Sexually Transmitted Infections

Editorials

HIV prevention and homosexual men: should we be optimistic about the new millennium?

In the United Kingdom, HIV prevention among homosexual men is recognised as a long term and evolving challenge.[1] For more than a decade, sustained and innovative prevention efforts have formed part of the statutory and community response to the national AIDS epidemic. The adoption of safer sex and health protective behaviours (for example, completed hepatitis B vaccination, routine STI screening including HIV tests) are testimony to their relative success.[2 3] More recently, however, the availability of effective antiretroviral therapies, dramatic reductions in reported AIDS cases and deaths, and the apparent stabilisation of the HIV epidemic have resulted in a gradual relaxation of the crisis response to AIDS. This relaxation is also apparent within some affected communities[4] and among the people working with them,[5] while the exclusion of HIV/AIDS from the government's white paper for England, *Saving Lives: Our Healthier Nation*, also reflects changing national priorities.[6] In many circles, a new optimism prevails—the worst seems to be over.

However, this optimism may be unfounded if it is based solely on removal of the threat of death, rather than a demonstrated reduction in HIV incidence and sustained behavioural change. Each year about 1500 homosexual men are newly diagnosed as HIV positive, a figure that has remained relatively unchanged for a decade.[7] Behavioural surveillance of homosexual men in London shows gradual and significant increases in the proportion of men reporting unprotected anal intercourse (UAI) with a partner of unknown or discordant HIV status.[2] Other national studies show a stabilisation of UAI rates across the country,[8] and rates of gonorrhoea among homosexual men, particularly those aged 35 and over, appear to be increasing.[9] So, has HIV prevention among homosexual men reached an impasse? Where should HIV prevention be heading in the future?

HIV prevention—how far have we come?

HIV prevention among homosexual men has progressed through several stages, reflecting developments in our understanding of the natural history of infection, disease epidemiology, diagnostic and therapeutic advances. Early prevention messages reflected our limited knowledge of disease transmission. Increased understanding of the protective role of condoms led to their promotion for all acts of anal intercourse (100% condom use), a strategy that failed to fully incorporate evidence on their efficacy or homosexual men's concerns about their acceptability and appropriateness.[10] The widespread availability of HIV testing enabled its incorporation into primary prevention strategies and the promotion of risk reduction strategies such as "negotiated safety."[11] Today, the range of HIV prevention interventions utilised with homosexual men is varied, and includes conventional health education, outreach, one to one counselling, group work, peer led education, and community development. Theoretically derived behavioural interventions, targeting individuals perceived to be at increased risk (for example, men with acute STDs or reporting UAI) are increasingly delivered in a wide range of settings, including those where risky behaviours are likely to occur.[12]

However, at the end of the 1990s, HIV prevention in homosexual men is being forced to confront new realities. Changes in disease epidemiology, public and sexual health priorities,[6] "prevention fatigue," and increased budgetary pressures make general approaches to HIV prevention less tenable, and force us to reconsider whether targeting those who are unlikely to be at "increased risk" is appropriate and cost effective. We believe what is required is a refocusing of our efforts, and that four key areas offer new opportunities for intervention, and potentially important prevention dividends.

Younger homosexual men—a different generation with different need

It would be incorrect to believe that safer sex strategies are passed from one generation of homosexual men to the next, or that young homosexual men whose sexual careers began after the emergence of the epidemic have had the same experience of AIDS as older homosexual men.[4 13 14] Men under 30 years constitute almost one third of newly diagnosed HIV infections in homosexual men each year.[7] Gonorrhoea diagnoses increased by 17% in homosexual men aged 16–19 years between 1995 and 1998.[15] Behavioural surveys show that young homosexual men are more likely to report UAI than older men, particularly so with unknown or serodiscordant partners.[2 16] With high levels of knowledge and familiarity with safer sex,[8 16] many do not see HIV as a concern for themselves, but one for older homosexual men.[14] This generational effect may worsen as the crisis response to AIDS diminishes and changes in men's understanding of their "gay identity" threaten the cohesiveness of the gay community.[10]

The challenges for HIV prevention among young homosexual men are many. As with all groups of homosexual men, but particularly younger men, efforts to control STDs (which facilitate the transmission and acquisition of HIV) must be prioritised. Messages that focus on single behaviours (for example, condom use) for a single objective (preventing HIV infection) are no longer appropriate. Messages need to be flexible, engage young men in a

variety of ways, and adapt to changing attitudes and behaviours.[14 17] We must also recognise that legal obstacles (for example, the age of consent and Section 28)* prevent a frank discussion of sexual diversity at an early age and create environments where discrimination, homophobia, and poor self esteem are allowed to flourish. Interventions specifically tailored to young homosexual men's perceived needs, and appropriate strategies (for example, peer education and community based development projects) have been successful in reducing UAI rates over time.[17] The long term dividends of successful HIV prevention with this group are worth the additional efforts required and fit neatly with the government's objective of "increasing the length of people's lives and the number of years people spend free from illness."[6]

Working with HIV positive homosexual men

Targeting prevention interventions at HIV positive individuals is difficult and it is no surprise that they have been, and remain, relatively unengaged in prevention planning. Affected communities are understandably concerned about stigmatisation and discrimination; prevention workers feel ill equipped to tackle pertinent issues; and clinicians often fail or are reluctant to incorporate prevention discussions into the clinical context—a missed opportunity. However, as the stigma and exceptionalism associated with HIV diminishes, an opportunity exists to re-evaluate individual and collective responsibilities for preventing onward transmission. People living with HIV have indicated that issues directly related to primary prevention—partner notification, disclosure of HIV serostatus, managing relationships—are part of living with the disease.[18] Clinicians are increasingly aware that widespread antiretroviral prescribing carries a responsibility for ensuring that the risk of transmission of resistant or virulent strains is minimised. Targeting primary prevention interventions within routine HIV clinical care may allow for more tailored and cost effective approaches that are better suited to individual requirements. However, this will require that consideration is given to the skills mix and the resources needed to support such programmes. More generally, those committed to delivering accessible and appropriate prevention interventions for people living with HIV must also be committed to establishing genuinely productive partnerships.

Understanding and managing risk

Recent prevention work has attempted to support strategies for reducing the risk of sexual transmission of HIV based on knowledge of HIV status, partner seroconcordancy, and the ability to negotiate contexts where UAI may occur (for example, negotiated safety).[11 19] However, communicating issues around risk and risk management can be difficult, and promoting risk reduction strategies may conflict with other prevention messages that focus on increasing condom use.[14] Additionally, as risk reduction activities involve undertaking multiple and often complex tasks, men employing them may do so less than perfectly.[20 21] But this does not mean that we should abandon risk reduction. On the contrary, since we know UAI is occurring, the challenge is finding ways to support homosexual men in understanding and minimising their risks as much as possible. Not all UAI is "high risk." In many instances, UAI may be relatively low risk depending on the partner, context, and local epidemiology. Prevention messages that promote risk reduction should aim to provide the right information so that when UAI does happen, it is more likely to happen in contexts that are "lower risk," rather than only in very restricted setting(s) where there is virtually no risk. This may provide a more pragmatic approach to dealing with the realities of safer sex "fatigue" and "lapses" in safer sex behaviours currently being observed.[2] However, we must also be mindful that little is actually known about the effectiveness of risk reduction strategies at the population level, or how they compare with other prevention strategies.[20 21] In addition to developing and delivering these interventions, concomitant evaluation will be required.

Improving evaluation of interventions

Despite advances in our understanding of behavioural theory and prevention models, the success of many interventions continues to be measured in numbers of condoms distributed, self reported behaviour change, and rates of UAI. More sophisticated tools to measure the effectiveness of innovative, theory based interventions—use of biological markers (HIV seroconversion rates or STD acquisition) and wider sexual health outcomes (for example, psychosocial wellbeing)—have received only limited consideration. There are only a few examples of evaluations in the United Kingdom in which disease outcomes or experimental methodologies have been employed to measure the effectiveness of prevention interventions.[22–24] Even carefully designed behavioural interventions should not be assumed to bring benefit; they need to be evaluated to prove their effectiveness.

Good evaluations do not need to be expensive or labour intensive if they are included at the intervention's design stage. But outcome measures (biological or behavioural) must be appropriate. If an intervention is designed to reduce disease incidence by reducing risky behaviours, then an objective measure of disease incidence must be considered as the most powerful indicator of its effectiveness.[25] Well conducted, rigorous evaluations are the only way to demonstrate efficient use of increasingly limited resources. Although prevention workers may not feel they have the skills or resources to undertake them routinely, much can be gained by creating alliances between academic units and service providers,[26] utilising and disseminating models of good practice.

Conclusions

The dramatic prevention achievements of the 1980s, largely attributable to the response of a galvanised gay community, are clearly a thing of the past. In sexual health, a growing political and specialty interest in other areas (for example, chlamydia and teenage pregnancy) suggests that HIV prevention could soon be relegated to the back seat. It could easily be argued that HIV prevention in homosexual men has reached the stage where ever increasing resources and skills are required to achieve ever smaller dividends. Nevertheless, dividends are still there, and in the case of young homosexual men, they may be among our most important prevention achievements. We believe that by focusing our energies in the areas discussed we can maximise the remaining potential benefits of HIV prevention in homosexual men. Our success will depend to some extent on the presence and strength of supportive infrastructures among those involved in HIV prevention, treatment, and care. These include establishing creative and enduring partnerships between sexual health providers, community based organisations, and academic institutions; critically reappraising the HIV clinician's role in facilitating primary prevention; ensuring that prevention workers keep abreast of, adapt, and incorporate evidence based prevention strategies into planning; and finally, adopting a more holistic approach to sexual health in which the wider determinants of sexual health are tackled.[4]

We have been warned already about becoming complacent with respect to HIV prevention and homosexual

men.[13] If there is reason to be optimistic, it is because the immediate and long term challenges of HIV prevention in homosexual men are better understood, as are some of the tools with which to tackle them.

* Different ages of consent apply for heterosexual sex (16 years) and homosexual sex involving two males (18 years). Lesbian sex is not explicitly mentioned in the law; however, a female under 16 is deemed not capable of consenting to any sexual act. Section 28 of the 1988 Local Government Act states: 2A (1) A local authority shall not (a) intentionally promote homosexuality or publish material with the intention of promoting homosexuality; (b) promote the teaching in any maintained school of the acceptability of homosexuality as a pretend family relationship.

Funding: none.
Conflict of interest: none

JOHN IMRIE
Department of Sexually Transmitted Diseases, Royal Free and University College Medical School, Mortimer Market Centre, off Capper Street, London WC1E 6AU

KEVIN A FENTON
Department of Sexually Transmitted Diseases, Royal Free and University College Medical School, Mortimer Market Centre, off Capper Street, London WC1E 6AU and Public Health Laboratory Service, Communicable Disease Surveillance Centre, Colindale, London NW9 5EQ

ANDREW BILLINGTON
Body Positive, 14 Greek Street, London W1V 5LE

1 HIV/AIDS Health Promotion Strategy Group. *HIV and AIDS prevention: an evolving strategy.* London: UK Departments of Health, 1995.
2 Dodds J, Mercey D, Nardone A. *Monitoring high risk sexual behaviour amongst gay men in London—1998.* London: Department of Sexually Transmitted Diseases, Royal Free and University College Medical School, 1999.
3 Hart GJ, Flowers P, Der GJ, *et al.* Homosexual men's HIV related sexual risk behaviour in Scotland. *Sex Transm Inf* 1999;**75**:242–6.
4 Rofes E. *Dry bones breathe—gay men creating post-AIDS identities and cultures.* Binghamton, NY: Harrington Park Press, 1998.
5 Lowth A, Yallop S, Reid J, *et al.* HIV health care services: are we responding to the changing needs of PLWHA? (Oral presentation.) 4th International Conference on the Biopsychosocial Aspects of HIV Infection (AIDS Impact), Ottawa, 15–18 July 1999.
6 Department of Health. *Saving lives: our healthier nation.* London: Stationery Office, 1999.
7 PHLS AIDS and STD Centre, Communicable Disease Surveillance Centre and Scottish Centre for Infection and Environmental Health. Unpublished quarterly surveillance tables, No 44:99/2, table 20, June 1999.
8 Hickson F, Reid D, Weatherburn P, *et al. Making data count—findings from the National Gay Men's Sex Survey 1997.* London: Sigma Research and Terrence Higgins Trust, 1998.
9 Sexually Transmitted Disease Quarterly Report. Gonorrhoea in England and Wales. *Commun Dis Rev CDR Wkly* 1999;**9**:270–2.
10 Rotello G. *Sexual ecology—AIDS and the destiny of gay men.* Harmondsworth, Mddx: Penguin, 1998.
11 Billington A, Hickson F, Maguire M. *Thinking it through—a new approach to sex, relationships and HIV for gay men.* 2nd ed. London: Camden and Islington Community Health Services NHS Trust, 1997.
12 Gay Men Fighting AIDS, Health First, RS Health, Terrence Higgins Trust. *The six provider initiative—programme of gay men's HIV prevention work in London 1999/2000.* London: Camden and Islington Health Promotion Service, 1998.
13 Stall R. How to lose the fight against AIDS among gay men. *BMJ* 1994;**309**: 685–6.
14 Billington A, Imrie J, Chopin M, *et al.* Young gay and bisexual men and HIV testing in the time of combination therapy: any new barriers to testing? (abstract). 4th International Conference on the Biopsychosocial Aspects of HIV Infection (AIDS Impact), Ottawa, 15–18 July 1999.
15 Lamagni TL, Hughes G, Rogers PA, *et al.* New cases seen at genitourinary clinics: England 1998. *Commun Dis Rep CDR (Suppl)* 1999 (in press).
16 Fenton KA, White B, Weatherburn P, *et al. What are you like? Assessing the sexual health needs of black gay and bisexual men.* London: Big Up, 1999.
17 Kegeles SM, Hays RB, Pollack LM, *et al.* Mobilizing young gay and bisexual men for HIV prevention: a two community study. *AIDS* 1999;**13**:1753–62.
18 Kramer I. New treatments: a PWA perspective. (Oral presentation.) 4th International Conference on the Biopsychosocial Aspects of HIV Infection (AIDS Impact), Ottawa, 15–18 July 1999.
19 Kippax S, Crawford J, Davis M, *et al.* Sustaining safe sex: a longitudinal study of a sample of homosexual men. *AIDS* 1993;**7**:257–63.
20 Elford J, Bolding G, Maguire M, *et al.* Sexual risk behaviour among gay men in a relationship. *AIDS* 1999;**13**:1407–11.
21 Kippax S, Noble J, Prestage G, *et al.* Sexual negotiation in the AIDS era: negotiated safety revisited. *AIDS* 1997;**11**:191–7.
22 James NJ, Gillies PA, Bignell CJ. Evaluation of a randomised controlled trial of HIV and sexually transmitted disease prevention in a genitourinary medicine clinic setting. *AIDS* 1998;**12**:1235–42.
23 Imrie J, Stephenson JM, Cowan FM, *et al.* A randomised controlled trial of brief small-group behavioural intervention workshop for 'high risk' gay men using an STD clinic. (abstract No 003), 13th Meeting of the International Society for Sexually Transmitted Diseases Research (ISSTDR), Denver, 11–14 July 1999.
24 Elford J, Bolding G, Maguire M, *et al.* Does peer-led HIV prevention work among gay men in London? Findings from a controlled trial (abstract). 4th International Conference on the Biopsychosocial Aspects of HIV Infection (AIDS Impact), Ottawa, 15–18 July 1999.
25 Aral SO, Peterman TA. Do we know the effectiveness of behavioural interventions? *Lancet* 1998;**351**(SIII):33–5.
26 Somlai A, Kelly JA, Hackl KL, *et al.* Improving HIV/AIDS prevention interventions through program evaluation at the community level (abstract No 44302) conference record. 12th World AIDS Conference, Geneva, 28 June–3 July 1998.

Are STIs underreported in rural Australia?

Bowden *et al* (p 431) using specimens collected by tampon and polymerase chain reaction (PCR) technology from indigenous women in the Northern Territory of Australia, have shown that the prevalence of *Trichomonas vaginalis*, *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and human papillomavirus infection are very high in this group of women, and that the prevalence of *N gonorrhoeae* and *C trachomatis* was more than four times the official notification rate from the Northern Territory Health Service.

The interpretation of these data is complicated by several factors. Firstly, the authors provide limited information on the attendees and how many accepted or declined the screening. The second issue relates to the fact that, of women with symptoms, the proportion of these who presented with symptoms, or whose symptoms were elicited on direct questioning, is unclear. However, it would still appear that less than 10% of patients presented with symptoms and that the majority of patients notified to the Northern Territory Health Service would only have been tested as a consequence of genital symptoms. Thirdly, as mentioned by the authors, the PCR methodology is more sensitive than existing techniques of culture and microscopy, and consequently will detect more patients. Finally, community based prevalence studies will always detect more patients that those notified through routine reporting systems.

Despite these reservations, the disparity between the proportion of patients detected in this study and those found through routine surveillance systems is enormous and is a great cause of public health concern. This potential for underreporting STIs which are endemic in this part of Australia has serious repercussions for service funding provision. This in turn will affect morbidity and mortality as it will lead to an underestimation of the population infected and at risk.

Currently, each of the states and territories in Australia is responsible for surveillance. This usually occurs through a process of individual case notification by clinicians and/or laboratories. All states and territories notify cases of syphilis and gonorrhoea, and chlamydia has recently been added to the list. Genital herpes, human papillomavirus infection, and trichomoniasis are not notified. Consequently it is difficult to determine the true incidence and prevalence of STIs in Australia.

There are several possible strategies for improving the situation. This first is to consider abandoning the current state based surveillance system and, instead, instituting a

national surveillance system whereby data from across the country are collected and coordinated. This would enable a national sexual health strategy to be developed and evaluated. The second initiative is to improve the network of STI (sexual health) clinical services throughout Australia. Most urban areas are reasonably well serviced, the same is not true in many rural and remote communities. Some of this could be provided by improved training for general practitioners, whereas in other communities the possibility of using sexual health nurse practitioners should be explored.

Experience from other countries suggests that the fundamental principle of a sexual health service should be that it is free at the point of access, that it is confidential, that the service is provided in a non-confrontational non-judgmental fashion, and that wherever possible, a choice of services and/or providers should be available. Involvement of local communities in service provision is essential. Until such initiatives are set in place, it is likely that STIs will continue to be a problem in some rural communities in Australia.

ADRIAN MINDEL
Department of Sexual Health, Sydney Hospital, Sydney, NSW 2001, Australia

MELINDA TENANT-FLOWERS
Department of Genitourinary Medicine, King's College Hospital, London SE5 9RS

Why is *Trichomonas vaginalis* ignored?

> "In discussing trichomoniasis, it seems advisable . . . that the term 'venereal disease' should be avoided . . . Although the infestation is acquired by sexual intercourse in most cases, it is not 'VD' . . ." (Editorial, *British Journal of Venereal Disease* 1960[1])

If editorials have any power to influence the way a profession behaves then the confusion and neglect that currently surrounds *Trichomonas vaginalis* infection may have been seeded nearly 40 years ago. While delegates fill the lecture theatres at conferences where new diagnostics for *Chlamydia trachomatis* are being showcased, the concurrent session on *T vaginalis* laboratory techniques attracts only a handful of scientists and clinicians. Despite the rapid advances in nucleic acid amplification assays (for example, polymerase chain reaction (PCR), ligase chain reaction (LCR)), there is still no commercial kit available for the diagnosis of *T vaginalis*. This relative lack of scientific and medical interest has left public health policy makers "underwhelmed" by calls for the introduction of trichomoniasis control programmes.

Why is it so? Common reasons cited include the fact that *T vaginalis* is uncommon and that infection is associated with minimal morbidity and sequelae. Many clinicians and microbiologists who work in urban practices state that they see the disease only occasionally and these opinions are extrapolated to the wider population. But is this the true state of affairs?

The success and failure of metronidazole: is trichomoniasis a common disease?

In his 1947 monograph of *T vaginalis* infection Trussel estimated that between 20% and 25% of the female population of the United States were infected with the organism.[2] Although many considered this to be an overestimate, a number of contemporary surveys had confirmed that the disease was common. A study of women attending the obstetrics and gynaecology clinic at a US naval hospital in 1938 showed that 24.6% were infected,[3] and 14% of male inductees into the US military in 1943 were shown to be infected.[4] Samples collected in the course of a US vaginal cytology survey in the 1950s showed infection in 60.9% of black women and 8.1% of white women,[5] while examination of the Papanicolaou smears of 38 000 "healthy workers" in the United States in the 1960s demonstrated infection in 30.4% of black women and 10.7% of white women.[6]

The picture greatly improved after the introduction of metronidazole for treatment of *T vaginalis* in 1959. In the first placebo control trial of metronidazole, microbiological and clinical cure occurred in only 6.7% of women on placebo but in 89.9% of women who received active drug.[7] Nevertheless, the disparity of risk between ethnic groups continued into the post-antibiotic age. By 1979 infection was detected in only 0.5% of antenatal clinic attenders in an urban Australian setting,[8] in 1.9% of European men with urethritis by the 1990s,[9 10] but 22.8% of black women attending an antenatal clinic in the United States were infected, compared with 6.1% of white women.[11]

T vaginalis has been detected in 24.7–49% of women in Africa,[12 13] in 25% of indigenous women in northern Australia[14] and in around 45% of Melanesian women and 10.9% of Melanesian men in Papua New Guinea.[15]

Comparison of prevalence data is problematic owing to the significant biases present in all the studies. Although the diagnosis of *T vaginalis* is not technically difficult and, apart from PCR, is essentially unchanged since the 1940s, it is dependent upon the collection of suitable clinical specimens, the use of appropriate culture media, and on the experience and diligence of the microscopist. Nevertheless, the above data unequivocally show that *T vaginalis* is a common sexually transmitted disease, especially in poor women in both the developing and developed world.

Are the clinical manifestations of *T vaginalis* infection trivial?

The bulk of the pre-antibiotic literature on *T vaginalis* is anecdotal and at times idiosyncratic (witness McCullagh's contention that almost all *T vaginalis* transmission occurred from toilet seats and that the introduction of the "gap seat" in public facilities would control the infection in the United Kingdom[16]) but most authors are agreed that trichomoniasis was a troubling and, at times, distressing condition. The absence of simple, safe, and effective treatment meant that infected individuals were subjected to prolonged and often unpleasant treatment courses; their willingness to undergo this is suggestive of the degree of discomfort associated with the infection.

In women who present with symptoms, the major features are vaginal discharge (42%), abnormal vaginal odour (50%), and vulval itching (60%). The signs include vulval erythema, purulent discharge, and colpitis macularis (or "strawberry cervix"). The "green and frothy" discharge so often considered pathognomonic of the disease probably

occurs infrequently. In men the major manifestations are discharge, dysuria and, rarely, penile ulceration. The symptoms are usually mild but prostatitis may occur.[17]

In four age structured surveys (Bowden F, Garnett G. Trichomonas vaginalis: epidemiology and treatment interventions for an HIV cofactor STD, submitted for publication)[5 6 15] the prevalence of *T vaginalis* increases with age, a phenomenon that is not seen with *Neisseria gonorrhoeae* and *Chlamydia trachomatis*, infections for which prevalence typically decreases with age. Increasing prevalence with age is consistent with a disease with an extremely long duration of infectiousness, which is predominantly asymptomatic and which is not treated inadvertently by antibiotics given for unrelated medical conditions. Another implication of this for control is that those targeted for *T vaginalis* treatment must include the older age groups, at least in the initial stages.

Recent community based studies from the developing world have demonstrated that sexually transmitted diseases (including *T vaginalis*) are usually asymptomatic.[18 19] The definition of what constitutes symptomatic disease is crucial here: different cultural, educational, and economic contexts are as important as the biology of the organism in determining whether people seek medical care for genital complaints. Symptoms that would prompt a woman to seek urgent treatment in Sydney might be seen as a normal part of life for a woman in rural South Africa where as many as one in two women are infected.[12]

Are there serious sequelae of infection with *T vaginalis*?

The florid manifestations of acute pelvic inflammatory disease or the more insidious complications such as infertility and ectopic pregnancy are important reasons for controlling *C trachomatis* and *N gonorrhoeae*. *T vaginalis* is not thought to be a major cause of any of these sequelae and, as a result, many clinicians have considered the disease more of a nuisance than a public health problem. It may be time to question this in the light of the hidden epidemic of premature labour and low birth weight in disadvantaged populations, and the more visible problem of HIV infection.

The vaginal infections in pregnancy (VIP) study found that infection with *T vaginalis* resulted in a modest increase in the risk of premature labour (relative risk 1.3).[11] While this is of minor importance in populations with a low *T vaginalis* prevalence, the attributable risk (that is, the proportion of cases of premature labour that can be attributed to infection with *T vaginalis*) will be substantial in endemic populations. In this study the attributable risk was only 1.5% in white women but 11% in black women. In Africa, assuming a similar pathogenesis, the attributable risk may be in the order of 20–25%. Further work to confirm the results of the VIP study and to demonstrate a risk reduction following treatment is urgently required.

Most studies on the relation between HIV transmission risk and co-factor STDs have highlighted genitoulcerative conditions and the bacterial causes of cervicitis and urethritis. Indeed, in one of the few adequate studies that have looked at *T vaginalis* and HIV risk[20] the odds ratio is low (and not statistically significant). However, as Sorvillo and Kerndt[21] point out, even a modest increase in the risk of transmission (say 90%) will translate into an attributable fraction for HIV of nearly 20% in areas where the prevalence of *T vaginalis* is 25%. The prevalence of *N gonorrhoeae* and *C trachomatis* is surprisingly low in many African studies and varies from region to region: in the Ugandan study of mass STD treatment to reduce HIV incidence the prevalence was 2.1% and 4.0% respectively in a subgroup of the study population[18] while the prevalence of the same organisms was 2.2% and 0.7% in a survey in Tanzania.[22] The attributable fraction of HIV cases caused by *N gonorrhoeae* and *C trachomatis* in these populations will therefore be small, even though the relative risk of transmission for the individual may be substantial. *T vaginalis* prevalence, on the other hand, was 24% in the Ugandan study and is consistently found at or above this level in STD surveys.

The other conditions of likely importance in HIV transmission (for example, herpes genitalis and bacterial vaginosis) are not easily treated. It is possible that control of *T vaginalis* could be the single most cost effective and achievable strategy for the reduction of HIV incidence.

Conclusions

Sadly, it has taken a catastrophe like the HIV pandemic to draw attention to the predominantly silent epidemic of other STDs and it would be counterproductive to redirect resources for *T vaginalis* control from the more "established" STD control efforts. However, it is a curious paradox that one of the reasons for *T vaginalis* infection's effective "invisibility" may be its ubiquity in some populations. How could something so common be important?

While specific options for control of *T vaginalis* are explored elsewhere (Bowden F, Garnett G. Trichomonas vaginalis: epidemiology and treatment interventions for an HIV cofactor STD, submitted for publication) a first step is to change the way the disease is viewed by the various layers of the health sector. A safe, well tolerated antibiotic costing a few cents a dose is available which could be dispensed in a wide range of clinical settings with minimal risk of clinically important antibiotic resistance developing in other organisms (something which is not true for the indiscriminate use of antibiotics for the treatment of *N gonorrhoeae*). The need for diagnostic testing before treatment is dependent on the endemic prevalence of the disease: because the earlier probability of being infected with *T vaginalis* in African women is between 25 and 50% there is little need for a diagnostic test. As the prevalence falls, the usefulness of testing rises but how this can be achieved in resource poor settings remains a problem.

Trichomoniasis is a disease which usually produces minimal or no symptoms and which is associated with a only a small risk of complications for the infected individual. However, if one analyses the problem of control from a population health perspective, control of the disease may, depending on its influence on HIV transmission, represent one of the most effective means of reducing HIV transmission risk and of improving the general wellbeing and reproductive health of a large proportion of the world's population.

FRANCIS J BOWDEN
Wellcome Trust Centre for the Epidemiology of Infectious Disease, Oxford University, Oxford OX1 3PS and Territory Health Services, PO Box 40596, Casuarina, NT, Australia, 0811

GEOFFREY P GARNETT
Wellcome Trust Centre for the Epidemiology of Infectious Disease, Oxford University, Oxford OX1 3PS

Financial support: FJB was supported by a Wellcome Trust travelling fellowship; GPG is a Royal Society university research fellow.

1 The control of human trichomoniasis. (Editorial) *Br J Vener Dis* 1960;**36**:145–6.
2 Trussell RE. Trichomonas vaginalis and trichomoniasis. Oxford, 1948.
3 Peterson P. Trichomonas vaginalis vaginitis. A comparative study of treatment and incidence. *Am J Obstet Gynec* 1938;**35**:1004–9.
4 Feo LG. The incidence and significance of Trichomonas vaginalis in the male. *Am J Trop Med* 1943;**24**:195–8.
5 Burch TA, Rees CW, Reardon LV. Epidemiological studies on human trichomoniasis. *Am J Trop Med Hyg* 1959;**8**:312–18.
6 Ipsen J, Feigl P. A biomathematical model for prevalence of Trichomonas vaginalis. *Am J Epidemiol* 1970;**91**:175–84.

7 Csonka GW. Long-term aspect of treatment with metronidazole (Flagyl) in trichomonas vaginitis. *Br J Vener Dis* 1963;**39**:258–60.
8 Tapsall JW, Puglisi J, Smith DD. Trichomonas vaginalis infections in Sydney: laboratory diagnosis and prevalence. *Med J Aust* 1979;**1**:193–4.
9 Lefevre JC, Lepargneur JP, Bauriaud R, *et al.* Clinical and microbiologic features of urethritis in men in Toulouse, France. *Sex Transm Dis* 1991;**18**:76–9.
10 Stefanik M, Rychna K, Valkoun A. Microbial causative agents of male urethritis. *J Hyg Epidemiol Microbiol Immunol* 1992;**36**:111–8.
11 Cotch MF, Pastorek JG 2nd, Nugent RP, *et al.* Trichomonas vaginalis associated with low birth weight and preterm delivery. The Vaginal Infections and Prematurity Study Group. *Sex Transm Dis* 1997;**24**:353–60.
12 O Farrell N, Hoosen AA, Kharsany AB, *et al.* Sexually transmitted pathogens in pregnant women in a rural South African community. *Genitourin Med* 1989;**65**:276–80.
13 Klouman E, Masenga EJ, Klepp KI, *et al.* HIV and reproductive tract infections in a total village population in rural Kilimanjaro, Tanzania: women at increased risk. *J Acquir Immune Defic Syndr Hum Retrovirol* 1997;**14**:163–8.
14 Tabrizi SN, Paterson B, Fairley CK, *et al.* A self-administered technique for the detection of sexually transmitted diseases in remote communities. *J Infect Dis* 1997;**176**:289–92.
15 Zigas V. An evaluation of trichomoniasis in two ethnic groups in Papua New Guinea. *Sex Transm Dis* 1977;**4**:63–5.
16 McCullagh WMH. The gap seat. *Lancet* 1953;i:698.
17 Krieger JN, Alderete JF. Trichomonas vaginalis and trichomoniasis. In: Holmes KK SP, Mardh PA, Lemon SA, Stamm WE, Piot P, Wasserheit JN, eds. *Sexually transmitted diseases.* New York: McGraw-Hill, 1999.
18 Wawer M, Sewankambo N, Serwadda D, *et al.* Control of sexually transmitted diseases for AIDS prevention in Uganda: a randomised community trial. *Lancet* 1999;**353**:525–35.
19 Passey M, Mgone CS, Lupiwa S, *et al.* Community based study of sexually transmitted diseases in rural women in the highlands of Papua New Guinea: prevalence and risk factors. *Sex Transm Inf* 1998;**74**:120–7.
20 Laga M, Manoka A, Kivuvu M, *et al.* Non-ulcerative sexually transmitted diseases as risk factors for HIV-1 transmission in women: results from a cohort study. *AIDS* 1993;**7**:95–102.
21 Sorvillo F, Kerndt P. Trichomonas vaginalis and amplification of HIV-1 transmission[[letter]]. *Lancet* 1998;**351**:213–4.
22 Grosskurth H, Mosha F, Todd J, *et al.* Impact of improved treatment of sexually transmitted diseases on HIV infection in rural Tanzania: randomised controlled trial [see comments]. *Lancet* 1995;**346**:530–6.

Avoiding HIV transmission: women need more options

There is an urgent need for effective, safe, and affordable methods that women can use to protect themselves and their newborn infants from infection with sexually transmitted diseases (STDs), including HIV/AIDS. International attention has increasingly turned to the prospect for microbicides—chemical substances in the form of a gel, cream, suppository, or film that kills or neutralises micro-organisms and bacteria—which, when applied vaginally or rectally, would be within women's personal control. The WHO, UNAIDS, and other international organisations have urged that high priority be given to microbicide development. However, there have to date been few resources allocated specifically for research in this area despite growing evidence of the devastating impact of STDs/HIV on women and their families.

There are an estimated 400 million cases of new STDs worldwide each year, and evidence that their rate is high and increasing.[1] STDs impose an enormous health burden on women and are also a risk factor for the acquisition of HIV infection.[2]

At the end of 1998, more than 33 million people globally were infected with HIV. Women now represent 43% of those over age 15 living with HIV and AIDS.[3] In some parts of sub-Saharan Africa, infection rates for women now substantially exceed those for men. In industrialised countries women comprise the fastest growing population with HIV infection. This trend is expected to continue, resulting in an even greater burden of disease for women.

Women who are infected with HIV also put children at great risk. Women in developing countries with HIV/AIDS have a 25–35% risk of transmitting the virus to their newborn infants,[4] and most infected infants die within 2 years. Vertical transmission of HIV (from mother to child) can occur in utero, at delivery, or post partum via breast feeding.

Fifteen years into the HIV epidemic, the public health community still has very little to offer women to protect themselves or their infants from infection. All currently available HIV/STD *prevention* strategies—monogamy, using condoms, and treating STDs—have significant limitations for women. Even if women are monogamous, their partners often are not. Women may risk rejection and violence if they try to urge their partners to use condoms, and condom use remains low in much of the world, even in many countries with high rates of HIV infection and active AIDS control programmes. Female condoms are not widely available, require partner cooperation, and are relatively expensive. Finally, diagnosis and treatment of other STDs are not available in most parts of the developing world.

Clearly, there is an urgent need for prevention technology that falls within women's personal control. Such methods would provide women with potentially life saving alternatives when they are unable to negotiate condom use. To date, the only candidate microbicides to reach advanced clinical testing are nonoxynol-9 containing spermicides. Nonoxynol-9 (N-9) is the active ingredient in the majority of existing vaginal spermicides available today. Conflicting results from two separate trials of an N-9 sponge[5] and an N-9 film formulation[6] may be resolved as early as next year when the results from an N-9 containing gel formulation study become available. Continued testing and evaluation of N-9 are certainly justified and important as this is a compound that, if proved effective against HIV, is poised for immediate distribution and is already available to a large proportion of the world's women who need immediate protection from their partners with HIV infection.

Ultimately, however, we will need more than just N-9. Nonoxynol-9 is spermicidal and we have learned from numerous studies[7–9] that for many of the world's women, a spermicidal microbicide will not be suitable for those women who want to be able to conceive while protecting themselves from HIV infection.

A variety of other candidate microbicides are currently nearing the clinical testing stage. These tend to work in one (or more) of four ways: killing or inactivating the virus (for example, N-9, benzalkonium chloride, buffer gel); blocking adhesion or inhibiting viral entry to the vaginal or cervical cells (for example, sulphated polymers, bioengineered molecules); attacking the virus with immunological weapons; or inhibiting viral replication once the virus has already entered the cells (for example, nucleoside/nucleotide reverse transcriptase inhibitors).[10] Of the 50 plus compounds currently under development, 23 are in some form of clinical testing. Two potentially microbicidal compounds have advanced to clinical evaluation. Unfortunately, owing to the complicated nature of these trials and numbers of participants needed to show effectiveness, we expect it will be 3–5 years before we have any kind of conclusive data showing effectiveness against HIV.

For those women who are *already* HIV positive and pregnant, antiretroviral therapy with zidovudine given to the mother during pregnancy and delivery, and to the

infant, can reduce transmission by approximately 70%.[11 12] Unfortunately, the cost of antiviral therapy such as AZT (approximately US$1000 per woman) carries it out of reach for most women of the world. Studies are under way in the developing world that examine the efficacy of using lower levels of the AZT regimen in order to reduce viral load and decrease vertical transmission of HIV during pregnancy.

One compound that has been shown to inactivate HIV in in vitro studies is benzalkonium chloride.[13 14] Benzalkonium chloride has been used for years as a spermicide and may have tremendous potential as a vaginal microbicide in pregnant women and, because it has been shown to have no toxic effect,[15] may be useful in solution as a neonatal wash to reduce transmission of HIV. The study described in this issue of *STI* (p 420) conducted in west Africa by Msellati *et al*, explores the safety of benzalkonium chloride in HIV infected pregnant urban women and their newborns in Côte d'Ivoire and Burkina Faso. This is a well designed, randomised, double blinded placebo controlled trial to look at the effects of a benzalkonium chloride solution on vaginal and cervical mucosa, and on neonates when bathed in the solution. The researchers found that the incidence of adverse events (primarily leucorrhoea) did not differ significantly between the women's treatment groups. In the neonates, the incidence of dermatitis and conjunctivitis did not differ between the benzalkonium chloride and placebo groups. Given these encouraging first results, it will be important to continue assessment of this compound in effectiveness trials to evaluate benzalkonium chloride's ability to actually reduce vertical transmission of HIV.

In addition to further work with benzalkonium chloride, it is critical that researchers and funding agencies actively pursue all approaches to preventing the spread of HIV and other STDs. The need for an HIV vaccine is urgent, and it is encouraging that a number of funding agencies have made major investments in this technology. However, since it remains unclear how long it will take to develop a safe and effective vaccine, and since neither vaccines nor microbicides are likely to be 100% effective or available everywhere, there is a compelling need to pursue both approaches vigorously and simultaneously.

CHRISTIANA COGGINS

Santa Fe, New Mexico, USA

1 World Health Organisation. *An overview of selected curable sexually transmitted diseases. Global programme on AIDS*. WHO/GPA/STD/95.1. Geneva: WHO, 1995.
2 Wasserheit JN. Epidemiological synergy: interrelationships between human immunodeficiency virus infection and other sexually transmitted diseases. *Sex Transm Dis* 1992;**19**:61–77.
3 UNAIDS/WHO. AIDS epidemic update: December 1998. Geneva: UNAIDS/WHO, 1998.
4 UNAIDS/WHO. *Report on global HIV/AIDS and STD surveillance*. Geneva: UNAIDS/WHO, 1998.
5 Kreiss J, Ngugi E, Holmes K, *et al*. Efficacy of nonoxynol-9 contraceptive sponge use in preventing heterosexual acquisition of HIV in Nairobi prostitutes. *JAMA* 1992;**268**:477–82.
6 Roddy R, Zekeng L, Ryan K, *et al*. A controlled trial of nonoxynol 9 film to reduce male-to-female transmission of sexually transmitted diseases. *N Engl J Med* 1998;**339**:504–10.
7 Blanchard K, Coggins C, Friedland B. Men's attitudes toward a potential vaginal microbicide in Mexico, the United States and Zimbabwe, Poster session 33141. 12th World AIDS Conference, Geneva, 28 June–3 July 1998.
8 Coggins C, Elias CJ, Atisook R, *et al*. A study of women's preferences regarding the formulation of over-the-counter vaginal spermicides. *AIDS* 1998;**12**:1389–91.
9 Van de Wijgert J, Khumalo-Sakutukwa GN, Coggins C, *et al*. Men's attitudes toward vaginal microbicide use in Zimbabwe. *Int Fam Plan Persp* 1999;**25**:15–20.
10 Elias CJ, Coggins C. Female-controlled methods to prevent sexual transmission of HIV. *AIDS* 1996;**10**(suppl 3):S43–51.
11 Connor EM, Sperling RS, Gelber R, *et al*. Reduction of maternal-infant transmission of human immunodeficiency virus type 1 with zidovudine treatment, Pediatric AIDS Clinical Trials Group Protocol 076 Study Group. *N Engl J Med* 1994;**331**:1173–80.
12 Carmichael C. Preventing perinatal HIV transmission: zidovudine use during pregnancy. *Am Fam Phys* 1997;**55**:171–4, 177–8.
13 Sattar SA, Stringthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev Infect Dis* 1991;**13**:430–47.
14 Wainberg MA, Spira B, Bleau G, *et al*. Inactivation of human immunodeficiency virus type 1 in tissue culture fluid and in genital secretions by the spermicide benzalkonium chloride. *J Clin Microb* 1990;**28**:156–8.
15 Erny R, Siboni C. Les tampons au chlorure de benzalkonium: tolérance locale et effets sur la glaire cervicale. *J Gynec Obstet BioReprod* 1983;**12**:767–74.

Sexual dysfunction associated with treatment of cervical cancer

Women treated for cervical carcinoma often develop psychosexual dysfunction,[1] and have to cope with the fact that this is a sexually transmitted disease. Therefore, feelings of guilt are common. Fears that intercourse would cause the cancer to recur, or that cancer could be transmitted to a sexual partner are frequently expressed among these women.[2] Treatment of cervical cancer may result in vaginal abnormalities that severely interfere with sexual function. Although numerous studies have documented the distress associated with the loss of a breast, changes in the vagina have been neglected in this respect.[3] The paucity of research on the effect of vaginal changes on sexual function is remarkable. It seems inexcusable that patients can undergo months or years of invasive and expensive treatment when simple clear questions about their sexual lives are not discussed at all. One explanation may be that for men female breasts may have aesthetic as well as sexual value which may influence research policies in academic medicine where male investigators predominate. Lowered self esteem, poor body image, loss of femininity, anxiety about desirability, depression, and aversion to the changes in physical appearance are common in patients following radical genital tract surgery. Common sexual problems include vaginismus, dyspareunia, loss of desire, and anorgasmia. Specific problems after surgery, radiotherapy, or both for cervical cancer include a shortened vagina, reduced vaginal lubrication, and reductions in vaginal elasticity or genital swelling during sexual stimulation. Although decline in sexual function is the most common cause of disease specific distress in men with prostate cancer,[4] such effects of vaginal changes on the women's sexuality have received little study.

Table 1 Percentage of older men and women who are sexually active and find it enjoyable

	Age group (years)		
	50–59	*60–69*	*70–79*
Sexually active subjects			
All women (n=1844):	93	81	65
Married (n=1245)	95	89	81
Unmarried (n=512)	98	63	50
All men (n=2402)	98	91	79
Married (n=1895)	98	93	81
Unmarried (n=414)	95	85	75
Sex highly enjoyable (sexually active subjects)			
Women	71	65	61
Men	90	86	75

*Modified from Read.[5]

Table 2 Frequency of sexual dysfunction in women with cervical carcinoma and controls

Variable	*Women with cancer (n=256)*	*Controls (n=350)*	*Age adjusted RR (95% CI)* *
Frequency of vaginal intercourse			
None in previous 6 months	80/247 (32)	94/330 (28)	1.2 (1.0–1.5)
Vaginal changes in previous 6 months			
Moderate or substantial reduction in length of vagina during intercourse†	52/197 (26)	(8/240 (3)	8.1 (4.4–14.99)
Moderate or substantial reduction in elasticity of vagina during intercourse†	45/195 (23)	9/246 (4)	6.7 (3.6–12.5)
Women reporting any vaginal changes	62/127 (49)	25/97 (26)	1.8 (1.3–2.6)
Problems during intercourse			
Superficial dyspareunia in previous 6 months†	31/196 (16)	5/246 (2)	8.5 (3.5–18.6)
Deep dyspareunia in previous 6 months†	24/196 (12)	6/245 (2)	5.2 (2.4–11.4)
Vaginal bleeding during intercourse at least every other time in previous 6 months†	14/177 (8)	1/246 (<1)	20.6 (4.8–88.7)
Vaginal lubrication moderately or very insufficient in previous 6 months	46/177 (26)	27/248 (11)	2.5 (1.6–3.8)

*RR denotes relative risk, and CI confidence interval.
†Respondents included only sexually active women.
Modified from Bergmark *et al.*[3]

An additional problem for women in the older age groups may be that there has been a widespread tendency to assume that elderly people are too old for sexual activity and that sexuality of both men and women rapidly declines with advancing years. However, this is not evidence based.[5] People who have been sexually active on a frequent basis throughout their life will show a lower rate of decline in activity with advancing age than those who have been less sexually active. In fact, a surprisingly large proportion of men and women over 70 years old remain sexually active (table 1).

Bergmark and others[3] recently conducted an important study to determine the prevalence of vaginal changes among women who had been treated for cervical cancer and the extent to which these changes affected their sexuality and caused stress and sexual dysfunction. The study group consisted of 332 women under the age of 80 years with a history of early stage (IB or IIA) cervical cancer who had been treated in Sweden between January 1991 and December 1992, and who were registered and alive in November 1996. They randomly selected 489 matched control women without cervical cancer from the Swedish population register. The questionnaire included 136 questions. The response rate was 77% among the cases and 72% among the controls. Of the cases 68% and 72% of the controls reported regular vaginal intercourse. Sexual dysfunction was more common among cases than among controls: 26% of the women who had cancer and 11% of the controls reported insufficient vaginal lubrication; 26% of the women who had cancer and 3% of the controls reported a short vagina; and 23% of the women who had cancer and 4% of the controls reported insufficient vaginal elasticity. Overall, 26% of the women who had cancer reported moderate or much distress because of vaginal changes compared with 8% of the women in the control group. Dyspareunia was also more common among the women who had cervical cancer (16% *v* 2%). The type of treatment had little if any effect on the prevalence of specific vaginal changes (table 2).

The authors conclude that women who have been treated for cervical cancer have persistent vaginal changes which compromise sexual activity and result in considerable distress. The women in the two groups reported a similar level of sexual satisfaction with their partners and quite similar overall sexual activity although more of the women who had cancer were single, suggesting that some relationships end as a consequence of cervical cancer. However, it would have been even more helpful if they had had another control group of women undergoing gynaecological surgery for benign disease.

Clearly, physicians caring for women with cervical cancer should discuss possible treatment related vaginal changes that may affect sexual function. This topic should be thoroughly addressed both before and after treatment. These findings suggest that women should not only have sexual education and advice at the time of cancer treatment, but should have follow up assessment of sexual function between 6 months and 1 year to identify late appearing sexual morbidity.[6] By providing these services we could improve the quality of life as well as quality of sexual function of these women. It is important to emphasise that there is really no age above which sexual function is not important to women undergoing treatment for cervical cancer. It is essential to remember that elderly people may have just as many sexual interests and preferences as younger people. Myths and beliefs about sexual activity and attractiveness and what it is may affect older women even more than younger women and therefore may contribute to low self esteem, body image, and depression. In conclusion, we applaud Dr Bergmark and her collaborators for their case-control study highlighting an important but largely neglected research area.

JORMA PAAVONEN
Department of Obstetrics and Gynaecology, University of Helsinki, Haartmaninkatu 2, 00290 Helsinki, Finland

1 Corney RH, Crowther ME, Everett H, *et al.* Psychosexual dysfunction in women with gynaecological cancer following radical pelvic surgery. *Br J Obstet Gynaecol* 1993;**100**:73–8.
2 Seibel MM, Freeman MG, Graves WL. Carcinoma of the cervix and sexual function. *Obstet Gynecol* 1980;**55**:484–7.
3 Bergmark K, Åvall-Lundqvist E, Dickman PW, *et al.* Vaginal changes and sexuality in women with history of cervical cancer. *N Engl J Med* 1999;**340**:1383–9.
4 Helgason AR, Adolfsson J, Dickman P, *et al.* Waning sexual function—the most important disease-specific distress for patients with prostate cancer. *Br J Cancer* 1996;**73**:1417–21.
5 Read J. Sexual problems associated with infertility, pregnancy, and ageing. *BMJ* 1999;**318**:587–9.
6 Schover LR, Fife M, Gershenson DM. Sexual dysfunction and treatment for early stage cervical cancer. *Cancer* 1989;**63**:204–12.