



Changes in the patterns of sexually transmitted infection among South African mineworkers, associated with the emergence of the HIV/AIDS epidemic

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Objective. To study temporal changes in the relative prevalence of STI syndromes and the aetiology of genital ulcer disease (GUD) among migrant mineworkers in a goldmining area in South Africa during the period 1992 - 2000 and to explore the epidemiological synergy and interactions between these conventional STIs and the emergence of HIV infection.

Method. The records of all STI patients presenting with new episodes of STI seen at a dedicated STI clinic in the Carletonville area, near Johannesburg, between 1992 and 2000 were reviewed and analysed. In addition, cross-sectional studies to determine the aetiology of genital ulcerations were conducted.

Results. During the study period, 36 686 new STI episodes were treated at the clinic with a mean annual STI incidence rate of 137.4 per 1 000 miners. The STI incidence remained high throughout the period 1994 - 1999. A total of 35 789 HIV tests were performed during the study period; the overall HIV prevalence was 35.3%. Between 1986 and 1994, the relative prevalence of genital herpes rapidly increased among GUD patients co-infected with HIV: 0% in 1986, 4.7% in 1990 and 20.8% in 1994.

Conclusions. Syndromic and microbiological surveillance indicates that there was a high incidence of non-herpetic genital ulcerations among miners during the early phase of HIV epidemic. This sustained high incidence of GUDs was followed by a rapid increase in HIV prevalence and changes in the aetiology of the STI syndrome.

As with many other infectious diseases, the epidemics of conventional STIs and HIV/AIDS have largely been driven by social, cultural, economic, behavioural and biological factors. Inevitably, both epidemics triggered intense debate over the issues related to wider socio-economic, racial and gender inequities which have plagued many societies for decades.¹⁻³

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During the early 1990s, a high burden of STIs and HIV infection had already been reported in many countries in sub-Saharan Africa, especially in central Africa. Rapid and sustained spread of HIV followed, and the disease became rampant in many southern African countries during the mid-1990s. By 2002, South Africa had become the country with the largest number of HIV-infected people on the continent.⁴

The southern African epidemic has been characterised by several unique features which could have contributed to its rapid progression. During the 1980s, many countries in the region enjoyed stable economies. In particular, the labour-intensive mining and farming sectors played a crucial role in maintaining South Africa's economy. The creation, in the early 1900s, of the migratory labour system to serve South African mines led to an influx of large numbers of sexually active men without their families, from both within the borders of South Africa and elsewhere in the region, into the mining areas. It is apparent that the STI situation in the early 1990s should have served as an early warning of the subsequent accelerated spread of HIV infection after its introduction into southern Africa during the early 1980s. Unfortunately, major social and political developments such as the transition to democratic rule coincided with the early exponential phase of HIV dissemination.

In this paper, we have endeavoured to describe the temporal changes in the relative prevalence of the various STI syndromes together with changes in the aetiology of sexually acquired GUD that occurred among migrant mineworkers in a goldmining area of South Africa during the period 1992 - 2000. The epidemiological synergy and interactions between conventional STIs and HIV were also explored in these studies.

Patients and methods

Study sites

All studies described here were conducted at dedicated STI clinics providing care for mineworkers from participating goldmines near Carletonville, situated approximately 50 km south-west of Johannesburg. During the study period, these mines employed over 80 000 miners from different regions of southern Africa. There were some fluctuations in the size of the workforce in response to local/international economic situations. However, most employees tended to work for the same company for a significant number of years.

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Study population

All symptomatic STI patients from the participating mines were referred to a dedicated STI clinic situated at a referral hospital or a mine medical station. During this period, the average daily workforce at the participating goldmines ranged from 38 468 in 1992 to 18 300 in 2000; a sharp decline occurred in 1997 as a result of restructuring by the mining companies.

Syndromic STI surveillance

The records of all STI patients presenting with a new episode of STI at the study clinic from 1992 to 2000 were captured in a confidential database. All such patients were offered voluntary counselling and testing for HIV and serological testing for syphilis.

GUD aetiology studies

The causative organisms of GUD were determined by using microbiological surveillance. During the study period, four cross-sectional studies to determine the aetiology of GUD were conducted using the most sensitive and specific laboratory tests available at the time.

During each cross-sectional study, consecutive patients presenting with GUD were enrolled after written informed consent had been obtained. Demographic details and presenting symptoms and signs were recorded by interviewers using standard questionnaires. Blood was collected from all consenting patients for syphilis (RPR and TPHA tests), lymphogranuloma venereum (chlamydial micro-IF), and HIV serological testing. Ulcer scrapings were taken from the edges of the largest ulcer and tested for *Treponema pallidum* and Donovan bodies by darkfield microscopy and Giemsa staining, respectively. Swabs were also taken from the bases of active lesions and cultured for *Haemophilus ducreyi* on selective media and for herpes simplex virus (HSV) in human embryo fibroblast cell cultures.

All patients were subsequently treated using the appropriate syndromic management algorithm for GUD at that time. All HIV-seropositive patients were provided with post-test counselling and referred to local HIV clinics for follow-up and appropriate management.

All study protocols were approved by the Committee for Research on Human Subjects of the University of the Witwatersrand, Johannesburg.

Results

1156 STI incidence (1992 - 2000)

During the study period, a total of 36 686 new STI episodes were treated at the clinic; the mean annual STI incidence rate was 137.4 per 1 000 miners (range: 99.6 - 162/1 000). The STI incidence remained at a high level throughout the period 1994 - 1999 and declined significantly from 157/1 000 in 1999 to 100/1 000 in 2000 ($\chi^2=266.3, p<0.0001$) (see Fig. 1).

STI syndromes such as GUD, urethritis, genital warts and epididymo-orchitis were the most frequent presentations recorded during the period 1992 - 2000, representing 91% of total episodes. The relative prevalence of the major STI syndromes is shown in Fig. 2.

The relative prevalence of GUD, including clinically diagnosed vesicular lesions (genital herpes) and lymphogranuloma venereum (LGV), was significantly higher than that of urethritis during the period 1992 - 1998. An increasing relative prevalence of GUD was detected during the period 1992 - 1994, rising from 50.6% (95% CI 49 - 52.1%) in 1992 to 57.1% (95% CI 55.8 - 58.4%) in 1994 ($\chi^2=38.6, p<0.0001$). Thereafter, a decline in the proportion of cases presenting as GUD occurred.

Meanwhile, there was a steady increase in the relative prevalence and incidence of urethritis, which was the most common presentation among STI patients in 1999 and 2000, representing 44.3% and 43.5% of cases respectively. Similar increases were also found when comparing the relative prevalence of genital warts and epididymo-orchitis. The relative prevalence of genital warts and epididymo-orchitis

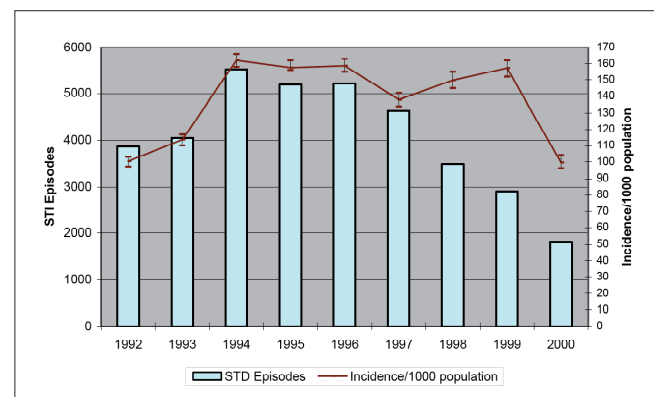


Fig. 1. Annual incidence of STI episodes seen among Carletonville mineworkers (1992 - 2000).

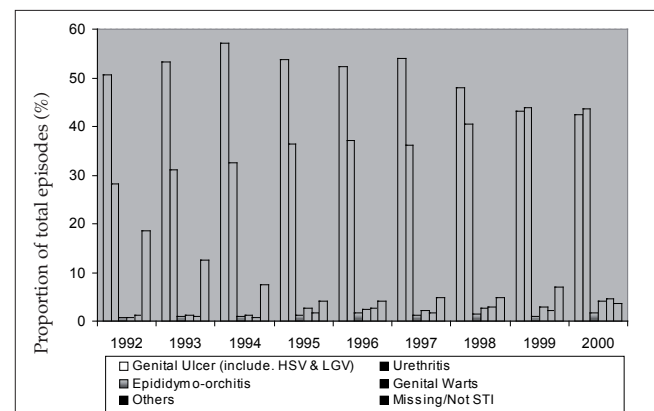


Fig. 2. Relative prevalence of specific STI syndromes diagnosed in Carletonville mine clinics (1992 - 2000).



increased from 0.8% in 1992 to 4% ($\chi^2=70.6, p<0.0001$) and 0.8% to 1.7% ($\chi^2=9.2, p=0.002$) respectively in 2000.

The proportion of GUD cases clinically diagnosed as herpetic increased significantly from 9.1% (of 1 951 GUD episodes) in 1992 to 36% (of 767 GUD episodes) in 2000 ($\chi^2=285.5, p<0.0001$). The relative prevalence of cases clinically diagnosed as LGV ranged between 8.8% in 1993 and 15.3% in 2000. A sharp decline in non-herpetic, non-LGV GUD episodes occurred between 1992 and 2000 (80.5% in 1992 v. 48.8% in 2000, $\chi^2=272.9, p<0.0001$). The majority of these ulcerations were diagnosed clinically as chancroid.

HIV prevalence

During the same period, a total of 35 789 HIV tests were performed; the overall HIV prevalence was 35.3% (95% CI 34.8 - 35.8%). During the period 1992 - 1994, the HIV prevalence was high but stable within the range of 18 - 20.8%. However, it increased significantly in 1995 (20.7% in 1994 v. 42.4% in 1995, $\chi^2=579.6, p<0.0001$), and this trend continued until 1998, when HIV prevalence peaked at 51.6% (95% CI 49.9 - 53.3%). Thereafter, the prevalence declined to 47.3% (95% CI 36.7 - 41.3%) in 2000 (Fig. 3).

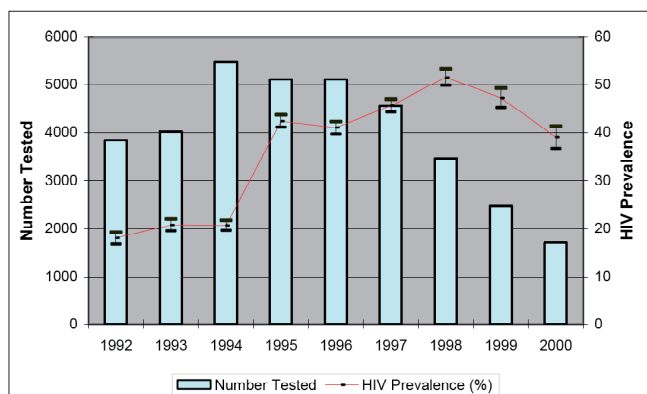


Fig. 3. Number of miners tested and prevalence of HIV, Carletonville (1992 - 2000).

The HIV prevalence rates recorded among STI patients originally recruited in Mozambique and the Eastern Cape were consistently lower than those recorded among patients from Swaziland, KwaZulu-Natal and Botswana. Except for those from Mozambique, the HIV prevalence rate peaked in 1998. HIV prevalence was consistently higher among STI patients from Swaziland, with a peak prevalence of 63.5% (95% CI 57.1 - 69.4%) in 1998. The lowest HIV prevalence was found in STI patients from the Eastern Cape, which peaked in 1998 at 43.8% (95% CI 40.5 - 47.2%).

Changing patterns of STI syndromes (1992 - 2000)

The trend in HIV prevalence among miners presenting with different STI syndromes is shown in Fig. 4. A consistent increase in HIV prevalence among all patients with the various STI syndromes during the period 1992 - 1998 was recorded.

Overall, the HIV prevalence among STI patients increased significantly from 18% in 1992 to 51.6% in 1998 ($\chi^2=918.0, p<0.0001$). HIV prevalence was significantly higher among patients with genital warts (59.8%, 95% CI 56 - 63.4%), followed by those presenting with GUD (40.1%, 95% CI 39.4 - 40.8%) and epididymo-orchitis (36.3%, 95% CI 41.2 - 31.8%). HIV prevalence among urethritis patients was the lowest (29.6%, 95% CI 31.8 - 41.2%), and was significantly lower than that among epididymo-orchitis patients (29.6% v. 36.3%, $\chi^2=8.89, p=0.003$).

The HIV prevalence peaked in 1998, and a steady decline followed during the period 1998 - 2000. When GUD was compared based on clinical diagnosis, patients with non-herpetic genital ulcerations had a higher HIV prevalence rate than those with clinical herpes. However, the overall difference in HIV prevalence rates between the two groups was not statistically significant (41.4% v. 40.5, $\chi^2=0.98, p=0.32$).

During the period 1995 - 2000, 17 659 patients with new STI episodes were screened serologically for syphilis; 1 688 (9.6%, 95% CI 9.1 - 10%) were positive. The annual syphilis seropositivity rate ranged from 11.7% (95% CI 10.3 - 13.1%) in 1995 to 5.2% (95% CI 4.1 - 6.1%) in 2000. The decline was most evident in 1999 and was consistent for patients presenting with all major STI syndromes. There was a slight increase in the syphilis seropositivity rate among genital ulcer patients in 2000, although this increase was not statistically significant ($\chi^2=1.47, p=0.22$).

Changing aetiology of GUD

The results of the cross-sectional aetiological studies of GUD using conventional microbiological techniques are shown in Table I.

The proportion of patients with a positive *H. ducreyi* culture alone declined significantly from 53.1% in 1986 to 38.2% in 1998 ($\chi^2=9.42, p=0.002$). The overall relative prevalence of chancroid (both alone and mixed with other aetiologies) also declined from 68.6% in 1986 to 44.6% in 1998 ($\chi^2=24.7, p<0.001$).

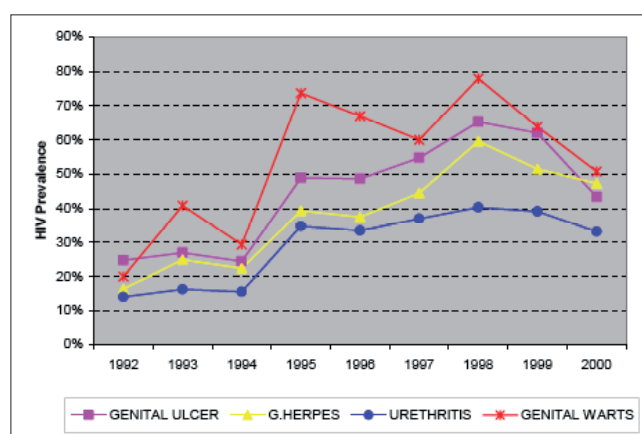


Fig. 4. HIV prevalence versus STI syndrome among mineworkers, Carletonville (1992 - 2000).



Table I. Aetiology of genital ulcer disease, Carletonville (1986 - 1998)

	1986 N = 239		1990 N = 213		1994 N = 232		1998 N = 186	
Single aetiology	167	69.8%	129	60.6%	142	61.2%	120	64.5%
Chancroid*	127	53.1%	107	50.2%	93	40.1%	71	38.2%
LGV†	8	3.3%	2	0.9%	6	2.6%	9	4.5%
Genital herpes‡	3	1.3%	3	1.4%	23	9.9%	31	16.7%
RPR +ve	29	12.1%	17	8.0%	20	8.6%	9	4.8%
Mixed infection	41	17.2%	27	12.7%	28	12.1%	15	8.1%
Chancroid + RPR	28	11.7%	18	8.5%	19	8.2%	10	5.4%
Chancroid + LGV	6	2.5%	3	1.4%	1	0.4%	1	0.5%
Chancroid + LGV + RPR	0		1	0.5%	0		0	
LGV + RPR	2	0.8%	0		1	0.4%	2	1.1%
Herpes + LGV + RPR	0		1	0.5%	0		0	
Herpes + LGV	0		0		0		0	
Herpes + RPR	2	0.8%	0		2	0.9%	1	0.5%
Herpes + chancroid + RPR	1		0		1	0.4%	0	
Herpes + chancroid	2	0.8%	4	1.9%	4	1.7%	1	0.5%
Indeterminate	31	13.0%	57	26.7%	62	26.7%	51	27.4%

**H. ducreyi* culture positive from lesions.

†*C. trachomatis* culture positive from lesions.

‡HSV culture positive from lesions.

LGV = lymphogranuloma venereum; RPR = confirmed rapid plasma reagin test positive.

Similarly, rates of syphilis seropositivity among GUD patients declined steadily from 25.9% in 1986 to 11.8% in 1998 ($\chi^2=13.1$, $p<0.001$). In contrast, the relative prevalence of HSV increased significantly from 3.3% in 1986 to 17.7% in 1998 ($\chi^2=24.9$, $p<0.001$).

There was an increasing prevalence of HIV among patients presenting with GUD during the study period. The HIV prevalence rapidly increased from 1.5% in 1986 to 22.1% in 1990, and 56.0% in 1994 stabilising at 58.8% in 1998.

During the period 1986 - 1994, the relative prevalence of genital herpes rapidly increased among GUD patients with HIV co-infection: 0% in 1986, 4.7% in 1990, and 20.8% in 1994 ($\chi^2=56.6$, $p<0.001$). However, a stable trend of the relative prevalence of HSV was found among GUD patients without HIV co-infection: 3.3% in 1986, 3.9% in 1990, and 2.8% in 1994, respectively ($\chi^2=0.09$, $p=0.96$).

By 1998, the proportion of GUD patients with genital herpes co-infected with HIV stabilised at 19.6%, while a significant increase was found among those without HIV co-infection (2.8% in 1994 to 14.7% in 1998, $\chi^2=8.16$, $p=0.004$). Although the relative prevalence of genital herpes was still higher among GUD patients with HIV infection in 1998, the difference was smaller and not significant (42.1% in 1994 v. 29.6% in 1998, $p=0.09$) as a result of a similar increase in the relative prevalence of genital herpes among HIV-seronegative patients with GUD. The attributable risk of HIV in the increase of HSV as cause of GUD was estimated to be 17% in 1990, 86.5% in 1994, and 25% in 1998.

Discussion

In 1990, HIV prevalence among antenatal clinic attendees in South Africa was less than 1%; this figure increased to 2.2% by 1992.⁵ However, conventional STIs were already a significant public health problem in the region, and sentinel surveillance data indicated the HIV prevalence among STI patients in South Africa to be approximately 6% in the same year.⁶ Similar patterns were observed in neighbouring countries where migrants were recruited to work on South African mines.⁷ The migratory labour system required the majority of mineworkers to live in single-sex hostels, which resulted in a thriving informal sex trade at localities surrounding the mines. The sexual networks among migrant miners, their spouses or regular sexual partners, and casual sexual partners in so-called 'hot-spots' contributed towards a sustained high prevalence of STIs during the 1980s and early 1990s. These sexual networks also fuelled the rapid spread of STIs not only in the mining areas but also at the miners' places of origin.^{8,9} The mobility of these sexually active individuals within the region posed major difficulties in implementing effective STI (and later HIV) control programmes during the early 1990s.

Unfortunately, strategies for the control of the STI epidemic in the mining industry during the 1980s to mid-1990s focused mainly on providing appropriate treatment for symptomatic STIs. This traditional approach to STI control seemed to have had a limited impact on the STI epidemic in sexually active male migrant workers in the mines. As a result, STI incidence among migrant miners in Carletonville remained high during



the mid-1990s, despite significant improvements in patient management.

Despite the rapid introduction of HIV into the population – most notably among high-risk local groups – the general complacency of communities and their respective health authorities delayed a scaling-up of intervention activities. Most countries had either underestimated or ignored early warning indicators derived from STI incidence data and information on existing complex sexual networks associated with the cross-border migratory labour system. As a consequence, most countries in the region failed to take advantage of the window of opportunity to contain the HIV epidemic before it could reach its exponential growth phase. The events leading to the rapid spread of HIV, as documented in our studies among miners, clearly support the existence of epidemiological synergies operating between conventional STIs and HIV. Thus, the high STI incidence among miners, coupled with existing complex social and sexual networks, might have led to the rapid and explosive spread of HIV in the region in the mid-1990s.

Syndromic STI surveillance data indicated that a high incidence of genital ulcerations among miners during the early phase of the HIV epidemic (1992 - 1994) which was followed by a rapid increase in HIV prevalence in 1995.¹⁰ During this early phase, our surveillance systems indicated a high incidence of GUD among miners, largely due to chancroid (and to a lesser extent, syphilis) and that the prevalence of HIV among patients with GUD was almost twice that recorded among patients with urethritis.

Subsequently, the 'accelerated HIV transmission phase' was documented during the mid-to-late 1990s, and continued until the epidemic peaked. During this period, there was exponential growth in HIV in many countries in the region, including among miners with STIs in Carletonville. The relative proportion, as well as absolute incidence of non-vesicular GUD, declined while vesicular ulcerations (clinically suggestive of genital herpes) increased rapidly. This trend was supported by aetiological studies conducted during the period, where the relative prevalence of laboratory-confirmed chancroid declined and genital herpes increased significantly. The relative prevalence of genital herpes among HIV-seropositive GUD patients increased sharply, and genital herpes was highly associated with HIV-seropositivity. Owing to the nature of our cross-sectional study design, we were unable to verify whether this association was due to an increase in the frequency of recurrent herpes induced by HIV-related immunodeficiency or an increase in the acquisition of HIV in patients with genital herpes due to recurrent micro/macro-ulcerations caused by genital herpes. However, our studies indicated that the attributable risk of HIV associated with the increase of genital herpes among miners with GUD could have been over 80% by 1994. HIV immunosuppression is probably

responsible for the disproportionately high number of cases of genital warts associated with HIV-seropositivity, but the nature of the association between HIV infection and epididymo-orchitis remains unclear.

A rapid increase in the relative prevalence of genital herpes among HIV-seronegative patients also occurred in 1998. This could have been due to a rapid decline in bacterial GUD responding to effective intervention activities, and/or an increase in transmission of genital herpes attributable to an increasing pool of infectious individuals associated with HIV infection.

In 1984, syndromic STI management for GUD was implemented in mine clinics, and all patients were treated with effective antibiotic therapy covering both primary syphilis and chancroid. In addition, all patients presenting with any new STI episode were routinely screened for syphilis using a quantitative RPR; treatment for syphilis was provided if they were found to be positive. As a result, the incidence of non-vesicular GUD among miners decreased significantly and similar declines in the relative prevalence of chancroid and syphilis have been documented in aetiological studies conducted in 1994 and subsequently using more sophisticated molecular techniques.^{11,12} Following enhanced syphilis screening and treatment activities initiated on the mines, RPR seropositivity rates among GUD patients also declined steadily, attaining their lowest level in 1998 - 1999.

Many studies have reported that the probability of HIV acquisition and transmission is enhanced in patients with ulcerative STIs.¹³⁻¹⁷ It is also epidemiologically plausible that the high incidence of genital ulcerations in the mines during the early 1990s might have contributed to the rapid and explosive increase in HIV among miners with STIs.

Since 1994, several other intervention activities have been implemented that targeted both high- and low-risk population groups in the community residing in the areas surrounding the mines. These activities included the improvement in quality of STI care (by training and implementation of syndromic management); behavioural interventions including condom promotion;¹⁸ and targeted interventions for high-risk women using periodic presumptive therapy for STIs.¹⁹ A more effective contact tracing system was also implemented in collaboration with a network of private and public STI services situated in areas surrounding mines.²⁰ These interventions, which were largely directed against bacterial (and thus curable) STIs, might have contributed to the slowing of the HIV epidemic. However, the emergence of genital herpes as the major cause of genital ulceration indicates the urgent need to include specific anti-herpes antiviral therapy in revised syndromic management algorithms for GUD.

In summary: our studies contribute towards better understanding of important epidemiological determinants which have fuelled the rapid spread of HIV in southern Africa.



In addition, the findings highlight the utility and usefulness of STI surveillance data in predicting the magnitude of vulnerability to HIV transmission among high-risk population groups. The importance of linking these two surveillance systems should be promoted and applied in tracking the HIV epidemic globally. It is also crucial for individual countries to implement an adequate STI surveillance system as recommended by the World Health Organization.

References

1. Gupta S, Anderson RM, May RM. Networks of sexual contacts: implications for the pattern of spread of HIV. *AIDS* 1989; 3: 807-817.
2. Brandt AM. AIDS in historical perspective: four lessons from the history of sexually transmitted diseases. *Am J Public Health* 1988; 78: 367-371.
3. Aral SO, Holmes KK. Social and behavioral determinants of the epidemiology of STDs: industrialized and developing countries. In: Holmes KK, Sparling PF, Mårdh P, *et al.*, eds. *Sexually Transmitted Diseases*. New York: McGraw-Hill, 1999: 39-76.
4. World Health Organization. *Epidemiological Surveillance Update for the WHO African Region 2002*. Harare, Zimbabwe: WHO Regional Office for Africa, September 2003.
5. Ministry of Health. *Summary Report: National HIV and syphilis seroprevalence survey of women attending public antenatal clinics in South Africa – 2001*. Pretoria: Ministry of Health, 2002.
6. United Nations Joint Programme on AIDS. *Epidemiological Fact Sheets on HIV/AIDS and Sexually Transmitted Infections: 2004 Update*. Geneva: UNAIDS, 2004.
7. World Health Organization. *HIV/AIDS Epidemiological Surveillance Update for the WHO African Region 2002: Country Profiles*. Geneva: WHO, 2003.
8. South African Department of Health. *HIV/AIDS and Migration: A Consultation*. Pretoria: Department of Health/UNAIDS, 1999.
9. Ye H, Ntsekhe P. *Report on Adolescent Sexual Behaviour in Lesotho: 1992*. Maseru, Lesotho: Ministry of Health and Social Welfare, 1992.
10. World Health Organization. *HIV surveillance report for Africa*. Harare, Zimbabwe: Regional Office for Africa (AFRO), November 2001.
11. Chen CY, Ballard RC, Beck-Sague CM, *et al.* Human immunodeficiency virus infection and genital ulcer disease in South Africa: the herpetic connection. *Sex Transm Dis* 2000; 27: 21-29.
12. Lai W, Chen CY, Morse SA, *et al.* Increasing relative prevalence of HSV-2 infection among men with genital ulcers from a mining community in South Africa. *Sex Transm Infect* 2003; 79: 202-207.
13. Simonsen JN, Cameron DW, Gakinya MN, *et al.* Human immunodeficiency virus infection among men with sexually transmitted diseases: experience from a center in Africa. *N Engl J Med* 1988; 319: 274-278.
14. Greenblatt RM, Lukehart SA, Plummer FA, *et al.* Genital ulceration as a risk factor for human immunodeficiency virus infection. *AIDS* 1988; 2: 47-50.
15. Wasserheit JN. Epidemiological synergy: interrelationships between human immunodeficiency virus infection and other sexually transmitted diseases. *Sex Transm Dis* 1992; 19: 61-77.
16. Gray RH, Wawer MJ, Sewankambo NK, *et al.* Relative risks and population attributable fraction of incident HIV associated with symptoms of sexually transmitted diseases and treatable symptomatic sexually transmitted diseases in Rakai District, Uganda. Rakai Project Team. *AIDS* 1999; 13: 2113-2123.
17. Rottingen JA, Cameron DW, Garnett GP. A systematic review of the epidemiologic interactions between classic sexually transmitted diseases and HIV: how much really is known? *Sex Transm Dis* 2001; 28: 579-597.
18. Williams B, MacPhail C, Campbell C, *et al.* The Carletonville-Mothusimpilo Project: limiting transmission of HIV through community-based interventions. *S Afr J Sci* 2000; 96: 351-359.
19. Steen R, Vuylsteke B, DeCoito T, *et al.* Evidence of declining STD prevalence in a South African mining community following a core-group intervention. *Sex Transm Dis* 2000; 27: 1-8.
20. Wright JM, Leong MG, Ye Htun, Rasego B, Ballard RC. *Partner notification for STI patients in a South African mining community – bridging the gap between public and private sectors*. A paper presented at Biennial Meeting of Infectious Diseases and STI Society of Southern Africa. Cape Town, December 2002.