukevitch A, Phillips AN, et al. Spectrum of disease in Africans with AIDS in London. *AIDS* 1996;10:1563–9.
- 5 Health Protection Agency, HIV/STI Division Communicable Disease Surveillance Centre and for the Scottish Centre for Infection and Environmental Health. HIV/AIDS Quarterly Surveillance Tables Cumulative UK data. CDSC, June 2003.
- 6 Health Protection Agency, HIV/STI Division Communicable Disease Surveillance Centre. *HIV/AIDS in the UK an epidemiological review*. London, 2000.

Failure to maintain patient access to GUM clinics

We read with interest the article published by Cassell *et al*¹ about the maintenance of patient access to genitourinary medicine (GUM) clinics following a switch to an appointment based system. Their data show no significant change in the age, ethnic mix, symptom status, and disease mix following the change to appointments. In addition, such a system of 35% prebooked appointments produced an increase in the number of patients seen over that time.

A new appointment based system was introduced at the John Hunter genitourinary medicine clinic at the Chelsea and Westminster Hospital in October 2001. This comprised 80% of appointments which were prebooked with a further 20% allocated on the day following triage by a nurse. All patients with symptoms were seen on the day of presentation.

We have analysed the results from two 9 month periods, taken immediately before the change and 3 months after the introduction of an appointment based system. The total number of patients and sex ratio seen

Table 1 Total number of STI diagnoses

	No (%)		Relative drop (%) (95% CI)
	Jan–Sept 2001	Jan–Sept 2002	
Total no of patients attending	11714	11345	3.2 (2.8 to 3.5)
Patients new to clinic	5191 (44.3)	4669 (41.2)	

Table 2 Details of STIs diagnosed in men and women

	Male			Female		
	Jan–Sept 2001 (n = 6920)	Jan–Sept 2002 (n = 6659)		Jan–Sept 2001 (n = 4794)	Jan–Sept 2002 (n = 4690)	
	No (prevalence per 100 patient)		p Value using χ^2 test	No (prevalence per 100 patient)		p Value using χ^2 test
A1	37 (0.5)	53 (0.8)	0.061	0 (0.0)	3 (0.1)	0.121*
B1	262 (3.8)	190 (2.9)	0.002	41 (0.9)	35 (0.7)	0.552
C4a/C4c	244 (3.5)	179 (2.7)	0.005	187 (3.9)	199 (4.2)	0.399
C4h	683 (9.9)	479 (7.2)	<0.001	–	–	–
C10a	89 (1.3)	55 (0.8)	0.009	111 (2.3)	80 (1.7)	0.035
C11a	264 (3.8)	254 (3.8)	0.998	147 (3.1)	164 (3.5)	0.239
Total diagnosed with an STI at this episode	1579 (22.8)	1210 (18.2)	<0.001	486 (10.1)	481 (10.3)	0.849

*p Value using χ^2 test with Yates's correction.

(A1) Primary diagnosis of syphilis; (B1) gonorrhoea; (C4a, C4c) uncomplicated chlamydia; (C4h) non-gonococcal urethritis; (C10a) first attack of genital herpes; (C11a) anogenital warts.

over this period did not change. We have shown however a dramatic change in the number of STI diagnoses made over these two periods.

Tables 1 and 2 highlight a significant fall in the total number of STI diagnoses for gonorrhoea (B1), uncomplicated chlamydia (C4a, C4c), non-gonococcal urethritis (C4h), and first attack of genital herpes (C10a) in our male patients. The only significant fall for women was seen in the diagnosis of a first attack of genital herpes. There was no significant change for both sexes in the diagnosis of anogenital warts (C11a) between the two systems. The rise in primary diagnosis of syphilis (A1) reflects the beginning of the current epidemic in London, boosted further by a proactive approach to diagnosis in our HIV positive population.²

This fall in acute STI diagnoses in men was approximately twice as marked for men who have sex with men (data not shown).

Our aim in planning the change to a primarily appointment based system was to improve patient experience, by reducing waiting times, and enhance access for symptomatic patients into reserved appointment slots. These data show evidence for an opposite effect which we believe has resulted from asymptomatic individuals requiring sexual health screening booking the majority of clinic appointments well ahead of their appointment, thereby reducing access at convenient times for symptomatic individuals who telephone.

To respond to this we have adjusted the ratio of prebooked versus emergency appointments and significantly amended our approach to triage of symptomatic patients, in an attempt to reverse these trends. Particular attention is now being given to our telephone booking protocol to facilitate symptomatic patients to achieve prompt, immediate appointments. We are publishing these findings to inform others who are implementing changes in clinic appointment schedules, designed to enhance access, to better tailor the booking and triage systems to achieve this goal. We will continue to audit our system to examine the effect of the revised system and to further examine why the change to our appointment system disproportionately affected those men who have sex with men.

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References

- 1 Cassell JA, Brook MG, Mercer CH, *et al.* Maintaining patient access to GUM clinics: is it compatible with appointments? *Sex Transm Infect* 2003;**79**:11–15.
- 2 Winston A, Hawkins DA, Mandalia S, *et al.* Is increased surveillance for asymptomatic syphilis in an HIV outpatient department worthwhile? *Sex Transm Infect* 2003;**79**:257–9.

Prevalence of HSV-1/HSV-2 antibodies in HIV seropositive patients in Coventry, United Kingdom

The seroprevalence of herpes simplex virus (HSV) antibody among HIV patients within the United Kingdom is unknown. We therefore conducted a HSV seroprevalence study in HIV patients attending our genitourinary medicine clinic from January 2000 to December 2001. Our previous study¹ revealed an overall prevalence of HSV-1 (60%), HSV-2 (20%), and both HSV-1 and HSV-2 (12%) among male and female genitourinary medicine clinic attendees who were either HIV negative or whose HIV status was unknown.

Serum samples from 96 consecutive ethnically diverse HIV patients were collected during routine investigations, and tested for HSV type specific antibodies by monoclonal antibody blocking enzyme linked immunoassay.² Out of 96 patients, two HSV-1 and three HSV-2 antibody test results were equivocal in four individuals. These were excluded from the analysis and results are presented here for 92 patients.

There were 56 men and 36 women in the study: 46 (50%) were white, 43 (47%) black African, and three were from other ethnic groups. All the black Africans were heterosexuals and 71% of men were homosexuals. The median age was 35 years (range 21–80).

HSV-1 seroprevalence was 86% among men and 97% among women ($p = 0.14$). HSV-2 seroprevalence was 50% among men whereas it was 94% among women ($p = 0.0001$). There was no statistically significant difference between the seroprevalence of HSV-1 between white and black

people. However, seroprevalence of HSV-2 and both serotypes was significantly higher among black than among white people.

This study shows very high seroprevalence of HSV-1 (90%), HSV-2 (67%), and both HSV-1 and HSV-2 (64%) among our HIV positive cohort in Coventry. The high prevalence of HSV-2 in women is possibly because most of them were black African and acquired HIV through sex. These findings may have important public health implications as the high rate of HSV-2 is therefore likely to act as a cofactor in HIV transmission.

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References

- 1 Narouz N, Allan PS, Wade AAH, *et al.* Genital herpes serotyping: a study of the epidemiology and patients knowledge and attitude among STD clinic attenders in Coventry, UK. *Sex Transm Infect* 2003;**79**:35–41.
- 2 Van Doornum GJJ, Slomka MJ, Buimer M, *et al.* Comparison of a monoclonal antibody-blocking enzyme-linked immunoassay and a strip immunoblot assay for identifying type-specific herpes simplex type 2 serological responses. *Clini Diagn Lab Immunol* 2000;**7**(4):641–4.

BOOK REVIEW

Effective Sexual Health Interventions: Issues In Experimental Evaluation

Ed Judith M Stephenson, John Imrie, and Chris Bonell. Pp 232; £55. Oxford: Oxford University Press, 2003. ISBN 0-19-850849-2.

HIV spreads more every day and there are epidemics of other STIs in both the developed and developing world at least in part because the fear of HIV appears to be receding in the population. Our current strategies to contain these problems are meeting with limited success and treatment of people who are already infected, important though that is in controlling bacterial infections, is much less effective with continuing viral infections. There is an urgent need to develop and to test better

methods of helping people to reduce their risky sexual behaviour.

This book is excellent, brief, fairly comprehensive, and very readable. Its focus is designing studies on the effectiveness of sexual health interventions. If we are to get anywhere in improving behavioural interventions it is essential that what is done is carefully evaluated.

The first three chapters of the book are concerned with methodology, particularly whether randomised controlled (RCTs) trials are an appropriate method for evaluating interventions in this area. While this section of the book is well argued on all sides it doesn't really break any new ground. The strengths and weaknesses of RCTs in behaviour change are pretty much what they are in any other area of medicine. Methodologies don't exist as stand alone phenomena, whether an RCT or some other methodology is appropriate depends simply on what question one is seeking to answer.

The second section of the book covers models of behaviour change and the choice of design and outcome measures. It is clear that one of the main problems in intervening in sexual health is the poor quality of the available psychological models and our real lack of understanding about why people behave as they do. Without understanding why people behave as they do it is difficult to help them to change. It is interesting that models of health behaviour never seem to get discarded, even the ones that are known to be weak. There are particularly strong chapters on cluster randomisation, an approach which probably gives rise to more inappropriate statistics than any other and on complex behavioural measures. The latter should be required reading for anyone measuring any aspect of risky sexual behaviour simply because it highlights how weak many studies of sexual behaviour—and not just of behaviour change—are in this respect.

The book ends by looking at generalisability in its broadest sense. Generalisability is an area that tends to get overlooked. Even a highly successful behaviour change programme would be of no use in developing countries if it was labour intensive and dependent on highly skilled staff for its delivery.

I would recommend this book to anyone planning a trial or simply seeking to understand the existing literature. I would however caution that to make sense of it you will have to look at some of the available reviews of the behaviour change literature since the book assumes some knowledge, or willingness to acquire knowledge, of these.

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B-1740 Ternat, Belgium (tel: +32 2 582 08 52; fax: +32 2 582 55 15; email: orgamed.ann@pandora.be; and website: <http://www.contraception-esc.com/edinburg.htm>).

CORRECTIONS

In the October issue of *STI* table 2 of the paper by Zheng *et al* (Zheng HP, Cao WL, Wu XZ, Yang LG. Antimicrobial susceptibility of *Neisseria gonorrhoeae* strains isolated in Guangzhou, China, 1996–2001. *Sex Transm Infect* 2003;**79**:399–402) was published with incorrect column headings. Under the heading spectinomycin only “S(%)” and “R(%)” should appear and under ceftriaxone “S(%)”, “I(%)”, and “R(%)” should appear, in that order. Under ciprofloxacin “S(%)”, “I(%)”, and “R(%)” should appear. A corrected version of the table can be found on the website at <http://sti.bmjjournals.com/cgi/content/full/79/5/399/DC1>.

The authors of a letter in the December issue of *STI* (Dave SS, Johnson AM, Fenton KA, Mercer CH, Erens B, Wellings K. Male circumcision in Britain: findings from a national probability sample survey. *Sex Transm Infect* 2003;**79**:499–500) were listed in the wrong order. The correct author list should be as follows: Dave SS, Fenton KA, Mercer CH, Erens B, Wellings K, Johnson AM.

In the corresponding author's address of a letter published in the December issue (Bhatia R, Prabhakar S, Shedde D, *et al*. Coexistent cranial tuberculomas and tuberculosis of the cervix in a postmenopausal woman. *Sex Transm Infect* 2003;**79**:496–7) All India Institute of Medical Sciences was incorrectly printed as AU India Institute of Medical Sciences.

NOTICES

Australasian Sexual Health Conference 2004: Behind the Mask

This conference will be held at the Adelaide Convention Centre, South Australia, on 31 March to 3 April 2004. For further details please contact Dart Associates (tel +61 2 9418 9396/97; email dartconv@mpx.com.au; and website <http://www.acshp.org.au>).

8th European Society of Contraception Congress

The 8th European Society of Contraception Congress will be held from 23–26 June 2004 in Edinburgh, Scotland, UK. For further details please contact ESC Central Office, c/o Orga-Med Congress Office, Essenestraat 77,