The interrelationship of sexually transmitted diseases and HIV infection: implications for the control of both epidemics in Africa

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Introduction

Sexually transmitted diseases (STD) have been a neglected area in public health in most of the developing world despite overwhelming evidence of their impact on health, particularly that of women and neonates. The emergence of the AIDS epidemic in the 1980s has highlighted both the importance of sexual transmission in the spread of infections and the lack of STD control programmes [1,2]. Furthermore, documentation of the interactions between HIV infection and various other STD is increasingly concerned with both the impact of STD on the sexual transmission of HIV, and the impact of HIV infection on the natural history and response to therapy of STD.

In this paper we will review some epidemiological characteristics of STD in Africa, current knowledge of interactions between HIV and STD, and the impact of these interactions on the HIV and STD epidemic and possible control strategies.

Epidemiology of STD in Africa

The burden of STD, including HIV infection, is high in many parts of Africa although there are major differences within regions and even within countries. As is the case for for HIV, the highest rates for STD are found in urban men and women aged between 15 and 35 years. STD may be responsible for as many as 17% of productive life years lost to disease in sub-Saharan urban populations [3]. In Cameroon, STD were the fifth most common diseases reported by the health services, while, in Zambia, gonorrhoea ranked third after malaria and diarrhoea as a health problem seen in primary health-care centres [4].

Incidence figures of STD in the general population in Africa are scarce, and the few available data are usually based on selected high-risk populations. The monthly incidence of STD among prostitutes in Kinshasa was 10.1, 6.4 and 10% per month for gonorrhoea, chlamydial infection and trichomoniasis, respectively. The incidence of HIV infection among this population was 15% per year [5]. In Rwanda, the annual incidence of gonorrhoea among military personnel before the start of a condom promotion campaign was 22–35% [6], and among male university students the incidence rates of gonorrhoea and syphilis were 30 and 8%, respectively, over a period of 9 months in 1972 [7]. Prevalence figures are more easy to obtain; a selection of results from recent studies among low- and high-risk female populations in Africa is presented in Tables 1 and 2. All studies were conducted in urban areas.

Many factors contribute to these high prevalence rates. The demographic explosion and growing urbanization have resulted in a concentration of young, sexually active people in cities. In many cities, particularly in eastern and southern Africa, men largely outnumber women, and a core group of prostitutes provides a significant proportion of sexual services. Up to 80% of male STD patients in these areas named prostitutes as a source of infection, compared with $<20\%$ in Europe and North America [3]. In other cities, frequent change of partners contributes to the spread of STD [20], as do economic factors, including impoverishment, unemployment and a high dowry. Accessibility to or acceptability of existing health services are often insufficient, particularly for women. Self-medication, inadequate treatment and antimicrobial resis-

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Table 1. Prevalence of sexually transmitted diseases among urban pregnant women in Africa.

Country [reference]	Year	Prevalence (%)				
		Gonorrhoea	Chlamydial infection	Trichomoniasis	Syphilis*	Genital ulcer disease†
The Gambia [8]	1984	7	7	–	17	–
Ghana [9]	1985	3	6	–	–	–
Kenya [10]	1988	7	18	–	4	0.1
Malawi [11]	1991	5	3	32	11	17
Rwanda [12]	1987	5	16	–	–	–
Senegal [13]	1990	2	15	30	7	0.2
Uganda (unpublished)	1991	1	5	43	10	1
Zaire [14]	1990	2	6	17	1	0.1
Zambia [15]	1985	11	–	–	10	–

*Positive syphilis serology defined as rapid plasma reagin/venereal disease research laboratory tests and *Treponema pallidum* haemagglutination/fluorescent treponemal antibody test positive; †genital ulcer disease as a syndrome (no aetiologic diagnosis).

Table 2. Prevalence of sexually transmitted diseases among female prostitutes in Africa.

Country [reference]	Year	Prevalence (%)				
		Gonorrhoea	Chlamydial infection	Trichomoniasis	Syphilis	Genital ulcer disease
Cameroon [16]	1990	–	–	–	38	–
Kenya [17]	1985	50	25	–	32	12
Senegal [18]	1990	16	14	46	30	0.3
Zaire [19]	1988	23	13	28	18	5

tance all contribute to the problem. Health education, health seeking behaviour, and perception of the problem by the authorities are often not optimal. These factors combined result in longer infectious periods and high medical complication rates.

During the past decade, there have been a number of important developments in research into the epidemiology of STD in Africa. Genital chlamydial infection has been shown to be as important in Africa as it is in the industrialized world (Table 1) and, together with gonorrhoea, has been shown to have very serious repercussions on maternal and child health. This is illustrated by the fact that pelvic inflammatory disease (PID) is the most common cause of admission in gynaecology wards in Africa [21]. In Kenya, 44% of women with postpartum PID had gonococcal or chlamydial cervicitis [22]. PID itself may lead to chronic abdominal pain, ectopic pregnancy or infertility [21]. The rate of ectopic pregnancy in Africa is up to three times that found in industrialized countries [23] and remains an important cause of maternal mortality in developing countries, especially in rural areas where easy access to critical care facilities is often lacking. A recent study in Zimbabwe showed an association between ectopic pregnancy and a history of PID, and high titres of both antigonococcal and antichlamydial antibodies. A similar association was also demonstrated for infertility [24].

Despite high rates of population growth in most African countries, in some regions more than 20% of women are involuntarily childless [25]. A worldwide World Health Organization (WHO) study suggested that, in contrast to other continents, the pattern of infertility in Africa was strongly related to PID, again suggesting that STD play an important role in the aetiology of infertility [26]. Prematurity and stillbirth are frequently caused by STD, while congenital syphilis, ophthalmia neonatorum and chlamydial neonatal pneumonia remain frequent causes of infant morbidity in Africa [10,27,28].

The emergence of antimicrobial resistance has become such a problem in the treatment of gonorrhoea and chancroid that more expensive, and in many regions unavailable, treatment regimens have to be recommended [29].

Genital ulcer disease (GUD) is also more frequent in Africa than in developed countries, *Haemophilus ducreyi* being the most common identifiable cause [30]. As many as 20–70% of patients seen in STD clinics in Africa may present with a genital ulcer [31]. The high proportion of GUD amongst STD seen at clinical services may, however, be overrepresentative because of the difference in behaviour when seeking health care for different STD. Data on the prevalence and incidence rates of GUD in different populations are scarce. In Kinshasa, the prevalence of GUD

among 1233 prostitutes was 5% while the monthly incidence was <1% [5]. In Kenya, the prevalence of GUD among female prostitutes was 12% [17]. Prevalence rates of GUD among 'low-risk' populations such as pregnant women, if available, are usually very low (<1%; Table 1). There seem to be marked geographical variations in rates of GUD (and, more specifically, syphilis and chancroid), with much higher prevalence and incidence rates in eastern and southern Africa compared with Central and West Africa. The reason for this discrepancy is not known, but recent evidence suggests that HIV infection has an impact on the incidence and response to treatment of GUD (see below). Populations with very high prevalence of HIV would therefore also have a high prevalence and incidence of GUD.

The seroprevalence rates of HIV infection (see other reviews in this volume) are generally high in urban Africa, but also show a very scattered pattern, with important differences from region to region. The reasons for this are not yet clear but are probably many and due to demographic, economic, behavioural and biological factors, one of the most important being the STD epidemic.

Interactions between HIV and other STD

Since the beginning of the AIDS epidemic, researchers have been struck by the high rates of current STD or a history of STD among HIV-infected individuals as well as by the high prevalence of HIV infection among STD patients. In search of explanations for the rampant HIV epidemic among heterosexuals in Africa, it was decided that STD could facilitate the transmission of HIV. Since then, it has become clear that the relationship between HIV and other STD is much more complex, and several means of interaction have been postulated (Fig. 1). These include: (1) the presence of STD enhances the transmission of HIV; (2) HIV infection and consequent immunodeficiency alter the natural history, diagnosis and response to treatment of other STD; (3) STD may influence the natural history of HIV, for example, by accelerating progression to clinical disease.

This paper will examine the available data on the first two hypotheses, which are the best studied and most important from a public health point of view.

When analysing studies on relationship between STD and HIV, several methodological problems must be considered. First, such studies are complicated by the fact that HIV infection is itself an STD. STD and HIV are transmitted in the same way and share the same behavioural risk factors (such as multiple sexual partners, prostitute contact, being single and/or young); thus, the presence of other STD could merely be a marker for a high-risk behaviour rather than a causal link in HIV transmission. Analyses which control for this confounder (sexual behaviour) are therefore essential in establishing whether STD are independent risk factors for HIV transmission.

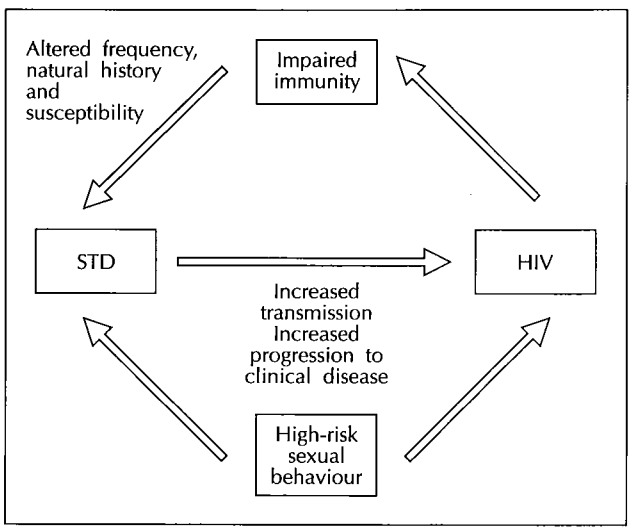

Fig. 1. Interactions between sexually transmitted diseases (STD) and HIV.

A second problem arises from the potential impact of HIV infection and related immunodeficiency on other STD, such as increased frequency of genital herpes lesions in HIV-infected people. Without prospective studies documenting the temporal sequence, it is impossible to determine whether a higher rate of STD among HIV-infected individuals indicates that STD facilitate HIV transmission or whether STD are simply markers for HIV-related immunosuppression.

As discussed by Cameron and Padian [32], even prospective studies may not establish a temporal sequence of events with certainty because of the varying delay between HIV infection and development of antibodies. However, community-based intervention trials, considered by some to be the only design which can provide proof of the enhancing effect of STD on HIV transmission, are extremely complex (i.e. in terms of identifying the right representative study population with high HIV and STD incidence, defining and monitoring the STD interventions, avoiding contamination of the control group, ethical issues), time-consuming and expensive [33], and may never yield definitive conclusions. Meanwhile, HIV is spreading rapidly in Africa, and the need for intervention on potentially modifiable risk factors for the sexual transmission of HIV is urgent.

The impact of STD on HIV transmission

Several authors have critically reviewed the data on the impact of STD on HIV transmission [34–37]. Here, we summarize the main studies which contributed to the

epidemiologic evidence that STD enhance HIV transmission.

Two questions should be asked when studying the impact of STD on HIV transmission: (1) whether among people not infected with HIV, there are some STD that increase susceptibility to HIV infection, and (2) whether among HIV-infected individuals, any STD enhance their infectivity, increasing the probability of transmission of HIV to their sexual partners.

At present, the biological variables which determine both infectivity of and susceptibility to sexual transmission of HIV infection are poorly understood, which reflects in part the absence of good quantitative measurements of infectious virus levels in blood, semen and cervical secretions. However, the role of STD in increasing the risk of HIV transmission is biologically plausible because (1) STD can cause disruption of the normal epithelial barrier (in the case of genital ulcers) or micro-ulcerations in the mucosa (in the case of gonococcal or chlamydial cervicitis or trichomonal vaginitis) and (2) STD can increase the pool of lymphocytes and macrophages (HIV-susceptible or HIV-infected cells) in the genital tract and therefore increase infectivity in a seropositive individual and susceptibility in a seronegative individual.

GUD and HIV transmission

Numerous studies have linked GUD, either as a clinical syndrome or as an aetiological diagnosis, such as syphilis or genital herpes, to an increased risk of HIV infection. However, the majority of studies documenting a higher rate of HIV infection among people with a genital ulcer or a higher rate of genital ulcers among HIV-infected individuals are cross-sectional. This makes it impossible to determine whether the ulcer was acquired prior to or after HIV infection.

A few prospective studies showing an association between genital ulcer and HIV seroconversion are summarized in Table 3. The most convincing evidence that GUD does enhance HIV transmission comes from a study by Cameron *et al.* [38] in Nairobi. This group showed, in a prospective study of the clients of female prostitutes, that acquisition of a genital ulcer (the majority being chancroid) was a highly significant risk factor for subsequent HIV seroconversion. This led to the hypothesis that GUD in the woman enhances transmission to the male partner. GUD disease was also shown to be a risk factor for HIV seroconversion in a prospective study of these women [39]. Prospective cohort studies in homosexual men in the United States documented a strong association between herpes simplex virus (HSV)-2 seroconversion, a history of syphilis and a serologic diagnosis of syphilis and HIV seroconversion [40,41].

Table 3. Selected prospective studies documenting sexually transmitted diseases (STD) as risk factors for HIV transmission.

Study population [reference]	STD studied	Relative risk*
Heterosexual men, Kenya [38]	Genital ulcer† (clinical diagnosis)	4.7
Homosexual men, USA [41]	Syphilis (serology self-reported)	1.5–2.2
Homosexual men, USA [40]	Herpes (serology)	4.4
Heterosexual women, Kenya [39]	Genital ulcer* (clinical diagnosis)	3.3
	Chlamydial infection (culture)	2.7
Heterosexual women, Zaire [44]	Gonorrhoea (culture)	3.5
	Chlamydial infection (chylamydiazyme)	3.2
	Trichomoniasis (direct examination)	2.7

*Relative risk adjusted after multivariate analysis. †The majority of genital ulcer disease in Kenya and Zaire was chancroid, confirmed by culture for *Haemophilus ducreyi*.

So far, two studies, although cross-sectional, have found an association between HIV-2 and GUD [42,43].

Non-ulcerative STD and HIV transmission

Data on non-ulcerative STD and HIV infection are far more limited than those on GUD, but evidence that these syndromes do facilitate HIV transmission has emerged recently. In the above mentioned prospective cohort of 124 female prostitutes in Nairobi [39], *Chlamydia trachomatis* infection was independently associated with HIV seroconversion. In Kinshasa, Zaire, 450 initially HIV-negative female prostitutes were followed-up for 2 years; 55 women seroconverted during follow-up. The 4-month period prior to seroconversion in these women was compared with a concurrent period in persistently HIV-negative women for incidence of STD and sexual exposure in a nested case–control analysis. Chlamydial infection, gonorrhoea and trichomoniasis were significantly associated with HIV seroconversion, with odds ratios of 3.2, 3.5 and 2.7, respectively. This persisted after controlling for self-reported number of partners and condom use [44].

The impact of HIV infection on other STD

HIV infection and its effect on the host immune response could theoretically affect other STD in a number of ways. HIV infection may alter the incidence or frequency of STD recurrences; it might also modify the

clinical expression and natural history of STD, resulting in more frequent and rapid development of complications, altered performance of laboratory tests and inadequate response to standard therapy.

The need for an HIV-negative control group is a critical factor which has often been ignored in reports assessing the impact of HIV infection on other STD. Our overall understanding of the natural history of some STD [such as syphilis or human papilloma virus (HPV) infection] in the absence of HIV infection is still incomplete. In this context, case reports or case series on altered natural history of STD in HIV-infected individuals may be difficult to interpret. Available data on the impact of HIV on other STD are summarized in Table 4.

The impact of HIV infection on GUD

Evidence is now emerging that the frequency of GUD is higher among HIV-infected than HIV-negative individuals with the same sexual exposure. This can be explained by either an increased number of recurrences of HSV-2, prolonged duration of the lesions and/or because of increased susceptibility to genital ulcers caused by *Haemophilus ducreyi* or other organisms. In a cohort study of 490 HIV-negative and 150 HIV-positive female prostitutes in Zaire, the incidence of GUD was three times higher in the HIV-positive women [45]. This could largely be explained by a higher rate of HSV-2 recurrences, though chancroid was also more common in HIV-positive women.

Decreased responsiveness to standard therapy for chancroid was documented in Nairobi, where treatment failure was at least six times more common in HIV-positive patients [46]. More recently, a 25% bacteriological failure rate was observed in the same clinic after treatment with 250 mg ceftriaxone, previously a highly effective drug for chancroid [47]. Poor clinical and bacteriological responses correlated with being HIV-positive and uncircumcised.

Large, chronic and persistent herpetic ulcerations are a frequent complication of HIV-infected individuals with advanced immunodeficiency and a number of reports suggest an increased incidence of HSV infection resistant to acyclovir in HIV-infected patients [48,49].

The impact of HIV infection on syphilis has been particularly worrying. Numerous case reports have suggested that in the context of HIV infection the clinical presentation of syphilis may be atypical, progression to neurosyphilis more frequent, serologic tests false-negative or false-positive and standard therapy for early infection inadequate [50]. Most of these studies should be interpreted with caution because they lack a comparison group of HIV-negative patients and the sample sizes are often very small. It should also be stressed that knowledge of the natural history, serologic diagnosis and response to treatment of syphilis in the absence of HIV infection is still incomplete.

Lukehart *et al.* [51] compared 15 HIV-positive and 25 HIV-negative patients with untreated primary or secondary syphilis in Seattle. *Treponema pallidum* was isolated from cerebrospinal fluid and, surprisingly, the rates for HIV-positive and HIV-negative patients were similar (27 compared with 32%), suggesting that central nervous system involvement may be more common in early syphilis than previously thought. However, in the same study, three HIV-infected patients with early syphilis were treated with benzathine penicillin, which failed to eradicate *T. pallidum* from the cerebrospinal fluid. It is not yet clear whether this was the result of the relative failure of standard treatment for primary syphilis for all patients (HIV-positive and negative), or whether this was an effect of HIV-induced

Table 4. Possible impact of HIV infection on the presentation, natural history, diagnosis and therapy of sexually transmitted diseases (STD).

STD	Clinical presentation	Natural history	Performance of standard laboratory tests	Response to therapy
Syphilis	?	More persistent lesions More central neurologic involement	Decreased sensitivity in serologic tests	Increased risk of treatment failure
Herpes	Larger lesions, atypical sites, longer duration	Increased incidence (or recurrence)		Increased incidence of acyclovir resistance
Chancroid	? Larger lesions	More persistent lesions Increased incidence		Increased failure with single-dose regimens
Genital warts HPV infection	Larger lesions	Increased incidence Increased progression to dysplasia/ neoplasia	Increased viral load Increased HPV detection	Decreased response to topical or laser therapy
Gonorrhoea		? Increased incidence of gonococcal pelvic inflammatory disease		

HPV, human papilloma virus.

immunodeficiency. In a study among Kinshasa prostitutes, we also found no significant difference between 40 HIV-positive and 50 HIV-negative women in terms of the initial rapid plasma reagin (RPR) titres or of the change in RPR titre in the year following therapy [52]. Studies with larger numbers of syphilis cases in HIV-infected individuals with a valid control group of HIV-negative patients, and with facilities for both rabbit inoculation to isolate *T. pallidum* and comprehensive clinical evaluations, will be necessary to elucidate the very complex problem of syphilis in HIV infection.

The impact of HIV infection on non-ulcerative STD

The best documented evidence of the effect of HIV infection on non-ulcerative STD is its impact on genital warts and HPV infection. Although genital warts could also facilitate HIV transmission through traumatization, it seems more plausible (taking into account the pathogenesis of this infection) that warts are promoted by HIV-related immunosuppression. In a cohort study of Kinshasa prostitutes, HIV-positive women had a significantly higher incidence of genital warts compared with HIV-negative controls [45]. One of the most alarming effects of HIV infection is its apparent ability to facilitate the development of anogenital dysplasia. Studies incorporating HIV-negative comparison groups have consistently demonstrated a significant association between HIV infection, a higher rate of HPV detection and cytological or histopathological abnormalities in cervical or anal specimens [53–55]. Longitudinal studies are now needed to clarify this issue; cervical and anal cancer may emerge as important opportunistic complications in HIV-positive patients.

There are currently no data to suggest that HIV infection alters the clinical presentation, diagnosis or response to treatment of lower tract gonococcal or chlamydial infections, or trichomoniasis. Whether the risk of complications of gonococcal or chlamydial disease is increased in the presence of HIV is not yet clear. Wambugu *et al.* [56] found that HIV-positive women in Nairobi had a higher incidence of clinically diagnosed gonococcal pelvic inflammatory disease than HIV-negative women [56]. However, in Kinshasa, the incidence of PID (with or without gonorrhoea or chlamydial infection) was not different in HIV-positive and HIV-negative women [45].

The impact of the HIV–STD interactions on the HIV and STD epidemics

Although the 'perfect' epidemiologic study has not yet been performed (and may never be), several prospective studies that controlled for behavioural risk factors now strongly suggest the hypothesis that STD, both ulcerative and non-ulcerative, facilitate the transmission of HIV. It has also become clear that HIV infection can have a considerable impact on the clinical spectrum and management of STD. The STD epidemic has therefore severely aggravated the HIV epidemic in many parts of the world, while the HIV epidemic confronts us with additional obstacles in the fight against STD.

How large the attributable risk of STD is in the spread of HIV in Africa depends on several factors, such as the relative risk of each STD in increasing the transmission, the prevalence of the different STD in the community and the relative occurrence of 'competing' risk factors for transmission (such as sexual behaviour or lack of circumcision). STD can be up to 20 times more prevalent among female prostitutes than in the general population (see Tables 1 and 2) and non-ulcerative STD are far more common than genital ulcers in most areas of Africa. The difference in attributable risk between prostitutes and members of the general population, and between ulcerative and non-ulcerative STD, has important implications for the design of HIV control programmes [57].

In a cohort study among female prostitutes in Kinshasa the prevalence of GUD was 5%, and out of 55 women who seroconverted during a 2-year period, only four had an episode of GUD prior to seroconversion, while 71% had gonorrhoea, chlamydial or trichomonal infection. Prevention and control of GUD would have prevented not more than 7% of the seroconversions in this population. Attributable risks were 32, 23 and 12% for gonorrhoea, trichomoniasis and chlamydial infection, respectively, in this population. However, there are areas in eastern and southern Africa where GUD is highly prevalent and where the attributable risk of GUD in the total number of HIV infections may be significant. Attributable risks for GUD in Nairobi prostitutes were estimated to be around 33%, while the estimates for the general population were very low (<5%) [57].

Possibly one of the most critical factors in the rapid spread of HIV through sexual transmission in Africa is the establishment of a sub-group of efficient, high-frequency HIV transmitters: prostitutes and their clients, the military and truck drivers. Urban female prostitutes with very high rates of STD and a high number of different partners were initially a very susceptible group, as indicated by very high rates of HIV infection. After the introduction of HIV in this population, the prostitutes become a highly infectious group for their clients. Although overemphasis on prostitution as an explanation for the spread of HIV is an inadequate portrayal of urban sexual behaviour in such areas of Africa, it is striking that countries with a typical 'core group sexual pattern' (a large group of men having sex with a small number of women) have had the highest incidence rates of HIV (for example, Rwanda and Malawi).

HIV-induced immunosuppression may increase the susceptibility of individuals to certain STD, as suggested by cohort studies of prostitutes in Kinshasa. Analysis has shown that HIV infection increased the rate of recurrence and/or susceptibility to GUD and genital warts, especially when immunity was compromised. On the other hand, HIV infection seemed to have no influence on the recurrence of or susceptibility to gonorrhoea, chlamydial infection, trichomoniasis and PID. These interactions may in fact have some influence on the STD epidemic in Africa. The prevalence of GUD, such as chancroid and herpes, may increase, especially in high-risk populations with high HIV seroprevalence, creating an opportunity for increased HIV transmission.

The synergistic interactions between STD and HIV, which mutually reinforce each other, may severely aggravate both the HIV and the STD epidemic (Fig. 1). STD will enhance HIV transmission, resulting in a larger pool of infections; the consequent HIV-related immunodeficiency in this population will then result in a higher burden of some STD (such as GUD, through increased susceptibility and treatment failures and/or more frequent complications, such as dysplasia) [37].

Implications for STD and HIV control programmes

HIV–STD interactions have led to the development of strategies for the coordination of synergistic AIDS and STD control programmes [58]. Since the major modes of transmission of HIV and STD are identical, and behavioural risk factors are shared, similar target populations and behavioural interventions are beneficial for the control of both epidemics. Promotion of condoms is likely to reduce the incidence of HIV directly, and indirectly through a reduction of incidence of STD (see Fig. 2). STD diagnosis and treatment decreases duration of infection, which would eventually also result in a decrease of HIV incidence. The relative impact of both interventions in controlling the HIV epidemic is still unknown, but it seems plausible that, in high-risk groups with high STD prevalence rates, STD treatment may play an important role. In an intervention programme focusing on STD control and condom promotion in Kinshasa, the incidence of both HIV and STD was drastically reduced after 2 years [59].

STD prevention and control have now become an integral part of HIV control programmes in most of Africa, but STD services should be strengthened and even established in some areas. Programmes should focus on the full spectrum of locally prevalent STD rather than exclusively on genital ulcers. Although both AIDS and STD programmes can benefit enormously from coordination and integration, certain specific considerations of both programmes should not be neglected [60]. The importance of early diagnosis and treatment as a control strategy in STD is one of the major current differences in approach between STD and AIDS, where primary prevention is essential. In addition, specific interventions, such as control of congenital syphilis through screening of pregnant women and eye prophylaxis at birth should not be neglected in view of their high cost-effectiveness and cost-benefit [3]. Equally, care of people with AIDS also requires a specific approach.

In areas with high rates of HIV infection, STD control strategies may have to be revised. New effective therapeutic regimens for chancroid and syphilis may have to be determined, and screening programmes for cervical dysplasia established.

Although the programme and policy implications of these HIV–STD interactions are clearcut and urgent, many research questions are still unresolved: what will the ultimate impact of reducing levels of STD in different populations be on the incidence of HIV? Which strategy of STD control should be recommended (screening, improving care of STD patients, mass treatment)? And, probably most crucially, what will the impact of focused intervention in core groups be on the incidence of HIV in the general population? It is crucial that carefully monitored interventions be conducted to answer these questions.

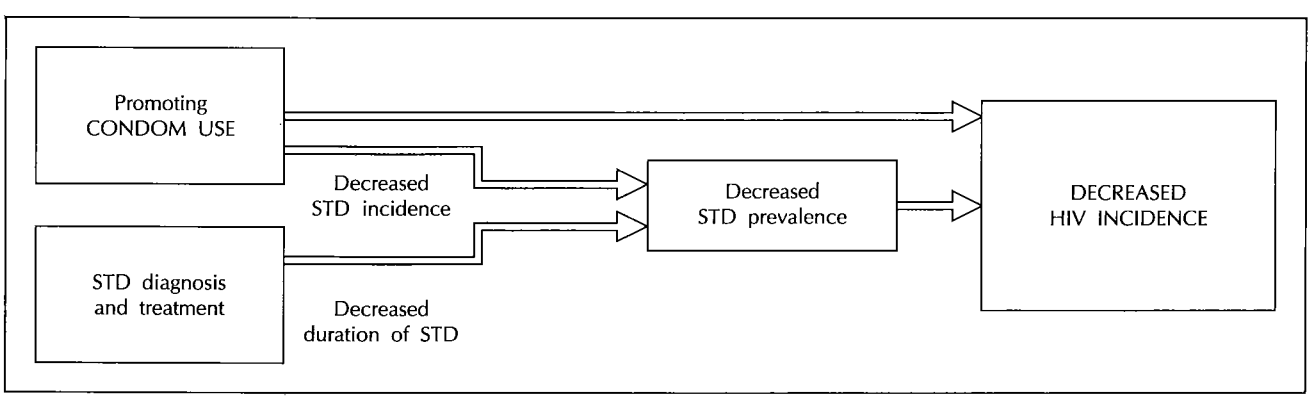

Fig. 2. Interrelationships between condom use, sexually transmitted diseases (STD) incidence and incidence of HIV.

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