

52. **Morris M et al.** Concurrent partnerships and HIV transmission in Uganda. XIth International AIDS Conference, July 7–July 11, Vancouver, Canada, 1996 (Abstract TuD473).
53. **Kelly R et al.** Concurrent sexual partnerships and risk of prevalent and incident HIV. XIII International AIDS Conference, Durban, South Africa, 9–14 July 2000 (Abstract MoPpC1027).
54. **Obbo C.** HIV transmission through social and geographical networks in Uganda. *Social Science and Medicine*, 1993, **36**: 949–955.
55. **Kinloch-de Loes S et al.** Symptomatic primary infection due to HIV-1: review of 31 cases. *Clinical Infectious Diseases*, 1993, **17**: 59–65.
56. **Bratt GA et al.** Two cases of oral-genital HIV-1 transmission. *International Journal of STD and AIDS*, 1997, **8**: 522–525.
57. **Kamenga M et al.** Evidence of marked sexual behaviour change associated with low HIV-1 seroconversion in couples with discordant HIV serostatus. *AIDS*, 1991, **5**: 61–67.
58. **Anderson J et al.** Idiopathic genital ulcers in women infected with HIV. *Journal of Acquired Immune Deficiency Syndromes*, 1996, **13**: 343–347.
59. **Fiscus SA et al.** Transient high titers of HIV-1 in plasma and progression of disease. *Journal of Acquired Immune Deficiency Syndromes*, 1995, **9**: 51–57.
60. **Seage GR et al.** Increased suppressor T cells in probable transmitters of HIV. *American Journal of Public Health*, 1989, **79**: 1638–1642.
61. **Wawer M et al.** Preventing HIV-1: lessons from Mwanza and Rakai. *Lancet*, 1999, **353**: 1523–1524.
62. **Mosha F et al.** A population-based study of syphilis and sexually transmitted disease syndromes in north-western Tanzania. I. Prevalence and incidence. *Genitourinary Medicine*, 1993, **69**: 415–420.
63. **Barongo LR et al.** The epidemiology of HIV-1 infection in urban areas, roadside settlements and rural villages in Mwanza, Tanzania. *AIDS*, 1992, **6**: 1521–1528.
64. **Wawer MJ et al.** Incidence of HIV infection a rural region of Uganda. *British Medical Journal*, 1994, **308**: 171–173.
65. **Konde-Lule J, Musagara M, Musgrave S.** Focus group interviews about AIDS in Rakai district of Uganda. *Social Science and Medicine*, 1993, **37**: 679–684.
66. **West B et al.** Antimicrobial susceptibility, auxotype and plasmid content of *Neisseria gonorrhoeae* in North Tanzania. *Genitourinary Medicine*, 1995, **71**: 9–12.
67. **Kamali A, Nabaitu J, Kinsman J.** A randomized controlled community intervention to reduce HIV-1 infection in Uganda. XIII International AIDS Conference, Durban, South Africa, 9–14 July 2000 (Abstract WePpC1314).
68. **Basajja V et al.** A community-based condom social marketing strategy in rural Uganda. XIII International AIDS Conference, Durban, South Africa, 9–14 July 2000 (Abstract MoOrC244).
69. **Seeley JA, Kengeya-Kayondo J, Mulder D.** Community-based HIV/AIDS research. *Social Science and Medicine*, 1992, **34**: 1089–1095.
70. **Laga M.** STD control for HIV prevention. *Lancet*, 1995, **346**: 518–519.
71. **Shilts R.** *And the band played on: politics, people, and the AIDS epidemic.* New York, St Martin's Press, 1987.

Commentaries

STI care: one of many necessary approaches for prevention of HIV infection

Kevin R. O'Reilly¹ & Antonio Carlos Gerbase²

Challenging orthodoxy always has an appeal to some, and we must confess we are part of that group. In his article (1), Hudson is challenging a hastily built orthodoxy — derived from the results of the Mwanza trial — that spread rapidly and largely without question throughout the public health world. Following publication of the trial results, which seemed to indicate that management of sexually transmitted infection (STI) could result in significant reduction of HIV incidence, many were quick to conclude that an important AIDS-prevention tool was at hand (2). The fact that the original hypothesis of the study, linking STI management and HIV transmission, was not proven was eclipsed by an important decline in HIV incidence in Mwanza — at a time when the public health community needed hope. Perhaps STI prevalence did not decline as much as HIV incidence, reasoned the authors, because of the high prevalence of asymptomatic

infection among women. It is not possible to address asymptomatic infection with syndromic management, the main STI management strategy used in the Mwanza trial.

Enter the results of the Rakai trial, which was meant to address the problem of asymptomatic STI by presumptive mass treatment. However, for reasons made clear by Hudson and others (1, 3–5), the Rakai trial failed to reduce HIV incidence at all. Hudson suggests this failure may stem from a misreading of the results of the Mwanza trial. To assume that the link between STI management and HIV transmission was conclusively proven at Mwanza is to ignore the nuances and context of the trial: these must be considered in order to make sense of the complicated epidemiology of HIV/AIDS.

Many facts argued for caution in interpreting the Mwanza results: the well-known observation that “first trials” commonly produce more positive results than subsequent trials; the fact that the original hypothesis was not really proved; and the seemingly inexorable drive to find a single intervention, a “magic bullet”, that will make control of the epidemic possible. It has long been known that no magic bullet exists nor is likely to for some time to come, and that a multiplicity of approaches is needed (6). One key approach must be to encourage behavioural change. Changing sexual behaviour, the first element of any public health programme to control HIV, is a strategy that often enjoys too little confidence in such programmes, despite the fact that there are no national examples in which the spread of HIV has

¹ Scientist, Department of Reproductive Health and Research, World Health Organization, 1211 Geneva 27, Switzerland. Correspondence should be addressed to this author.

² Team Coordinator on Prevention, HIV/AIDS/STI Initiative, World Health Organization, Geneva, Switzerland.

been diminished or brought under control without behavioural change playing an important, if not the key role.

Efforts to prevent HIV infection by encouraging behavioural change are either being increasingly ignored (7) or are being forced to compete against other approaches for already insufficient resources. Successful interventions involving behavioural change, are — we know now — not notable for their sophistication, but rather for their consistency, intensity, and duration. More research is needed to determine if there are ways to achieve the same ends with less effort. Currently, however, we know how to prevent HIV infection, we lack only the will.

If the architects of these two key trials in Mwanza and Rakai can be blamed for anything, it is for paying insufficient attention to behaviour and behavioural change, either in their efforts to reduce sexual risk (8) or, as Hudson points out (1), by failing to consider important behavioural changes already taking place. If the interpreters of the Mwanza trial can be held accountable for anything, it is for overstating the results and trying to justify STI management through HIV control. STI care certainly has a role in HIV prevention, but it is also an important public health activity. Overselling STI management as the sole intervention for HIV control will only damage efforts to address an epidemic that has long preceded HIV/AIDS.

The history of public health efforts in AIDS prevention will undoubtedly show the folly of ignoring what we know in favour of what we might prefer. Hudson has reminded us (1), as we must be reminded frequently, it seems, that there is no single answer, that multiple approaches must be used probably everywhere, and that behavioural change remains a key component everywhere. To place undue effort on any one intervention is ill-advised and certainly not justified by the evidence produced to date. ■

References

1. **Hudson CP.** Community-based trials of sexually transmitted disease treatment: repercussions for epidemiology and HIV prevention. *Bulletin of the World Health Organization*, 2001, **79**: 48–58.
2. **Laga M.** STD control for HIV prevention. *Lancet*, 1995, **346**: 518–519.
3. **Hitchcock P, Fransen L.** Preventing HIV: lessons from Mwanza and Rakai. *Lancet*, 1999, **353**: 513–515.
4. **Grosskurth H et al.** Control of sexually transmitted diseases for HIV-1 prevention: understanding the implications of the Mwanza and Rakai trials. *Lancet*, 2000, **355**: 1981–1987.
5. **Wawer M et al.** Preventing HIV-1: lessons from Mwanza and Rakai. *Lancet*, 1999, **353**: 1523–1524.
6. **Cates W, Hinman AR.** AIDS and absolutism — the demand for perfection. *New England Journal of Medicine*, 1992, **327**: 492–494.
7. **Merson M, Rosenfeld A.** The AIDS epidemic: lessons learned? *Lancet*, 2000, **356**: 1204.
8. **O'Reilly K et al.** Impact of improved treatment of STD on HIV. *Lancet*, 1995, **346**: 1158.

More community-based trials of STD control or more appropriate interventions: which is the priority for preventing HIV-1 infection in developing countries?

Michel Alary¹

Much debate has followed the publication of the results of the Rakai trial in 1999 (1). Given the outcome of the Mwanza trial published a few years earlier (2), most of the AIDS scientific community was expecting positive results from Rakai. Indeed, how could periodic mass treatment for sexually transmitted disease (STD) fail to prevent HIV-1 transmission at the community level when a much more modest intervention (appropriate STD treatment for people with STD symptoms attending primary health care centres) had led to a 38% reduction in HIV-1 incidence?

The main reason evoked up until now for these discrepant results is the difference in the stage of the HIV epidemic at the study sites (3, 4). Indeed, as confirmed by a sub-analysis of the Rakai data (5), the population attributable fraction for STD in HIV-1 transmission will be quite low when the HIV epidemic reaches a high but stable prevalence with a low to moderate incidence. Other reasons put forward have been the increase in herpes simplex virus type 2 infection (an incurable STD) at the population level in mature epidemics, which could then contribute to further HIV-1 transmission; the possibility of reinfection occurring between mass treatment cycles; the possibility that symptomatic STDs play a more important role in HIV-1 transmission than asymptomatic STDs; and chance (4), with imbalance between the study arms at baseline resulting in wide confidence intervals (6).

In this issue of the *Bulletin of the World Health Organization*, Hudson presents new arguments (7), adding to the debate. He draws three main points: firstly, in contrast to most previous analyses, he considers that the Rakai trial should be considered as the gold standard, and it is the Mwanza results which need to be “explained”, mainly because reinfection between mass treatment rounds is unlikely to be a major factor accounting for the negative results from Rakai. Secondly, behavioural counselling, rather than STD treatment, could explain the Mwanza results, mainly because STD symptoms, in people contracting HIV-1 and an STD simultaneously, may prompt attendance at a clinic prior to manifestations of primary HIV-1 infection; this would lead to abstinence or consistent condom use during the earliest period of viraemia associated with primary

¹ Visiting Professor, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London, WC1E 7HT, England.

Ref. No. 00-1121