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HIV Decline Associated with Behavior Change in Eastern Zimbabwe

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Few sub-Saharan African countries have witnessed declines in HIV prevalence, and only Uganda has compelling evidence for a decline founded on sexual behavior change. We report a decline in HIV prevalence in eastern Zimbabwe between 1998 and 2003 associated with sexual behavior change in four distinct socioeconomic strata. HIV prevalence fell most steeply at young ages—by 23 and 49%, respectively, among men aged 17 to 29 years and women aged 15 to 24 years—and in more educated groups. Sexually experienced men and women reported reductions in casual sex of 49 and 22%, respectively, whereas recent cohorts reported delayed sexual debut. Selective AIDS-induced mortality contributed to the decline in HIV prevalence.

Surveillance data indicate that HIV prevalence has declined in several countries in east Africa (1). However, even in Uganda, the country with the most extensive evidence of a large-scale, long-term decline in HIV prevalence (2, 3), controversy surrounds both the existence of a decline and its attribution to sexual behavior change (4–6). This controversy has been fed by three factors: (i) doubts surrounding the representativeness of HIV surveillance data drawn from pregnant women attending antenatal clinics at selected sites within the country (7); (ii) the possibility that declines in HIV prevalence could occur in the absence of the deliberate adoption of safer behaviors (8, 9), i.e., as a result of saturation of infection and selective mortality within high-risk groups; (iii) a paucity of data directly linking declines in HIV prevalence to the adoption of safer sexual behaviors (10). Until now, there has been no evidence for decline in HIV prevalence in southern African countries (1).

We examined changes in HIV prevalence and sexual behavior occurring between 1998 and 2003 in Manicaland, Zimbabwe, in an open population cohort of 9454 adults recruited in two household censuses—the first conducted between July 1998 and February 2000, the second 3 years later—and concurrent changes in HIV prevalence among local antenatal clinic attendees. Patterns of mortality and new infection were explored in the closed cohort of individuals recruited at baseline. Data were collected from 12 communities enumerated in succession in four socioeconomic strata (11), including small towns, forestry/tea/coffee estates, roadside settlements, and subsistence farming areas. Participation rates compared well with those obtained in similar studies (table S1).

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HIV prevalence in the 12 study sites was observed to decline over an average 3-year intersurvey interval from 23.0% to 20.5% [adjusted odds ratio (AOR), 0.87; 95% confidence interval (CI), 0.80 to 0.95] (table S2). HIV prevalence had declined from 19.5% to 18.2% in men aged 17 to 54 years (AOR, 0.84; 95% CI, 0.74 to 0.96) and from 25.9% to 22.3% in women aged 15 to 44 years (AOR, 0.88; 95% CI, 0.79 to 0.98). Absolute declines in HIV prevalence were recorded in all four socioeconomic strata (table S3) and in 10 of the 12 study sites (significantly more than half of the sites enumerated; $P = 0.039$).

HIV prevalence declined in men aged 17 to 34 years and women aged 15 to 29 years (Fig. 1). The decline in HIV prevalence was most pronounced in men aged 17 to 29 years, from 10.6% to 8.1% (a decline of 23%; $P < 0.01$), and in women aged 15 to 24 years, from 15.9% to 8.0% (49%; $P < 0.001$). HIV prevalence increased among respondents aged over 35 years ($P < 0.025$) (table S2). The age pattern of change in HIV prevalence is consistent with one that occurs through the natural dynamics of an HIV epidemic (12), but the quantum of the declines recorded in younger age groups, over a relatively short 3-year period, and the concentration of the decline among people with sec-

ondary school education (13) strongly suggested a contribution of sexual behavior change (3). Through the phased sampling, it was possible to see that the change in HIV prevalence occurred consistently over time (table S3).

Surveillance data from local antenatal clinic attendees indicated modest declines in HIV prevalence overall (21.1% to 19.2%; AOR, 0.87; 95% CI, 0.71 to 1.06) and at young ages (fig. S1). HIV prevalence in young pregnant women is lower than in women in the general population, owing to the reduced fertility of HIV-infected women (14).

Overall, HIV prevalence increased over time among individuals seen at baseline (i.e., members of the closed cohort). The contributions of mortality and new HIV infections to changes in HIV prevalence observed in the closed cohort are shown in Fig. 2 and table S4. HIV incidence was highest in men aged 20 to 44 years and women aged 15 to 29 years. Mortality among all uninfected individuals was low (less than one death per 100 person-years). As in other populations without access to antiretroviral treatment (15), the risk of death was an order of magnitude greater for HIV-infected men (relative risk, 11.3) and women (relative risk, 9.6). Mortality is greater and is concentrated at older ages in HIV-infected men than in HIV-infected women. These patterns reflect the older male average age at infection, which is caused by a combination of less risky early-age sexual activity and the lower female-to-male than male-to-female HIV transmission probability (16), and results in faster disease progression (17). In addition, the spread of infection may have occurred earlier in men (18).

Within the closed cohort, HIV prevalence increased in younger people (men aged 18 to 26 years and women aged 15 to 23 years at baseline), for whom HIV incidence was high (approximately 3%) and mortality in infected individuals was relatively low. In the older age groups (men aged 35 to 54 years and women aged 30 to 44 years at baseline), HIV prevalence fell, owing to high mortality in infected individuals and moderate levels of HIV incidence.

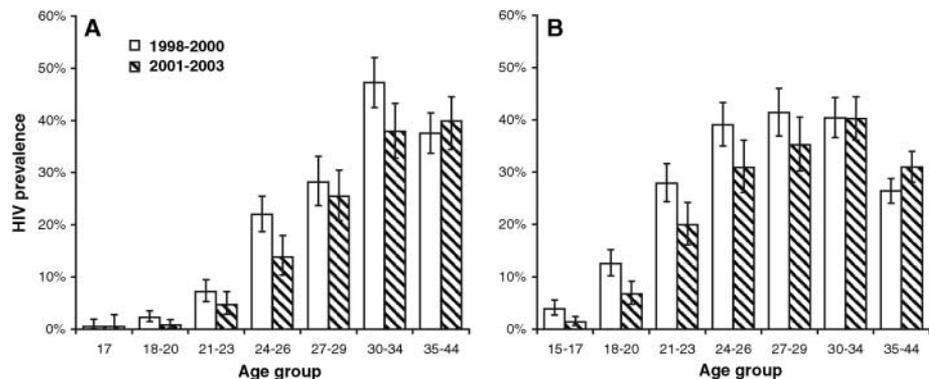


Fig. 1. Change in HIV prevalence by age group over a 3-year intersurvey period from 1998–2000 to 2001–2003, Manicaland, Zimbabwe. (A) Men. (B) Women. Error bars, 95% CIs around sample means.

When the effective reproductive number, R_t (the number of secondary infections arising from each primary infection at time t in an HIV epidemic), is less than 1, an HIV epidemic is in a period of decline (11). The data yielded approximations for R_t of 0.57, 0.73, 0.48, and 0.49 for the town, estate, roadside, and subsistence farming populations, respectively.

For both men and women, the reported number of lifetime sexual partners in the baseline survey was strongly associated with HIV infection (19). In the cohort of sexually active

individuals uninfected at baseline, HIV incidence was higher in those who reported multiple sexual partners during the 3-year intersurvey period than in those who reported a single partner [men: adjusted hazards ratio (AHR), 1.82; 95% CI, 1.17 to 2.85; women: AHR, 3.35; 95% CI, 2.13 to 5.27]. The risk rose progressively with increasing number of sexual partners reported for women (AHR, 1.14; 95% CI, 1.07 to 1.21) but not for men (fig. S2). For men, consistent condom use in casual partnerships lessened the risk of HIV

infection (AHR, 0.38; 95% CI, 0.15 to 0.99). These data, along with the extremely low HIV prevalence in teenagers, the rapid increase in prevalence from age of sexual debut, and an absence of association between recent receipt of medical injections and incident HIV infection (6, 20), indicate that HIV in Zimbabwe is transmitted primarily through heterosexual sex.

We found evidence for a delay in onset of sexual activity among teenage men and women, and for reductions in the proportions of sexually experienced men and women engaging in casual sexual relationships (Fig. 3A, fig. S3, and table S5). At baseline, 45% of 17- to 19-year-old men reported having commenced sexual activity; 3 years later, 27% of the same age group reported having started sexual activity (AHR, 0.55; 95% CI, 0.46 to 0.66). Over the same period, the percentage of 15- to 17-year-old women who reported sexual experience fell from 21% to 9% (AHR, 0.48; 95% CI, 0.36 to 0.63). Among those who had started sex, the proportion of men reporting a recent casual partner fell by 49% (25.9% versus 13.2%; $P < 0.001$); the decrease for women was not statistically significant (7.5% versus 5.9%; $P = 0.292$). Consistent condom use with recent casual partners remained at quite a high level in men (41.6% versus 42.2%) and increased in women (36.5% versus 26.2%; $P = 0.003$). There was no evidence for an increase in consistent condom use with regular partners. For each sex, there were statistically significant ($P < 0.05$) reductions in reported numbers of new sexual partners in the previous year (Fig. 3B), sexual partners in the previous month, and current sexual partners (table S5) (11).

Data on high-risk sexual behavior can be underreported (21), a tendency that may increase over time in an HIV epidemic. Caution is therefore needed in interpreting these results (11). Furthermore, when considering the impact of changes in sexual behavior on an HIV epidemic at the population level, it is important to recognize the contribution of selective AIDS-

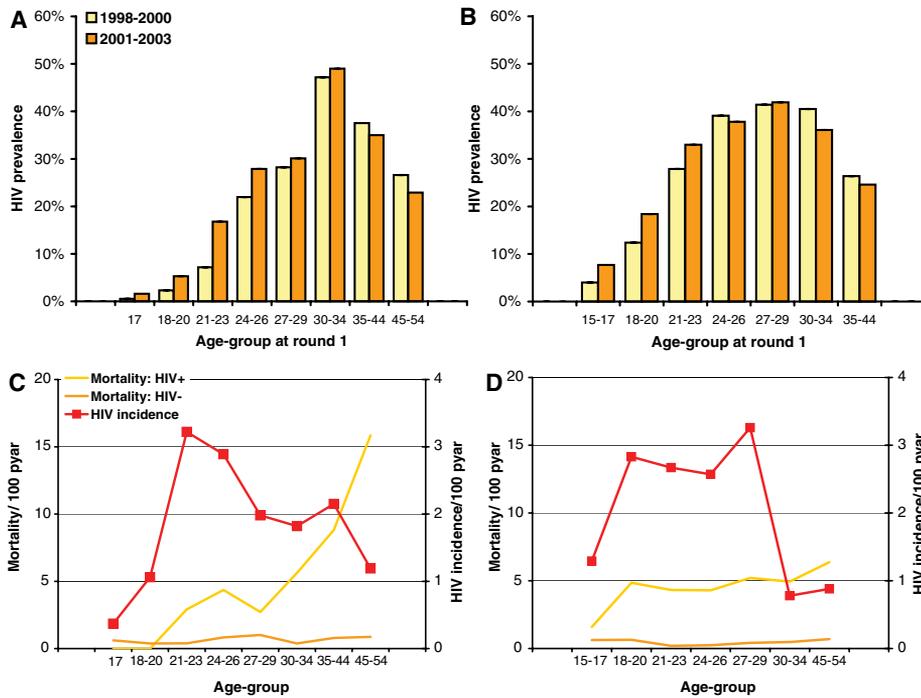


Fig. 2. Components of change in HIV prevalence in members of the closed cohort of 4320 men and 5134 women recruited at baseline, over a 3-year intersurvey period from 1998–2000 to 2001–2003, Manicaland, Zimbabwe. (A and B) Change in HIV prevalence, by age group at baseline interview, for (A) men and (B) women. (C and D) Mortality rates by HIV infection status at baseline (left y axis) and HIV incidence in persons uninfected at baseline and retested at follow-up (right y axis) for (C) men and (D) women.

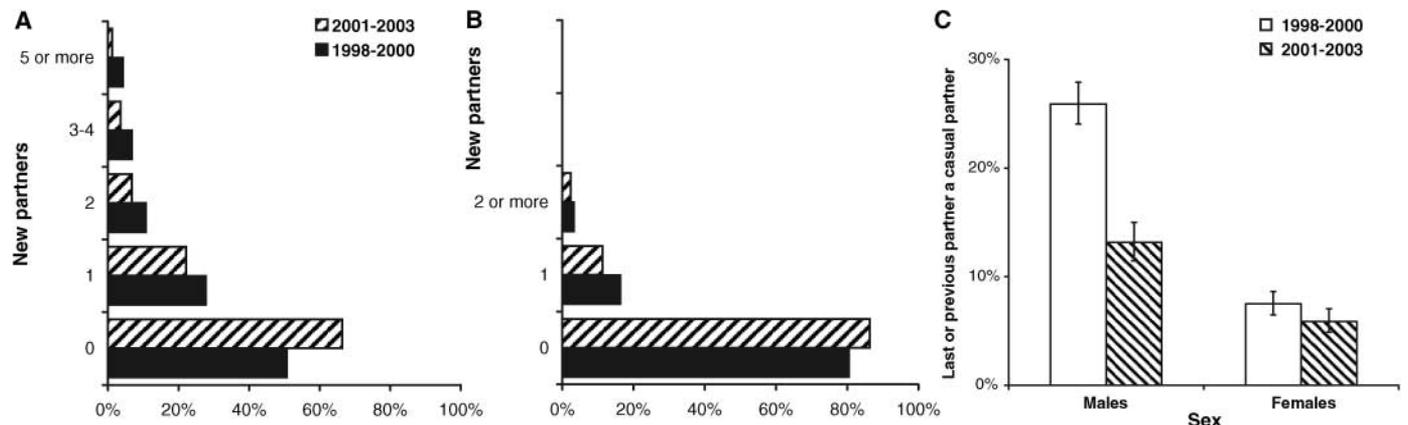


Fig. 3. Comparison of sexual behaviors reported at baseline (1998 to 2000) and follow-up (2001 to 2003) in an open cohort survey, Manicaland, Zimbabwe. (A and B) Distributions of number of new sexual partners in the previous year for (A) men and (B) women. (C) Percentages of men and women (with 95% CI) who reported that at least one of up to two most recent sexual partners in the previous month was a casual partner.

induced mortality (22). Death rates are particularly high in individuals with high numbers of lifetime sexual partners, and their death decreases the variance in reported numbers of sexual partners. This in turn, alters the sexual network through which HIV infection spreads within the population (23), for example, by forcing young women disposed toward forming relationships with older men to choose younger partners or abstain. In the current study, the individuals who died during the intersurvey period had reported higher numbers of lifetime sexual partners at baseline than those who survived to be reinterviewed (men: mean, 13.6 versus 8.7, $P = 0.001$; women: mean, 3.0 versus 2.0, $P < 0.001$). Given the relatively small numbers of individuals who died, AIDS-associated mortality explained only 6.3% of the observed reduction in sexual partner change in men and 8.6% in women, but it cannot be discounted as an important long-term factor.

Selective migration of individuals with high levels of HIV infection could also contribute to changes in HIV prevalence and associated sexual behavior measured at the population level. High numbers of young adults migrated from our study areas to search for employment (11). However, we found no differences in HIV prevalence or sexual risk behavior between migrants and residents at baseline (24). By contrast, there was a lower HIV prevalence among recent migrants into the study areas at follow-up than at baseline (AOR, 0.62; 95% CI, 0.50 to 0.77), which contributed to the observed decline in prevalence (table S3).

HIV prevalence in eastern Zimbabwe is similar to national estimates (25), and the decline occurred equally in areas with and without focused interventions (table S3). Furthermore, the trends in HIV prevalence in women attending antenatal clinics in our study areas mirror those seen nationally in recent surveillance data (11). Thus, these data support the interpretation that HIV prevalence in the general population in Zimbabwe has begun to decline.

Owing to the long average incubation period of HIV infection, HIV prevalence reflects the accumulation of infections over a period of more than 10 years and is insensitive to behavior change. A change in behavior can precede any observed decline in HIV prevalence and would explain the absence of concurrent reductions in HIV incidence in Uganda when HIV prevalence fell during the late 1990s (9). Indeed, behavior change may have been most rapid in Uganda in the early 1990s when HIV prevalence first began to decline (3). If so, Uganda could provide a model for the pattern of change now seen in Zimbabwe. Our data suggest that the changes in behavior occurring in Zimbabwe are similar to those underpinning the long-term decline in HIV prevalence in Uganda, i.e., a delay in age at first sex and a reduction in casual sex (5), but that consistent condom use with casual partners has also contributed.

Given the stage of the HIV epidemic (26), fear of AIDS mortality may have influenced behavior (22). Zimbabwe's well-educated population (27) and good communications and health service infrastructure could have facilitated HIV prevention (13). HIV prevention activities in Zimbabwe (28) have included early control of sexually transmitted infections, social marketing of condoms, voluntary counseling and testing services (26), television and radio serial dramas, and the activities of the Zimbabwe National AIDS Trust Fund (29).

References and Notes

1. E. Asamoah-Odei, J. M. Garcia Calleja, J. T. Boerma, *Lancet* **364**, 35 (2004).
2. G. Asimwe-Okiror, A. A. Opio, J. Musinguzi, G. Tembo, M. Carael, *AIDS* **11**, 1757 (1997).
3. A. Kilian *et al.*, *AIDS* **13**, 391 (1999).
4. J. O. Parkhurst, *Lancet* **360**, 78 (2002).
5. R. L. Stoneburner, D. Low-Beer, *Science* **304**, 714 (2004).
6. D. Gisselquist, R. Rothenberg, J. Potterat, E. Drucker, *Int. J. STD AIDS* **13**, 657 (2002).
7. K. Fylkesnes, Z. Ndhlovu, K. Kasumba, R. Mubanga Musonda, M. Sichone, *AIDS* **12**, 1227 (1998).
8. UNAIDS, "Trends in HIV incidence and prevalence: Natural course of the epidemic or results of behaviour change?" (UNAIDS, Geneva, 1999).

9. M. J. Wawer *et al.*, paper presented at the 12th Annual Retrovirus Conference, Boston, USA, 22–25 February 2005.
10. G. Mwaluko, M. Urassa, R. Isingo, B. Zaba, J. T. Boerma, *AIDS* **17**, 2645 (2003).
11. Methods and supplementary data are available as supporting material on Science Online.
12. R. M. Anderson, R. M. May, *Infectious Diseases of Humans: Dynamics and Control* (Oxford Univ. Press, Oxford, 1991).
13. S. Gregson, H. Waddell, S. K. Chandiwana, *J. Int. Dev.* **13**, 467 (2001).
14. S. Gregson *et al.*, *AIDS* **16**, 643 (2002).
15. D. W. Mulder *et al.*, *Lancet* **343**, 1021 (1994).
16. R. H. Gray *et al.*, *Lancet* **357**, 1149 (2001).
17. D. Morgan *et al.*, *AIDS* **16**, 597 (2002).
18. S. Gregson, G. P. Garnett, *AIDS* **14**, S85 (2000).
19. S. Gregson *et al.*, *Sex. Transm. Infect.* **80**, 36 (2004).
20. B. Lopman, G. P. Garnett, P. R. Mason, S. Gregson, *Public Library of Science* **2**, 142 (2005).
21. J. A. Catania, *J. Sex Res.* **36**, 25 (1999).
22. M.-C. Boily, C. M. Lowndes, S. Gregson, *AIDS* **16**, 2101 (2002).
23. G. P. Garnett, R. M. Anderson, *IMA J. Math. Appl. Med. Biol.* **11**, 161 (1994).
24. C. Mundandi, D. Vissers, H. A. C. M. Voeten, J. D. F. Habbema, S. Gregson, *Trop. Med. Int. Health*, in press.
25. Zimbabwe Ministry of Health and Child Welfare, "Zimbabwe National HIV and AIDS Estimates 2003" (Zimbabwe Ministry of Health and Child Welfare, 2003).
26. J. Decosas, N. S. Padian, *Sex. Transm. Infect.* **78**, S40 (2002).
27. World Bank, *World Development Report* (World Bank, Washington, DC, 2002).
28. A. Mahomva *et al.*, *Sex. Transm. Infect.*, in press.
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Supporting Online Material

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Materials and Methods

SOM Text
Figs. S1 to S3
Tables S1 to S6
References

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Rats Smell in Stereo

Raghav Rajan, James P. Clement, Upinder S. Bhalla*

It has been hypothesized that rats and other mammals can use stereo cues to localize odor sources, but there is limited behavioral evidence to support this hypothesis. We found that rats trained on an odor-localization task can localize odors accurately in one or two sniffs. Bilateral sampling was essential for accurate odor localization, with internasal intensity and timing differences as directional cues. If the stimulus arrived at the correct point of the respiration cycle, internasal timing differences as short as 50 milliseconds sufficed. Neuronal recordings show that bulbar neurons responded differentially to stimuli from the left and stimuli from the right.

Rats use olfactory cues to locate and identify objects in their environment (1, 2). Odor sources can be localized by one of two broad mechanisms (3): sequentially comparing odor concentrations at two different loca-

tions (4) or comparing simultaneous samples from two different locations of the body (5–7). The latter strategy requires separate sampling and parallel neuronal pathways that eventually converge for bilateral comparison. Rat nostrils

are about 3 mm apart and at first sight appear to be too close to each other to support separate sampling. However, studies of respiratory air-flow patterns have shown that air flow is directed laterally to the left and right of the respective nostrils, which suggests that separate sampling of the olfactory environment may occur (8). Further, the two nasal passages are almost completely isolated from each other and supply two distinct sheets of olfactory sensory epithelia. The axonal projections from the sensory epithelium maintain this separation into the first olfactory

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