ASSESSMENT OF HIV AMONG STUDENTS OF HIGHSCHOOLS AND COLLEGES ATTENDING CLINICS FOR SEXUALLY TRANSMITTED DISEASES IN ADDIS ABABA

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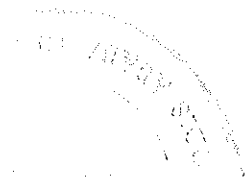

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ABSTRACT

To assess the prevalence of the Human Immunodeficiency Virus (HIV) infection, the risk factors involved and the association between HIV and Sexually Transmitted Diseases (STDs), a cross - sectional study was conducted among highschool and college students attending three clinics for STDs in Addis Ababa. Out of 324 samples studied, 63(19%) were positive for antibodies to HIV by Enzyme - linked immunosorbent assay and Western Blot analysis. Among 214 students with STDs, 48(22%) were positive for HIV antibodies. The seroprevalence rate for male students was 19% (95% confidence interval, 14 to 26) and for the female students 22% (95% confidence interval, 18 to 36). In addition, among 110 students without STD manifestations (controls), 15(14%) were positive for HIV antibodies. Among these, the seroprevalence rate for male students was 7% (95% confidence interval 1 to 13) and for the female students, 25% (95% confidence interval, 12 to 38). In both STD and non - STD smaples, highschool students had a higher rate of HIV seropositivity than college students. Students with STDs were more likely to be HIV seropositive than those without STD manifestations. A current diagnosis of syphilis was associated with HIV seropositivity among the total population of students (odds ratio, 2.6; $P < 0.00003$); among the STD samples (odds ratio, 2.2; $P < 0.009$); among total female students with STDs (odds ratio,

6.93; P < 0.002) and among total highschool students with STDs (odds ratio, 2.25; P < 0.01). A current diagnosis of chancroid was associated with HIV seropositivity among the total population of students (odds ratio, 2.0; P < 0.01) and among male students with STD cases (odds ratio, 2.38; P < 0.05). Multipartner sexual contacts, lack of condom use, past STD history and current STD cases were found to be the risk factors for both syphilis and HIV significantly, indicating that they are likely to be important confounders and also indicating that the association between HIV and syphilis can be influenced by these factors. The study finds that HIV infection is present in highschools and colleges in Addis Ababa at high rates and STDs usch as syphilis and chancroid are associated with HIV transmission among the students.

INTRODUCTION

The Human Immunodeficiency Virus (HIV) is a member of the lentivirus family and is identified and characterized as the causative agent of the Acquired Immune Deficiency Syndrome, AIDS (Barre-Sinoussi et al., 1983). AIDS is a group of disease proceses secondary to a defect of the immune system due to HIV infection in persons with CD_4 cell counts at or below 200 cells per cubic millimeter of blood (Blattner,1991).HIV is transmitted by sexual intercourse.Exposure to blood and blood products and perinatally from infected mother to new borns are the other confirmed modes of transmission.

There are two distinct types of HIV, HIV-1 and HIV-2. The former is the known etiologic agent responsible for the worldwide pandemic of AIDS. The second type clusters prominently in West Africa. Although associated with AIDS, HIV-2 appears to be less virulent in its effect (Kong et al.,1988). Structurally the HIV can be divided in to two major compartements, the outer lipid bilayer membrane which is covered by two envelope glycoproteins, gp120 and gp41 and the core proteins designated as p24, p17, p9 and p7. The p24 polypipetide forms the chief component of the inner shell, where as the p17 protein is associated with the inner surface of the lipid bilayer. The p7 protein binds directly to the genomic RNA and together with p9 forms the nucleoid core. This retroviral core also contains two copies of the single stranded HIV-1 genomic

major cellular targets for HIV-1 replication (Fauci, 1988), because they possess the principal high affinity cellular receptors for this retrovirus (Dalgleish et al., 1984). This allows the interaction of gp120 with the CD4 membrane receptor after which gp41 - mediated membrane fusion occurs leading to the entry of HIV-1 into the cell. HIV-1 virions are brought inside the cell by either classic receptor mediated endocytosis (Maddon et al., 1986) or virus mediated membrane fusion (Stein et al., 1987). After internalization, the HIV-1 virion is rapidly uncoated in preparation for the replicative phase of its life cycle. Replication begins with the transcription of viral RNA by the enzyme reverse transcriptase, resulting in the production of double stranded DNA. In turn the HIV-1 integrase promotes the insertion of this viral DNA duplex into the host genome, thereby giving rise to the HIV-1 provirus. Absence of host factors such as specific or nonspecific mitogenic stimulation of CD4+ T cell population, may result in either absence of viral replication (Cullen and Greene, 1989; Haseltine, 1991) or cause viral latency (Zack et al., 1990). Cellular activation and host transcription factors lead to the sequential production of various mRNAs. The first mRNAs produced encode the HIV-1 regulatory proteins (Kim et al., 1989). Subsequently the viral structural proteins are produced allowing the assembly and morphogenesis of virions. During the late stages of virion morphogenesis the efficient release of the budding virions is promoted by the viral protein product from the surface of the cell (Klimkait et al., 1990) which can then reinitiate the

retroviral life cycle by infecting other CD4+ target cells.

In general, it has been suggested that AIDS affects predominantly two age groups: adults of 20-40 years and infants and very young children. Throughout the world 75-90% of HIV infections and AIDS cases are occurring in the age group of 20-40 years (Sepulveda, 1988). Retroviruses generally have long incubation periods between the time of infection and the development of symptoms. Persistent antibodies to HIV normally develop within three month - however, delayed conversion has been reported in sexually transmitted HIV infection (Ranki et al., 1987).
Accute infection with HIV has been characterized as a mononucleosis - like syndrome with symptoms appearing 2-6 weeks after laboratory seroconversion. Symptoms may include maculopapular rash, fever, myalgia, arthralgia, headache, diarrhea and sore throat, and also neurological manifestations. One study suggests that 50-90 percent of adults may experience the acute syndrome at seroconversion (Tindal et al., 1988).

The mean incubation period of HIV following infection from a blood transfusion has been estimated at 8.23 years for adults and 1.97 years for children under five years old (Medley et al., 1987). A study in homosexual men has estimated the mean incubation period of AIDS to be 7.8 years (Curran et al., 1988).In hemophiliac and homosexual cohorts there was no progression in the first year after seroconversion, 0-2% progression at 2 years, 5-10% progression at

4 years, 10-25% progression at 6 years, and 30-40% progression at 8 years and 48% progression was observed at 10 years (Hessol et al., 1988). Estimates from empirical and theoretical studies cluster the median time range from infection to disease to be between 7-14 years (Polk et al., 1987; Kalbfleisch and Lawless, 1988; PAHO, 1989). Opportunistic infections, that is, invasions of microorganisms that proliferate widely only because the immune system is defective, accounted in the past for 50 to 70 percent of the deaths in patients with AIDS in the United States (Mills and Masur, 1990). The first infections are relatively benign but annoying infections of the skin and mucous membranes including Candida albicans that causes sores of the mouth, Vorticella - Zoster Virus that causes Shingles and infections of nerves and skin, several types of fungi that cause severe athlete's foot and Epstein-Barr Virus (EBV) that cause oral hairy leukoplakia. Once these appear, the person is said to have the AIDS related complex (ARC). With further breakdown of immunity, serious AIDS - defining infections of Pneumocystis carinii pneumonia (Small et al., 1983), cryptococcal Meningitis and toxoplasmosis (Fauci et al., 1984) arise as the three major killers. Today some of the opportunistic infections can be prevented by medication.

The first immunologic abnormality often seen in infected individuals is B - cell proliferation and elevated levels of immunoglobulin which results in hypergammaglobulinaemia (Benveniste et al., 1983). This is due to an effect on T lymphocytes. The

predominant classes of immunoglobulins found to be elevated have been serum IgG and IgA (Seligman et al., 1984). Serum IgG concentrations are elevated during the early stages of infections while serum IgA levels do not become significantly elevated until late in the course of the disease with concentrations that correlate strongly with increasing severity of HIV infection (Fling et al ., 1988). Increase in immunoglobulin production also leads to autoimmune syndromes with antibodies made to a variety of normal cellular proteins. This is suggested to be by blocking cell to cell interactions or by destruction of T cells through either antibody mediated complement lysis (AMCL) or antibody dependent cellular cytotoxicity (ADCC) (Ojo-Amaize et al., 1987; Rook et al., 1987). The human immunodeficiency virus has been shown to induce neutralizing antibodies that could help to prevent the transfer of virus in the host (Weiss et al., 1986) and in contrast, the virus has also been shown to induce antibodies that enhance Virus infection by spreading HIV in the infected individuals (Robinson et al., 1988). Antigenic variants presumed to be selected by the pressure of neutralizing antibody are generated during long term infection (Benn et al., 1985).

With hyperactivity of B cells and a depression of the T4-lymphocyte subset, majority of AIDS patients have shown a low T4 : T8 ratio (Fahley et al., 1984). The presence of T8-lymphocytes in blood suppresses HIV replication without killing the infected cell (Walker et al., 1986). This antiviral effect is suggested to

be due to the production of a lymphokine by T8 cells (Walker and Levy, 1989) but lymphocytes from patients with AIDS are deficient in the synthesis of lymphokines. T cells synthesize interleukin -2 (IL-2) whose production is impaired in patient with AIDS (Rook et al., 1983). The production of gamma-interferon (Y-IFN), synthesized by T4 cells is also defective in AIDS and this is suspected to account for susceptibility to opportunistic infections (Murray et al., 1984). HIV or its components might be recognized as a foreign MHC class II antigen, thus directly stimulating T-cells and indirectly stimulating B cells in a manner similar to that in immunostimulatory Graft-versus-host (GVH) reaction (Shearer et al., 1986). The gp120 and gp41 proteins of HIV are found to bear structural similarities to MHC class II molecules. Thus in HIV infection, as in immunostimulatory GVH model, Virus infected cells and the activated B cells may induce suppressor cells and the resultant depressed T cell function (Young, 1988).

HIV proteins can inhibit T cell response to mitogens, antigens and allogenic cells (Amadori et al., 1988) and it can directly stimulate normal B cells and cause the early events in HIV infection (Schnittman et al., 1986). Monocytes/macrophages, Langerhans cells in the skin, follicular dendritic cells in lymph nodes, microglial/monocytic precursors in bone marrow have been shown to be infected or functionally affected by HIV. Infected monocytes may serve as a reservoire, transporting the virus throughout the body (Gendelman et al., 1988), they may also have

decreased expression of HLA-DR+ antigens and display decreased phagocytosis and chemotaxis (Belisto et al., 1984). The production of immune response inhibitors by monocytes such as prostaglandin and IL-1 inhibitors, might also be responsible for some of the immune abnormalities seen in HIV infected patients (Burks et al., 1987).

Host immune response can modulate, reduce or enhance viral pathogenesis. Attack of cytotoxic T cells on both virus infected and uninfected CD4+ cells, autoantibodies produced against antigens induced by the virus on lymphocytes, normal cellular antigens made immunogenic by HIV may contribute to the disorders (Stricker et al., 1987; Weinhold et al., 1988). Viral gene products may be directly toxic to cells or interrupt production of cytokines by infected cells needed for the normal growth and functioning of immune cells, decrease expression of IL-2 receptors in infected cells, affect stem or early progenitor cells of the haematopoietic system, and reduce the capacity of reconstitution of the immune system (Brenneman et al., 1988). Endogenous TNF-alpha is explained to be responsible for the activation and maintenance of HIV expression in chronically infected cells impairing the function of T4 cells and other normal immune responses (Rosenberg and Fauci,1990).

HIV infected macrophages that release cytokines toxic to brain cells and to myelin are suggested to cause neurological disease

upon migration to the brain (Price et al., 1988). Dementia and other neurological symptoms are caused by infection of astrocytes and oligodendrocytes which damage the production of substances that help in maintaining the blood-brain barrier (Fontana et al., 1984). Neurologic manifestation of AIDS include acute encephalopathy, cerebral mass lesions, seizures, headaches, spinal cord disorders, neuropathy, lumbosacral polyradiculopathy and myopathy (So, 1989).

Direct infection of enterochromaffin cells of the bowel is said to induce low-grade small bowel atrophy, malabsorbtion and chronic diarrhoea observed in some HIV infected individuals (Ulrich et al., 1989) while infected macrophages in the stroma of the bowel are suggested to be an indirect source of pathology through production of toxic cytokines (Levy, 1989).

Since infection by one HIV subtype does not prevent infection by the other, an initial infection with a non-cytopathic HIV-2 might be followed by a subsequent infection with a pathogenic HIV-1 subtype or vice versa, influencing the progression of the disease (Evans et al., 1988).

The global pandemic of HIV infections remains dynamic and is continuing to expand in three ways. First, HIV infections are on the increase in already affected areas. Second, the epidemic has expanded its geographical scope, reaching countries and regions previously unaffected or only slightly affected by HIV.

and the Pacific. Different patterns may co-exist within a single country, or even within a large city (Piot et al., 1988).

According to WHO's (1991) update information on AIDS it is estimated that world wide a total of 5-6 million men and 3-4 million women have been infected with HIV. Of these 8-10 million infected adults,over 1 million have progressed to AIDS, and a similar number have developed less severe illness related to their infection.HIV infected mothers have thus far borne almost 1 million infected children, as well as another almost 2 million uninfected children who are already or potentially AIDS orphans.WHO's (1991) current conservative projection is that by the year 2000, a total of 30-40 million men, women, and children will be infected with HIV since the start of the pandemic.This will represent a tripling or quadrupling of the present total in 8 years time. By the end of the 1990's over a million adult AIDS cases and deaths can be expected per year, with the majority of these in the developing countries - about 500,000 in Africa and about 250,000 in Asia (Chin et al.,1990)

In the Americas there have been an estimated 2 million HIV infections thus far - 1 million in North America and 1 million in Latin America. Western Europe has had about 0.5 million infections. The highest prevalence is in Sub-Saharan Africa, where about 6 million adults have been estimated to be infected. In South and South East Asia - primarily India and thailand, there have been well over 1 million infections. Most other areas are believed to have relatively low levels of infection (WHO, 1991).

denies other risk factors and has had heterosexual contact with a person with AIDS or at risk for AIDS or was born in a country in which heterosexual transmission is believed to play a major role, although the precise means of transmission has not yet been fully defined (CDC, cited by Haverkos and Edelman, 1988). Heterosexual contact was first proposed as the means of HIV spread (Clumeck et al., 1984). The nearly equal sex distribution and lower mean age for female patients among subjects who are in their sexually active years of life is considered as strongly suggestive of heterosexual transmission of HIV (Melbye et al., 1986). Furthermore, the fact that the use of condom was associated with a significantly reduced risk of being HIV seropositive in Zairian prostitutes is an additional evidence that HIV transmission can occur through heterosexual intercourse (Mann et al., 1987).

It is estimated that at least 80 percent of people with HIV infection in Africa have contracted the virus through heterosexual intercourse, whereas the estimate for the USA is only 4 percent (Mann et al., 1988). Observations in central and eastern African countries provided the first strong evidence for the possibility of heterosexual HIV transmission and identified prostitutes and heterosexually promiscuous men as high risk groups promoting the transmission (Piot et al., 1984; Padian, 1987). In subsequent seroprevalence surveys in Zaire, Tanzania, Kenya, Rwanda and Ethiopia 5% to 88% of prostitutes were found to have antibodies to HIV (Mhalu et al., 1987; Kreiss et al., 1986; Van de Perre et al., 1985; Seyoum, 1987). It has been proposed that the vagina of a

prostitute may actually serve as a vessel for contaminated semen from a previous customer that could be mechanically passed onto the next customer (Wykoff, 1986).

The reasons for fast spread of HIV in Africa with a predominantly heterosexual transmission are still incomplete. A large number of sexual partners may be one explanation (CDC, 1988), inadequate health care or unhygienic living conditions were also suggested to affect susceptibility to infection and to account for higher rates of infection and disease among African heterosexuals (Collier, 1986) seropositivity in Africa was not associated with reported participation in fellatio or anal intercourse nor with kissing behaviour (Mann et al., 1987) while this practice was clearly related to higher rates of heterosexual transmission in Europe (De Vincenzi et al., 1989). It is clearly evident that unwillingness of infected individuals to admit homosexual behaviour or intravenous drug use may alter the general picture. Receptive anal intercourse has been shown to be a well known risk factor for transmission of HIV in homosexuals (Kingsley et al., 1987).

HIV exposure through parenteral antibiotic treatment with reused needles was excluded as a risk factor (Melbye et al., 1986) while others proposed it to account for African HIV infection (Biggar, 1986). But it has been suggested that HIV may be acquired more readily from circumcised women or more readily by uncircumcised men (Burton, 1986; Cameron et al., 1989). Blood

transfusion is a common hospital procedure in Africa and has been accounted for as much as 10 percent of HIV infections in African countries whereas in the USA it accounts only for 0.3 percent (Greenberg et al., 1988). There is also some suggestive evidence of a genetic risk or susceptibility although consistent data are lacking to support is (Lifson et al., 1988).

STDs have been identified as important risk factors for sexually acquired HIV infections in many countries (WHO, 1989). Case - control epidemiological studies have demonstrated that infections which cause genital ulceration may facilitate sexual transmission by increasing an HIV - seronegative partner's vulnerability to infection and also by increasing the infectivity of HIV - seropositive sexual partners (Serwada et al., 1985). The high rates of STD found in many areas of Africa, may contribute to the demonstrated relationship with heterosexual HIV infection.

HIV has a lower transmission probability than other heterosexually acquired infections. The best studied is gonorrhea, where the male to female transmission probability is around 0.5 for a single sexual contact (Platt, et al., 1983) and the corresponding female to male transmission probability is around 0.2 - 0.3 for a single exposure (Hooper et al., 1978). For HIV, the estimate of an average heterosexual transmission probability is 0.1 per partner for female to male and around 0.2 per partner for male to female provided the cofactor (STDs) are present (Anderson and May, 1988).

When STDs are not present the rate of male to female transmission of HIV have been estimated to be approximately 1 in 500 sexual exposures (Hearst and Hulley, 1988).

STDs have been implicated as the most likely cofactors in the African HIV epidemic (Padian, 1987) and it has been postulated that HIV and STDs may promote one another's spread (Aral and Holmes, 1991). Eventhough the data on association between STD and HIV infection are inconsistent and insufficient to assess their roles as risk factors for HIV transmission, it was found biologically plausible for all STD pathogenes that cause genital ulcers or inflammation to be risk factors for increased infectiousness or increased susceptibility to HIV infection (WHO, 1989). HIV seropositivity was associated with a history of genintal, anorectal or oral herpes, a history of syphilis, or positive serological tests for herpes simplex virus type - 2 (HSV-2) or Treponema pallidium after controlling for the number of sexual partners (Handsfie d et al., 1987). A recent history of STD was found to be more comm n among men whose partners were infected with HIV and women were more likely to be positive for HIV when they reported a history of STD in the past five years. The STDs reported include syphilis, gonorrhea, chlamydiosis, candidiasis, genital ulcers, genital warts, trichomoniasis , and genital herpes (De Vincenzi et al., 1989). Co-infection studies in vitro suggested STDs as cofactors since some have shown association with an increase in HIV expression (Lifson et al., 1988). Therefore, due to the high rates

of change in sexual partners and the likelyhood that STD infections increase the average transmission probability of HIV, attendees at STD clinics are considered to be particularly at risk (Anderson and May, 1988).

The prevalence of past history of STD in AIDS patients or HIV seropositives was high (35% - 75%) in Tanzania, Rwanda, Haiti, and Zimbabwe (Piot et al., 1984; Pape et al., 1983; Van de Perre et al., 1984; Mhalu et al., 1987). The increase in HIV seroprevalence in STD clinic attendees compared with the overall population was between twofold and twentyfold in Tanzania, Zambia, Burundi, and Rwanda (Mhalu et al., 1987; Melbye et al., 1986; Van de perre et al., 1987; Galli et al., 1987). In zaire and Zambia patients with a history of STDs were significantly more likely to be HIV seropositive than the control populations (Piot et al., 1984; Melbye et al., 1986). In Ethiopia HIV seropositivity increased with increasing episode of STDs and the frequency of sexual contact with prostitutes (Getachew et al., 1988).

HIV-1 and other STDs are spread from infected persons to their sex partners during unprotected sexual exposures. Like AIDS, nonsexual transmission of STDs in children occurrs most frequently from an infected woman to her child in utero or at the time of delivery. Infections acquired in utero include syphilis and occasionally herpes and HIV infection. Infections transmitted during delivery include gonorrhea, chlamydiosis, herpes, and human papiloma virus (HPV) (Schwarcz and Whittington, 1990). Among the

hypothesis which have been put forward to account for the causal part that STDs may play include occurrence of immunologic modifications because of infection. The detection of antigens in $OKT4^+$ lymphocytes from vaginal and cervical secretions of seropositive women, but not in genital epithelial cells suggests that conditions that increase the number of lymphocytes in the female genital tract, such as STDs that elicit an inflammatory response, may potentiate the risk of HIV transmission by increasing the pool of infected cells in a seropositive person or the pool of target cells in a seronegative person (Kreiss et al., 1988). Damage to the protective vaginal mucosal epithelium could also facilitate the entry of HIV into the blood stream.

In many developing countries where chancroid is a common genital ulcer disease (GUD), it has been associated with an increased prevalence of infection with HIV (Quinn et al., 1987). In a prospective study of men with STDs and recent sexual contact with prostitutes men with GUDs were more likely to seroconvert during a two to six month period than were men with urethritis (Cameron et al., 1987). Study on heterosexual men with current genital ulcers showed that 63 percent of the HIV seropositives reported a prior episode of GUD (Greenblatt et al., 1988). In another prospective study seroconversion was associated with GUDs and with being uncircumcised (Cameron et al., 1988) probably because intact foreskin predisposes to the acquisition of chancroid and other GUDs (Pepin et al., 1989). Mechanistically, GUDs greatly

increase the infectivity of seropositive people due to the disruption of integrity of the mucosal epithelium which allows easier penetration by the virus (Kreiss et al., 1985), while epidemiologically, the data are also consistent with the confounding effects of sexual promiscuity, acquisition of GUDs and HIV exposure (Haverkos and Edelman, 1988).

Associations have also been shown between HIV and Treponema pallidum, causative agent for Syphilis. Syphilis has been associated with HIV infection in heterosexual men and women (Hudson et al., 1988) and in homosexual men in the US and London (Evans et al., 1986; Samuel and Winkelstein, 1987). In Haiti 32 % of AIDS patients had a positive Veneral Diseases Research Laboratory (VDRL) test or Rapid Plasma Reagin (RPR) test (Pape et al., 1983). In Rwanda 44 % of AIDS patients were seropositive for syphilis (Van de Perre et al., 1984). In Ivory Coast a correlation was reported between reactive microhaemagglutination assay for Treponema pallidum (MHA - TP) and HIV (Dennis et al., 1987). In Baltimore STD clinic 28.6 % of hetero and homosexual male attenders with a positive Fluorescent Treponemal Antibody - absorbtion (FTA-ABS) were HIV positive while only 4.5 % of those with a negative FTA-ABS were HIV positive (Quinn et al., 1987).

Herpes viruses have a role in HIV infection. Infection with HSV is able to enhance the replication of infectious HIV. Data suggest that individuals with previous herpes virus infections are

particularly susceptible to either HIV infection or disease progression (Mosca et al., 1988). A study among San Francisco homosexuals indicated that 68% of HIV seroconverters had antibodies to HSV-2 and seroconversion to HSV-2 was found in 42 % of HIV seroconverters (Holmberg et al., 1988). Another study performed among African heterosexual men and women revealed that 82 % of the HIV seropositives had antibodies against HSV-2 (Hermans et al., 1987).

There was only a weak association between HIV positivity and a past history of gonorrhea in male heterosexuals and homosexuals in Baltimore STD clinic (Quinn et al., 1987). In Africa an association between HIV positivity and current gonorrhea had been found among a small cohort of 90 Nairobian prostitutes (Kreiss et al., 1986). In another study the incidence of gonococcal infections was identical in seroconverters and non-seroconverters (Plummer et al., 1988). Among a cohort of 340 men attending an STD clinic in Nairobi there was no association between HIV infection and past history of urethritis, the majority of which were gonococcal (Simonson et al., 1988). Cervical ectopy, a normal physiologic event in the female genital fract is, however, said to increase the susceptibility of women to HIV-1 infections (Moss et al., 1990). Cervical ectopy is known to increase the risk of chlamidia and gonococcal infection in women. A work in Zaire suggests that gonococcal infection and Trichomonas vaginalis also increase the susceptibility of women to HIV (Laga et al., 1990).

Eventhough some of the evidences for the association of STDs and HIV infections are strong and consistent, the need for further research and studies to confirm the role of STDs as cofactors of HIV transmission is immense. According to Pepin et al., (1989), this analysis of the relationship between STD and HIV is faced with two problems. First the conventional STDs may merely be markers of sexual promiscuity and not causal risk factors and hence the importance of controlling for sexual activity of study subjects and their partners. Second, that HIV induced immunosuppression may increase the susceptibility of individual to STDs thus making it important to conduct prospective studies to determine which comes first, HIV infection or infection with other STD agents. In the case of studies carried out in high risk groups, the limited generalizability of the results is stressed as a third problem in cross-sectional and case control studies of the interaction between HIV infection and other STDs (Mertens et al., 1990).

In Africa, AIDS affects the general heterosexual population with varied impact between geographic areas, ethnic groups, and other subpopulation (Haverkos and Edleman, 1988). Furthermore, STDs which may potentiate the sexual transmission of HIV are a major health problems among older adolescents and young adults in the continent. The age pyramid with large proportion of teens and young adults and a relatively few older adults which is a characteristic of developing countries together with other characteristics conducive to high incidence and rapid spread of STDs including: poor socio-economic minority conditions, high

population growth rate, high rate of urbanization, population movements of all types, rapid drastic economic changes, unstable power hierarchies, prostitution, and sexual behaviours (Aral and Holmes, 1991) seem to play the suggested role to the spread of STDs in Ethiopia also.

Every year 250 million cases of gonorrhea and 50 million cases of syphilis are reported world wide. Surveys conducted in Africa indicate an annual gonorrheal incidence rate of 20-50% among high risk groups such as prostitutes, 3% in general population group, and 17% in family planning attendees. In tropical countries 50-80% of STD male patients are found in the 20-29 years age group while the majority of female patients are in the 14-24 year age category (WHO, 1986).

During eight years of retrospective study (1982-1989) in all hospitals and health centres of Ethiopia 99,354 cases of syphilis, 72,344 cases of chancroid, 435,723 cases of gonorrhea and 55,405 cases of Lymphogranuloma Venerum (LGV) were recorded (MOH, 1988; MOH, 1990). In Addis Ababa, the assessment of the prevalence of the STD pathogens resulted in 28% N. gonorrhea, 21% T. vagnalis, 14.7% C. albicans and 13.8% syphilis, from 282 randomly selected sex workers (Aberra et al., 1990). This shows that N. gonorrhea is the most highly prevalent STD agent and the result goes well in line with other previous investigations (Habte-Gaber et al., 1983; MOH, 1988, 1990).

A study in Addis Ababa indicated 6.5% of VDRL positive samples

to be HIV seropositive (Seyoum et al., 1988), while STDs were the major risk factors identified in 39.3% of the AIDS patients in Ethiopia in 1989 (Hailu et al., 1990a). Furthermore, Getachew et al. (1988), found in a group of lumpen individuals in Addis Ababa, that HIV seropositivity increased with increased episode of STDs. Another study on the prevalence of HIV infection among military patients with STDs showed that 12% of the patients were positive for HIV-1 antibody. (Hailu et al., 1991). Among the current report of AIDS cases, eventhough most of the patients had two or more risk factors, multipartner sexual contacts (MPSCs), history of STDs, and injections outside medical institutions were the three major risk factors identified in 71%, 48% and 4.4% of the patients, respectively. History of blood transfusion and IVD use were noted as minor risk factors in 3.4% and 0.1% of the patients (MOH, 1991). Another study on the sexual practices of 2700 females involved in MPSCs in Addis Ababa showed that 98% practiced peno-vaginal sex only, 1.7% occassionally practiced peno-rectal sex and 0.2% peno-oral sex indicating that the latter are not commonly practiced (Mengistu et al., 1990a).

In all the senior highschools of Addis Ababa, including the technical and vocational schools, 124,000 students were enrolled in the 1990/91 academic year and the increase in the number of students is estimated to be 5 to 10 percent annually (MOE, 1991), while information from the Addis Ababa University (AAU) registrar office brings the number of students in all the colleges of AAU in Addis Ababa to 10,000. Most of the students in all the senior

highschools and colleges include adolescents and young adults.

A survey of the sexual behaviour and knowledge of AIDS and other STDs in senior highschool students in Addis Ababa has revealed that the students on the average have a modest knowledge of AIDS, while knowledge of STDs was found to be very low. The survey also showed that over a third of the students sampled in the study have actively experienced sex (Solomon, 1990). Such a study has not yet been conducted on college students.

In the USA the seroprevalence of HIV among university students was found to be 0.2% indicating the presence of HIV infection in university campuses and its potential for further spread in this population (Gayle et al., 1990). As many students in Ethiopian highschools and colleges seem to engage in behaviours that could place them at risk for HIV infection a far worse rate of HIV infection could be expected in those who especially put themselves into a risk for contracting STDs.

The present study aims to investigate the student population that attend clinics for STDs. The population of college and highschool students fall in the category of sexually active and hence as a group at a relatively high risk of contracting and transmitting AIDS. Therefore, the objectives of this study are: First, to assess the present prevalence of HIV on students of colleges and highschools in Addis Ababa that attend STD clinics. second, to study the correlation of HIV and STDs among students, and third, to contribute to the effort of national assessment of HIV in Ethiopia.

MATERIALS AND METHODS

STUDY POPULATION

Blood specimens were collected in 10 ml vacutainer tubes by laboratory technicians from presenting samples of students of colleges and highschool in Addis Ababa who attended the Arada, Shiromeda and Tekle-Haimanot health centres for sexually transmitted disease (STD) complaints. Students were identified as students by the health centres upon registration. Students who were positive for any one of the STDs at the time of enrollment and those who said they had STDs as diagnosed by clinicians during the last five years were considered as experimental population. Students who showed no clinical symptoms for any one of the STDs as determined by the physicians at each health centre at the time of enrollment and during the last five years were considered as control population. Students from Tikur Anbessa, Menelik II, Entoto, Yekatit 23 and kolfe Comprehensive Secondary Schools as well as students from the Natural Science. Technology, Pharmacy and Social Science Faculties of Addis Ababa University (AAU) were also invited by notices to enroll in this study voluntarily. The process of collecting and testing of smaple specimens was conducted between January 1, 1991 and January 1, 1992.

INTERVIEW AND EXAMINATION

Each participant was interviewed by a physician or by a loboratory technician or by the investigator with a detailed questionnaire which explored demographic characteristics, current and past health status emphasizing the symptoms suggestive of HIV infection, sexual practices, use of condom, blood transfusion and donation and occurrence and frequency of STDs in the past (Annex 1).

Blood was obtained from each participant with 10 ml vacutainer tubes by laboratory technicians for serological studies. Pelvic examination was performed by the physicians of the respective clinics. Samples were taken from cervical and urethral swabs and Gram Stain carried out and observed microscopically. For STDs such as chancroid, urethritis and Lymphogranuloma Venerum (LGV) the physician's diagnosis was used while for gonorrhea and syphilis further diagnostic laboratory examinations were made at the National Research Institute of Health (NRIH).

LABORATORY TESTS

A/Diagnosis of Neisseria gonorrhea - Laboratory diagnosis for N. gonorrhea was made based on the bench level laboratory manual for STDs of Van Dyck et al., (1987). A smear was made for direct microscopical examination. The swab was smeared over a clean slide to obtain a thin homogenous film and was dried by air. The smear

was fixed by passing the slide rapidly three times through a Bunsen Burner flame and prepared for gram staining. First, the fixed smear was flooded with crystal violet (mixed solution of (a) 2g. of Crystal Violet of 90% dye content with 20 ml of 95% ethanol and (b) 0.8g. of ammonium oxalate with 80 ml of distilled water) for 60 seconds, washed off in running water and flooded again with Gram's Iodine (1g. iodine, 2g. Potassium iodide and 300 ml distitilled water) for 60 seconds. Then the iodine was poured off and the slide rinsed with running water. It was decolorized with a 1:1 mixture of acetone and 95% ethanol for 10-20 seconds and was finally counterstained for 30 seconds with safranin (0.25g of safranin 0 of 90% dye content with 10 ml of 95% ethanol and 100 ml. distilled water). The slide was washed gently with water. blotted dry and gram negative gonococci were examined with a light microscope and immersion oil using a 100x objective.

The specimens collected from health centres far from NRIH were carried in Amies transport medium (Cultiplast 24/90, LP Italiana SPA, Milan) within 12 hours and inocculated into a Thayer Martin selective growth medium (Oxoid Ltd., Hampshire, UK).

Aimes Transport Medium composition per litre of distilled water.

Charcoal, pharmaceutical neutral	10.0g
Sodium Chloride	3.0g
Disodium phosphate	1.15g

Monopotassium phosphate	0.2g
Potassium chloride	0.2g
Sodium thioglycolate	1.0g
Calcium chloride	0.1g
Magnesium chloride	0.1g
Agar powder	4.0g

Modified Thayer Martin (MTM) selective growth medium

10 g. haemoglobin prepared in 500 ml distilled water and 10 ml of enrichment solution were added in 36 g of GC agar base prepared in 500 ml distilled water to produce chocolate agar and finally a VCNT selective antimicrobial was supplemented.

a) GC agar base (per litre of distilled water)

Peptone	15g
Corn Starch	1g
Dipotassium Phosphate	4g
Potassium phosphate	1g
Sodium chloride	5g
Agar powder	10g

b) Haemoglobin

Dried powder of bovine haemoglobin 10g

c) Isovitalex enrichment (per 10 ml distilled water)

Diphosphopyridine nucleotide

(coenzyme 1)	2.5 mg
Cocarboxylase	1.0 mg
P-aminobenzoic acid	0.13 mg
Thiamine - HCI	0.03 mg
Vitamin B^{12}	0.1 mg
L-glutamine	100.0 mg
L-cystine-2HCI	11.0 mg
L-cystine-HCI-$2H_2O$	259.0 mg
Adenine	10.0 mg
Guanine-HCI	0.3 mg
Ferric nitrate - $9H_2O$	0.2 mg
Dextrose	1.0 g

d) VCNT antimicrobials (Per 500 ml. distilled water).

Vancomycine	1.5 mg
Colistine	3.75 mg
Nystatine	6.25 units
Trimethoprim	2.5 mg

The swab containing the specimen was smeared over approximately one-fourth of the plate. The inoculum was spread on the remaining part of the medium by means of a sterile loop so as to be able to get isolated colonies. The inoculated plates were incubated in candle Jar to produce enough $C0_2$ at 36°C with a wet cotton. After incubation, plates were examined for the growth of a typical colony of gonococci.

A presumptive identification of colonies with gonococcal-like appearance were made by Gram's stain and oxidase test. For the oxidase test commercial oxidase disks containing dimethyl-P-Phenylene diamine hydrochloride were used. Colonies with suspected appearance were picked up by a loop and rubbed on to the disks. Positive reactions turned the paper purple within 20 seconds.

Final confirmatory diagnosis was made for oxidase positive diplococci by sugar tests. In this test fresh subcultures of strains to be tested were prepared on non selective chocolate agar (described above as part of the MTM growth Medium) with 5% blood (bovine). Two loopfuls of the isolate were taken in 24 h into a tube containing 0.4 ml Buffered Balanced Salt Indicator Solution (BSS).

Buffered BSS composition Per litre (pH 7.1-7.2)

Dipotassium phosphate	0.4 g
Potassium phosphate	0.1 g
Potasium chloride	8.0 g
Phenol red	0.6 g

Five tubes were used for this test. 0.05 ml 20% sterile glucose, maltose, sucrose and lactose were added to individual tubes follwed by 0.1 ml BSS to each one. The fifth tube without sugar was used as control tube. Finaly, 0.05 ml of the bacterial suspension was transferred to each of the five tubes, mixed, and

incubated in a 37°C water bath for 4 h. A colour change from red to yellow was considered as a positive diagnosis of gonorrhea.

B/ Diagnosis of Treponema pallidum - In the diagnosis of syphilis the laboratory procedures of Van Dyck et al., (1987) were used. Sera were first screened with a qualitative Rapid Plasma Reagin (RPR) card test. A volume of 50 ul of serum was placed on to a circle of the test card by using a sampling pipette delivered with the RPR - card kit. The drop was spread to fill the entire circle. One drop of antigen in charcoal suspension was added. The card was placed on a mechanical rotator with a wet cotton for approximately 8 minutes. After removing the card from the rotator, reactive sera were differentiated from non-reactive sera by brief rotating and tilting of the card by hand. Small to large clumps or block flocculation indicated reactive serum while no clumping or only very slight roughness indicated non-reactive serum.

A Treponema pallidum Haemagglutination Assay (TPHA) test kit was used for a qualitative assay of confirming reactive sera of RPR. Using a pipette 25 ul absorbing diluent (which consists of sonicated cell membranes from sheep and ox erythrocytes, normal rabbit testicular extract, sonicated treponemes, normal rabbit serum, Tween 80 and acacia powder in phosphate buffered saline-PBS) was placed for each test serum in a microtiter plate wells numbered 1,3,4, and 5 of horizontal rows and 100 ul in well number 2. 25 ul of test serum was brought in to well 1 (dilution 1:2); it was mixed

and 25 ul transferred to well 2 (1:10); the same was done to well 4(1:20) and to well 5 (1:40) from where 25 ul was discarded after mixing. The dilutions of all test sera were completed and the microplate was covered and incubated at room temperature for 30 minutes. After incubation, 75 ul of unsensitized cells (2.5% suspension of formalinized, tanned sheep erythrocytes not sensitized with T. pallidum) were added to well 3 (dilution 1:80) to serve as non-reactive control. With another pipette dropper 75 ul of sensitized cells (2.5% suspension of formalinized, tanned sheep erythrocytes which have been sensitized with sonicated T. pallidum) were added in wells 4 (1:80) and 5 (1:160). 25 ul of a prediluted (1:80) positive control was placed into 5 wells of a horizontal row of a microplate and diluted with 25 ul of absorbing diluent.

Finally, the microtiter plates were shaken gently, covered and incubated at room temperature for 2 hours. The settling patterns of the RBC were read with the naked eye from which haemagglutination or no haemagglutination was reported. A serum showing haemaglutination was considered as a reactive serum with a positive diagnosis of syphilis.

C/ Diagnosis of HIV - Sera were tested for antibody to HIV by an Enzyme Linked Immunosorbent Assay, ELISA (Voller et al., 1976) adapted to HIV by Brun Vezinet et al.(1984) and by Sarngadharan et al. (1984). The ELISA positive results were confirmed by Western

Blot analysis (Towbin et al., 1979) adapted to HIV by Sarngadharan et al. (1984). All the reagents for the ELISA procedure were provided with the Abbott Recombinant HIV-1 EIA test kit by the Abbott diagnostics division of Germany and the reagents for the Western Blot analysis were provided with the Western Blot IgG assay kit by the Diagnostic Biotechnology (Pte) Ltd. of Singapore. A specimen was considered positive for antibody to HIV if it was repeatedly reactive on two separate ELISA assays and was confirmed by Western Blot analysis.

Enzyme Linked Immunosorbent Assay - 10 ul volumes of the control and test specimens were dispensed into appropriate wells of reaction tray after which 400 ul of specimen diluent (bovine and goat sera in 0.1% sodium azide) was added to each well. Thereafter recombinant DNA derived HIV-1 core and evn antigen coated beads were added and the wells covered and incubated at 40°C for 30 minutes after which the liquid was aspirated and the beads washed. 200 ul of diluted conjugate (0.01 ug/ml of conjugate concentrate composed of Goat Anti-Human IgG with Horseradish peroxidase in 2-Amino-2-(hydroxymethyl)-1,3-propandiol (TRIS) buffer, pH 8.0, diluted in bovine and goat sera) was pipetted into each well for the second incubation (at 40°C) which was aspirated after 30 minutes and the beads washed. Then beads were transferred to assay tubes into which 300 ul of O-phenylenediamine. 2HCl (OPD) substrate freshly diluted in citrate-phosphate buffer (0.075M potassium phosphate and 0.05M Sodium citrate, pH 6.4, containing 0.02%

hydrogen Peroxide) was pipetted and incubated at room temperature for 30 minutes. After incubation 1 ml of 1N sulfuric acid (provided as an accessory to the Abbott Recombinant HIV-1 EIA kit) was added to each tube to stop enzyme reaction and finally, absorbance of controls and specimens was determined at 492 nm by using a spectrophotometer (Quantum II Analyzer 9/90 JS Abbott spectrophotometer). The Quantum Analyzer performed all result calculations automatically and determined reactive specimens. By relating the absorbance of the specimen to the cutoff value (which is the absorbance of the negative control mean plus 0.15 times the positive control mean) the Quantum Analyzer showed specimens with absorbance values less than the cutoff value to be negative and specimens with values greater than or equal to the cutoff to be reactive. Reactive Specimens were retested using the original sample sera and those which were found repeatably reactive were interpreted to be positive for antibody to HIV-1 by the criteria of Abbott Recombinant HIV-1 EIA.

Western Blot Assay - Nitrocellulose strips coated with separated, bound antigenic proteins from partially purified inactivated HIV-1 were incubated with 2 ml of diluted wash buffer (2-Amino-2-(hydroxymethyl)-1,3-propanediol (TRIS) with polyoxyethylene sorvitan monolaurate (Tween 20) pH 8.0) in each well for 15 minutes at room temperature. All incubations in this test were made at room temperature and on a rocking platform. The buffer was removed by aspiration and 2 ml of blotting buffer (TRIS

with goat serum and non-fat dry milk) was added followed by 20 ul of each sample's sera and controls in appropriate wells after which the tray was covered and incubated overnight (16-20 hrs.). After incubation the mixture was aspirated from samples and washed three times with 2 ml of diluted wash buffer and with 5 minutes' soak on a rocking platform between each wash. Then 2 ml of working conjugate solution (Goat anti-human IgG conjugated with alakaline phosphatase and diluted by blotting buffer) was added to each well and incubated for 30 minutes. The conjugate was aspirated and strips washed again 3 times. Finally 2 ml of working substrate solution (solution of 5-bromo-4-chloro-3-indolyl-phosphate (BCIP) and solution of nitrobluetetrazolium (NBT) in TRIS buffer) was added to each well and incubated for 10 minutes after which the substrate was aspirated and the strips rinsed with distilled water to stop the reaction. The strips were dried with paper towel and mounted on worksheet. The presence or absence of antibodies to HIV-1 in each sample was determined by comparing each nitrocellulose strip to the strips used for non-reactive and reactive controls. The test was considered positive if the nitrocellulose strips show bands at any two of the p24, gp41, gp120/gp160, structural gene products by the criteria of the Centers for Disease Control (CDC, 1989).

STATISTICAL ANALYSIS

Data analysis was performed at the National Research Institute of Health (NRIH) with Statistical Package Social Science (SPSS) and

seroprevalence for the total sample population and for each demographic variable was calculated. Statistically significant difference in rates of seroprevalence according to age, sex, and religion and rate ratios for different strata of these variables and other risk factors were calculated. Association between HIV and STDs were analysed by Chi-square and Fisher's exact test.

RESULTS

In this study a total of 324 blood specimens were collected and tested for HIV antibody. The mean and median ages of the students in the study were 19.8 and 20 years respectively (range 14 to 35). Sexually Transmitted Disease (STD) cases comprised 66% of the sample. The remaining 34% were control subjects found to be free from any one of the STDs at the time of examination and reportedly during the past five years. Demographic data for these samples are shown in tables 1,2, and 3. Out of these 78% were highschool students and 22% were students from colleges. Students in the age group 18 to 22 and male students comprised 69% and 65% respectively in the sample. Among the total male population 70% were from highschools while 30% were from colleges and in the total female population 93% were highschool students and 17% were college students. The majority of the college students (69%) were in the age group 19 to 22 years.

The STD cases included students with mean age of 19.6 years and median age of 19 years (range 14 to 28). Here also, highschool students, males, and ages 18 to 22 comprised 84,66 and 74 percents, respectively. Out of the males sampled, 79% were highschool students while 21% were college students. Similarly 95% of the females sampled were from highschools while only 5% were from colleges (Table 2). The control samples without STDs included students with a mean age 20.3 and median age 20 years (range 15 to 35). Male highschool students aged 18 to 22 years made up majority

of the sample. Among these, 52% of the male population were students from highschools while 48% were from colleges. Among the females 90% of them were from highschools while 10% were from colleges (Table 3).

Table 1 Demographic Characteristics of the total sample of students in the study.

Characteristic	No. tested	Highschool n(%)	College n(%)
Age (yr)			
< 15	1	1	-
15-18	118	117(99)	1(1)
19-22	157	110(70)	46(30)
23-26	39	24(62)	14(37)
27-30	8	2(25)	5(75)
>30	1	-	1
TOTAL	324	254(78)	67(22)
SEX			
male	210	148(70)	60(30)
female	113	105(93)	7(7)
TOTAL	323	253(78)	67(22)

On the whole, 63 out of the 324 serum specimens were positive for HIV antibodies by ELISA AND Western Blot giving a seroprevalence of 19%. Tables 4 and 5 show seroprevalence data for STD and non -STD samples separately. Among 216 students with STDs, 48 were positive for HIV antibodies giving a seroprevalence of 22%. Seropositivity decreased slightly with age. that is, from 23% among students of age 15 to 18 to 21% among those aged 19 to 22 years. Seropositivity was higher among female students (27%) than among male students (19%). The difference between that of male and female students was statistically significant ($p<0.05$) while that between the different age groups did not show significant difference (Table 4).

Among 110 students without an STD, 15 serum specimens were positive for HIV antibodies giving a seroprevalence of 14%. In this group seropositivity increased with age, from 8% among students of 19 to 22 years old to 25% among those 23 and older. Seropositivity was higher for female students (25%) than for male students (7%). Similar to that of students with STDs, data for male and female students among the control showed significant difference ($p<0.03$) while that on different age groups showed no

Table 2. Demographic Characteristics of Students with STD in the Study

Characteristic	No. tested	Highschool n(%)	College n(%)
Age (yr)			
< 15	1	1	-
15-18	79	79(100)	-
19-22	109	84(77)	24(23)
23-26	24	16(67)	8(33)
27-30	1	1	-
> 30	-	-	-
TOTAL	214	181(84)	32(16)
Sex			
male	141	112(79)	28(21)
female	73	69(95)	4(5)
TOTAL	214	181(85)	32(15)

Table 3. Demographic characteristics of students with No. STD manifestations

Characteristic	No. tested	Highschool n(%)	College n(%)
Age (yr)			
< 15	-	-	-
15-18	39	38(97)	1(3)
19-22	48	26(54)	22(46)
23-26	15	8(53)	6(47)
27-30	6	1(17)	5(83)
> 30	1	-	1
TOTAL	109	73(68)	35(32)
Sex			
male	69	36(52)	32(48)
female	40	36(90)	3(10)
TOTAL	109	72(66)	35(33)

significant difference (Table 5). Students with STD cases who were over the age of 19 were at a slightly greater risk of HIV infection than those that were 19 or younger and the reverse was true for students without STD. In students with STD and those without STDs females and highschool students were at a relatively higher risk of HIV infection. Among STD cases females and highschool students were respectively 1.5 and 10.1 times more likely to be seropositive than males and college students. Among cases without STDs, females and highschool students were respectively 4.3 and 3.6 times more likely to be seropositive than males and college students (Tables 6 and 7).

Frequency of sexual contact, the number of sex partners, contact with prostitues, lack of condom use, and history of STDs, did not show significant difference with regard to HIV seropositivity. 22% of those students who claimed to be beginners of sexual intercourse were positive for HIV antibodies while those who are not beginners and who frequent sexual intercourse were 16% positive for HIV antibodies. Students who reported frequent use of condoms were 23% positive while those who admitted lack of condom use, were 18% positive for HIV antibodies (Table 8). Students with a history of chancroid, syphilis , and gonorrhea were respectively 38,29,and 14 percent positive for HIV. Those who reported treatment of STDs by taking injections at home were 25% positive for HIV while those who took injection at clinics were found to be 17% positive for HIV (Table 8).

Analysis of the risk factors among HIV positive samples (Table 9) showed that most of the HIV positive beginners of sex are of the age group of 19 or less while those who frequent sexual intercourse and who are not beginners are of the age group 20 and more. In both cases males and highschool students were affected more than females and college students. Sexual contact with more than one partner, contact with prostitutes, and past STD history were all found to constitute the majority of students of ages 20 and more, while rare use or lack of use of condoms was reported among students of ages 19 or less. Most of the risk factors among most of the HIV positive samples were greatly manifested on male students except that 62% of the females reported that their partners do not use condoms while only 59% of males admitted lack of condom use. Due to a very small sample size of HIV positive college students, percentage comparison of the risk factors for HIV positive samples of highschool students with college students was not possible. But the various comparisons made in the total study population have indicated that highschool students were more likely to be HIV positive than college students. A comparison of rate ratios of HIV infection between students of STD cases and the controls (without STDs) indicated that students with STDs were at slightly greater risk for HIV seropositivity than students without STDs in all the variables considered (Table 10).

Table 4. HIV Seroprevalence of Students with STDs according to age and sex.

Variable	No. tested	HIV + (n)	% HIV + (95% CI)*	P Value
Age (yr.)				
15-18	79	18	23(15-31)	
19-22	109	25	23(15-31)	
23-26	24	5	21(5- 37)	
27-30	1	-	0	
TOTAL	213	48	22	0.3
Sex				
Male	141	28	19(14-26)	
Female	73	20	27(18-36)	
TOTAL	214	48	22	0.05

* CI = Confidence interval

Table 5. HIV Seroprevalence of Students Without STD manifestations, according to age and sex

Variable	No. tested	HIV + (n)	% HIV + (95% CI)*	P Value
Age (yr.)				
15-18	39	7	18(6-30)	
19-22	48	4	8(1-15)	
23-26	15	3	20(0-40)	
27-30	6	1	17(7-67)	
TOTAL	108	15	14	0.93
Sex				
Male	69	5	7(1-13)	
Female	40	10	25(12-38)	
TOTAL	109	15	14	0.03

*CI = Confidence Interval

Table 6. Rate ratios for HIV Infection According to age,Sex and Level of education of students of STD cases.

Variable	No. tested	HIV + (n)	OR* (95% CI)
Age (yr.)			
> 19	103	23	1.2(0.37-4.22)
≤ 19	111	25	1
Sex			
Male	141	28	1
Female	73	20	1.5(0.79-2.77)
Level of education			
Highschool	191	47	10.12(1.42-204.56)
College	32	1	1

*OR = Odds Ratio

Table 7. Rate Vatios for HIV infection according to Age, Sex and Level of education of Students without STD manifestations

Variable	No. tested	HIV + (n)	OR* (95% CI)
Age (yr.)			
> 19	62	7	1
≤ 19	48	8	1.6(0.72-3.68)
Sex			
Male	69	5	1
Female	40	10	4.26(0.6-30.1)
Level of education			
Highschool	73	13	3.58(0.7-24.48)
College	35	2	1

*OR = Odds Ratio

Table 8. Analysis of risk factors for HIV Seropositivity.

Risk Factors	No. tested	No. HIV+	% HIV+	(95% CI)*
Sexual Contact				
beginners	154	34	22	16 - 27
frequenters	131	21	16	1 - 22
MPSCS[+]	196	40	20	15 - 26
Contact with				
prostitutes	97	14	14	7 - 20
Condom use				
always	13	3	23	0.2 - 46
sometimes	66	17	26	16 - 36
rarely	24	3	13	0.5 - 26
No condom use	221	40	18	3 - 23
STD history				
Gonorrhea	107	15	14	7 - 20
syphilis	7	2	39	5 - 63
chancroid	13	5	38	12 - 64
STD treatment				
infection at clinic	80	13	17	9 - 25
injection at home	16	4	25	4 - 46
self prescription of antibiotics	30	5	17	4 - 30

* CI = Confidence Interval

+ MPSC = Multi-Partner Sexual Contact

Table 9*. Percentage distribution of some risk factors among HIV+ samples according to age and sex.

Risk factors	Age		Sex	
	≤19	>19	Male	female
	n = 33	n = 30	n = 34	n = 29
Sexual Contact				
beginners	66%	50%	65%	52%
frequenters	21%	40%	32%	27%
MPSCs+	52%	67%	65%	52%
Contact with prostitutes	9%	33%	38%	-
Condom use				
always	3%	7%	9%	-
sometimes	12%	37%	32%	13%
rarely	9%	-	-	10%
no use	64%	57%	59%	62%
STD history	15%	47%	56%	-

* Level of education is omitted in this table because of very low number of HIV+ samples among college students.

+ MPSC = Multipartner Sexual Contact.

Table 10. Rate ratios of HIV infection between STD and non-STD cases according to some variables.

Variables	STD case		Non-STD Cases		OR(95%CI)*
	No. tested	HIV+	No. testd	HIV+	
Sexual Contact					
beginners	113	29	41	5	2.49(0.83-7.98)
frequenters	91	15	40	6	1.12(0.36-3.56)
MPSCs+	141	31	55	9	1.44(0.6-3.55)
Contact with prostitutes	74	11	23	3	1.16(0.26-5.87)
Condom use Sometimes or rarely	58	18	32	2	6.75(1.34-45.69)
No condom use	150	27	71	13	0.98(0.45-217)
STD history	100	19	27	3	1.88(0.47-8.74)
STD treatment injection at clinic	66	11	14	2	1.2(0.22-12.51)
treatment at home	35	8	11	1	2.96(0.31-144.42)

* OR Odds Ratio; CI - Confidence interval

\+ MPSC = Multipartner Sexual Contact

Table 11 examines the relationship of current and previous STDs with HIV seropositivity. There was association between chancroid and HIV infection. That is, 18% of the students seropositive for HIV had current and previous chancroid, as compared with 9% of the students seronegative for HIV infection. Among the total STDs the HIV seropositivity result for gonorrhea was significant ($P<0.01$); that for syphilis was strongly associated with HIV seropositivity in such a way that 41% of the students seropositive for HIV had current syphilis, as compared to 19% of the students seronegative for HIV (Table 12). Diagnoses of other STDs mentioned did not correlate with HIV seropositivity.

A significant seropositivity (<0.05) in males with chancroid and association between HIV and syphilis in females were determined. That is, 38% of the female students seropositive for HIV had syphilis, as compared with 15% of the female students seronegative for HIV infection (Table 13 and 14). The association with gonorrhea was significant among females ($P<0.01$). Comparison of highschool and college students (Tables 15 and 18) showed no significant association between any of the STDs and HIV among total college students, whereas there was significant correlation between syphilis and HIV among the total highschool students. In these, 35% of the highschool students seropositive for HIV had syphilis, as compared with 22% of highschool students. Categorization of highschool students according to their sex showed no significant

association between HIV and STDs (Tables 16 and 17). However, significant (P<0.05) HIV seropositivity was associated with urethritis in highschool male students with gonorrhea in female students (P<0.02).

Table 11 Current and previous manifestation of STDs according to HIV Status.

Current/ previous STDs	No. tes- ted	Mean age	HIV+ n(%)	HIV - N(%)	P Value	OR*	CI+
Gonorrhea	132	20	35(27)	97(73)	<0.16	0.7	0.4-1.21
Syphilis	61	19.6	24(39)	37(61)	0.08	1.66	0.68-4.8
Chancroid	34	19.5	16(47)	18(53)	<0.01	2.43	1.1-5.3
Urethr- itis	49	19.6	10(20)	39(80)	0.1	0.6	0.24-1.49
LGV -	13	18.4	3(23)	10(77)	0.72	0.6	0.15-2.95

Table 12. Current total STDs according to HIV Status

Current STD	No. tes- ted	Mean age	HIV+ n(%))	HIV- n(%)	P value	OR*	CI +
Gonorrhea	109	19.7	20(8)	89(82)	0.01	0.5	0.27-0.93
Syphilis	61	19.6	24(39)	37(61)	<0.009	2.2	1.16-4.28
Chancroid	31	19.4	11(35)	20(65)	0.25	1.58	0.6-3.7
Urethr- itis	49	19.6	10(20)	39(80)	0.25	0.65	0.28-1.44
LGV -	13	18.4	3(23)	10(77)	0.5	0.8	0.17-3.3

Table 13. STDs of total males according to HIV Status

Current STD	No. tes-ted	Mean age	HIV+ n(%)	HIV- n(%)	P Value	OR*	CI+
Gonorrhea	72	19.1	11(15)	61(85)	0.07	0.5	0.2-1.16
Syphilis	40	19.3	12(30	28(70)	0.14	1.8	0.7-4.32
Chancroid	26	21	9(35)	1765)	0.05	2.38	0.8-6.4
Urethr itis	24	18.9	3(13)	21(87)	0.2	0.47	0.1-1.8
LGV-	8	16.8	2(25)	6(75)	0.5	1.21	016-7.08

Table 14. STDs of total females according to HIV status

Current STD	No. tes-ted	Mean age	HIV+ n(%)	HIV- n(%)	P Value	OR*	CI+
Gonorrhea	37	19.1	9(24)	28(76)	<0.01	0.32	0.11-0.91
Syphilis	21	19.2	12(57)	9(43)	<0.002	6.93	2.0424.32
Chancroid	5	21	3(60)	2(40)	0.2	3.05	0.38-27.9
Urethr-itis	25	18.9	7(28)	18(72)	0.6	0.67	0.22-2.01
LGV-	5	16.8	1(20)	4(80)	0.4	0.46	0.02-4.7

Table 17. HIV Status in highschool female students with STDs.

Current	No. test-ed	mean age	HIV+ n(%)	HIV- n(%)	P value	OR*	CI+
Gonorrhea	35	19	9(26)	26(74)	0.02	0.33	0.11-0.96
Syphilis	19	19.3	11(58)	8(42)	0.06	2.68	0.83-8.83
Chancroid	4	20.6	2(50)	2(50)	0.5	1.55	0.15-16.6
Urethri-tis	14	18.8	2(14)	7(86)	0.2	0.39	0.05-2.32
LGV-	5	16.8	1(20)	4(80)	0.07	3.14	0.72-14.4

Table 18. HIV status in total college students with STDs

Current STDs	No. test-ed	Mean age	HIV+ n(%)	HIV- n(%)	P value	OR*	CI+
Gonorrhea	21	21.4	1(5)	20(95)	0.1	0.16	0.01-1.89
Syphilis	6	21.6	2(33)	4(67)	0.16	4.83	0.4-59.67
Chancroid	6	21.7	2(33)	4(67)	0.16	4.83	0.4-59.67
Urethr-itis	5	21	-	5(100)	0.4	0.00	0.00-9.37
LGV-	-	-	-	-	-	-	-

* OR = Odds Ratio

\+ CI = 95 percent confidence interval

\- LGV = Lymphogranuloma venerum

Analysis of the relationship of HIV and STD in the total study population indicated that syphilis and chancroid were significantly correlated with HIV while gonorrhea and total STD cases did not show correlation (Table 19). In this analysis, 39% of the total study population of students who were seropositive for HIV had syphilis, as compared with 15 percent of those seronegative for HIV infection ($P<0.0003$), and 35% of the total population seropositive for HIV had chancroid as compared to 17% seronegative ($P<0.01$). The HIV seropositivity result for the total STDs among the total population studied was significant ($P<0.05$). The influence of confounding factors on the association of syphilis and chancroid with HIV seropositivity is shown in Tables 20 and 21. Table 20 shows that in all the strata of the confounding factors, the relative risk of HIV infection associated with syphilis were significant. Therefore, multipartner sexual contacts, contact with prostitutes, lack of condom use, past STD history, and current STD cases were risk factors for both syphilis and HIV indicating the association between HIV and syphilis can be influenced by these factors. But the factors did not show the same for chancroid (Table 21) indicating lack of influence by the variables of the association between chancroid and HIV positivity.

Table 19. Syphilis, chancroid, and total STDs and HIV status in the total population of students

	HIV+	HIV-	TOTAL	%HIV+	RR(CI)*
Syphilis +	24	37	61	39	2.6(1.73-4.06)
Syphilis -	39	244	263	15	P<0.00003
Total	63	261	324	19	
Chancroid +	11	20	31	35	2.00(1.17-3.4)
Chancroid -	52	241	298	17	P< 0.01
Total	63	261	329	19	
Gonorrhea +	20	89	109	18	0.93(0.58-1.5)
Gonorrhea -	43	175	218	20	P< 0.76
Total	63	264	327	19	
Total STDs	48	166	214	22	1.6(0.97-2.8)
Total Non-STDs	15	95	110	14	P<0.05
Total	63	261	324	19	

* RR = Relative risk

CI = 95 percent confidence Interval

Table 20. Syphilis and HIV Status in the total population of Students Stratified by some risk factors

	HIV+	HIV-	Total	%HIV+	RR (CI*)
a) MPSCs					
Syphilis +	16	22	38	73	2.77(1.64-4.68)
Syphilis -	24	134	158	15	P = 0.0002
Total	40	156	196	20	
b) Contact with Prostitutes					
Syphilis +	5	16	21	24	2.01(0.75-5.36)
Syphilis -	5	67	76	12	P + 0.16
Total	14	83	97	14	
c) No condom use					
Syphilis +	16	22	38	42	3.62(2.08-6.31)
Syphilis -	20	152	172	12	P = 0.000006
Total	36	174	210	17	
d) STD history					
Syphilis +	8	16	24	42	2.67(1.23-5.79)
Syphilis -	12	84	96	12	P = 0.01
Total	20	100	120	17	
e) Current STDs					
Syphilis +	17	31	48	35	1.96(1.19-3.23)
Syphilis -	30	136	166	18	P = 0.01
Total	47	167	214	22	
f) Total Population					
Syphilis +	24	37	61	39	2.6(1.73-4.06)
Syphilis -	39	224	263	15	P = 0.00003
Total	63	261	324	19	

RR = Relative risk

CI = 95 percent Confidence Interval

Table 21 Chancroid and HIV Status in the total population of Students Stratified by some risk factors.

	HIV+	HIV-	Total	%HIV+	RR(CI)*
a) MPSCs					
Chancroid +	8	15	23	35	1.88(0.9-3.57)
Chancroid -	32	141	173	18	P = 0.06
Total	**40**	156	196	**20**	
b) Contact with prostitutes					
Chancroid +	3	6	9	33	2.67(0.91-7.83)
Chancroid -	11	77	88	13	P = 0.09
Total	**14**	83	97	**14**	
c) No condom use					
Chancroid +	6	15	21	29	1.8(0.85-3.82)
Chancroid -	30	159	189	19	P = 0.14
Total	36	174	210	17	
d) STD history					
Chancroid +	3	5	8	38	2.47(0.91-6.69)
Chancroid -	17	95	112	15	P = 0.1
Total	20	100	120	17	
e) Current STDs					
Chancroid +	10	20	30	33	1.66(0.93-2.97)
Chancroid -	37	147	184	20	P = 0.1
Total	47	167	214	22	
f) Total population					
Chancroid +	11	20	31	35	2.00(1.17-3.4)
Chancroid -	52	241	293	17	P = 0.01
Total	63	261	324	19	

* RR = Relative risk

CI = 95 percent Confidence Interval

DISCUSSION

The seroprevalence results for HIV antibodies in this study support the hypothesis that HIV infection and the possibility of its transmission among students of highschools and colleges exists. The overall seroprevalence of 19% was within the range of those found in the surveys of other populations in Addis Ababa. A study in female sex workers in Addis Ababa showed 25% seropositivity for HIV antibody (Mengistu et al., 1990b) which is higher than the rate found for our student sample. Long distance truck drivers in Addis Ababa were 17% seropositive (Mengistu et al., 1990c), a value slightly lower than the rate in our sample. Previous survey has shown that patients with STDs in Addis Ababa are 12% seropositive for HIV (Hailu et al., 1991) while 7% of TB patients were positive for HIV (Hailu et al., 1990b).

In our findings, the seroprevalence of HIV antibodies among highschool students with STDs which was 22% and among students without an STD which was 14% were also very close to the above mentioned seroprevalences except that the rate for college students was only 4%. All these indicate that the virus is spreading at a high rate among the different groups studied so far. By contrast, on the basis of available data, it is estimated that the cumulative number of HIV infected people in Ethiopia will increase to over 500,000 by the year 1992 (Khodakevich et al., 1990b) which is

equivalent to a seroprevalence rate of 1% in a population of 50 million. This indicates that the seroprevalence rates in this and other studies shown above were higher probably because of the high risk behaviours manifested in the sampled groups and because the 1% includes non-adults in the denominator.

In this study, most of the recognized risk behaviours for HIV infection were found to exist among the student population at a very high degree which reveals the main reason for the results obtained. Multipartner sexual contacts (MPSCs), contacts with prostitutes, lack of condom use, past STD history and treatment for STDs at home did exist among these students in high levels. Various other works suggested that many students engage themselves in behaviours that could place them at risk of HIV infection due to their typical adolescent behaviour of experimenting sexuality. A survey conducted on senior highschool students in Addis Ababa indicated that 38% of the students experienced behaviours such as MPSCs and unprotected sex which are detrimental to young adults and which make them vulnerable to HIV infection (Solomon, 1990). Similar findings were obtained among secondary school students in cameroon (Mafany et al., 1990) and in the USA (CDC, 1990). College students also are often viewed as being at high risk for HIV infection due to their exploratory sexual behaviour and their need for peer social approval, and their sense of invulnerability (Hernandez and Smith, 1990). A study conducted on the prevalence of HIV among university students suggested that the use of alcohol

and other drugs by students impairs Judgment and may lead them the unsafe sexual behaviour (Gayle et al., 1990). These may also hold true to our student population in Addis Ababa.

The fact that there was a high HIV seroprevalence among students who claimed to be beginners of sexual intercourse than the frequenters may be because most beginners admitted most of their beginning sexual activities did so with prostitutes, which are the major risk groups of HIV infection, while most of the frequenters practiced sex mostly with other girls than prostitutes. In this study, lack of condom use by sexual partners of female students was associated with higher HIV infection rate than that found in male students reporting lack of condom use. Among the male students who reported frequent use of condoms, 9% were seropositive for HIV. This suggests that the use of condoms may be partially protective in the transmission of HIV or infections might have occurred prior to beginning of condom use. Similar suggestions were also put forward by other workers (Conant et al., 1986; Mann et al., 1988).

Among the students that acknowledged taking self prescribed oral medications for the treatment of STDs, 17% were HIV seropositive. While the significance of this factor in HIV infection is not clear for males, the relationship between HIV seropositivity and taking oral medications in females was suggested to be due to the effect of these drugs on the normal vaginal flora and cervical competency with increased exposure of the vaginal

endocervical canal, which is known to result in increased susceptibility to other STDs (Mann et al., 1988). The treatment of STD by injection at clinics was connected with a similar seroprevalence rate with that of taking oral medications indicating that health centers and clinics in Addis Ababa may need improvements in inforcing precautions and protective measures in their laboratories. The 25% seropositivity among students who took injections at their homes may be suggestive of how such practices may make individuals vulnerable to HIV infection as unsterilized needles or needles not well sterilized may be in use.

Our data showed that most of the HIV infections were between the ages of 15 and 22. Among the samples of students without STD manifestations, older students were infected more. This pattern is consistent with the result of another study (Gayle et al., 1990). On the basis of surveillance data at the national AIDS control programme, more than 50% of all cases of AIDS in Ethiopia are diagnosed in people between the ages of 15 and 29 (MOH, 1991). Eventhough most of the HIV infected samples of students with STD cases are in the same age group with that of the non-STD cases, younger students were infected more than older students among the STD cases probably because most of the STD cases were observed in this age group and the risk behaviours that made them vulnerable to STDs might have played a role in exposing them to the risk of HIV infection.

The higher seroprevalence of HIV among female students was not unexpected because heterosexual contact is the main route of HIV transmission in Africa (Brun-Vezinet, 1985) and in a heterosexual contact females are at a greater risk of getting the infection than males (Anderson and May, 1988). In countries where the majority of the AIDS cases have occurred among men with homosexual and bisexual behaviours and through intravenous drug use, higher seroprevalence is expected among males (MMWR, 1989). Inspite of the risk behaviour seen in both highschool and college students, our data have indicated that highschool students are at a greater risk than college students. This correlates well with the fact that most of the risk factors including current STD diagnoses were among highschool students sampled in the study.

Although our questionnaire did not consider the socioeconomic condition of the students who participated in the study, it is known that patients who usually attend government health centers in Addis Ababa are those who seek free examination and treatment services due to their low income. However, some students from high income families may attend government health centers to obtain treatment so as to avoid the knowledge of their parents that they are suffering from STDs. The opinion of the medical directors in various government health centers has confirmed these.

As for all interview-dependent assessments, specially on sensitive matters such as sexual behaviour, the accuracy of the answers in this study are to be viewed with caution. In one of the health centers where maximum cooperation was obtained, all the questions were asked by the medical doctor to whom the patients are not expected to give unreliable answers. In the other health centers, the questionnaires were handled by the investigator, by medical assistants, and by the laboratory technicians who were able to give some idea about the reliability of the answers and who suggested that some hesitant responses given concerning the frequency of sexual contacts, the number of sex partners and STD history might be unreliable. According to Mann et al., (1988), unreliable information could result in lack of significant differences from the sample population for a given variable. Therefore, we may attribute the lack of significant differences in the analysis of the above risk factors for HIV infection in this study, to some unreliable answers which might have been given by some students.

In this study we found significant associations between HIV infection and syphilis; HIV and chancroid. Since the two STDs are genital ulcer diseases (GUDs) the correlation is to be expected. The associations were calculated among the total student population and among the population of students with STD only. Within the STD cases associations were evident among total female STD cases and total highschool male and female STD cases. Gonorrhea showed a

significant result among the total population of students with STD and among total female STD cases. The presence of association between HIV and a GUD in the total sample and the lack of such an association within a given subsample as seen in our result may be due to the small sample size and due to differences in the confounding variables which may affect the associations differently.

Several works have shown associations between HIV and syphilis (Handsfield et al., 1987; Quinn et al., 1987; Simonsen et al., 1988), between HIV and chancroid (Greenblatt et al., 1987; Simonsen et al., 1988; Pepin et al., 1989) and a weak association between HIV positivity and gonorrhea (Kreiss et al., 1986; Laga et al., 1990). The reasons for relation of HIV infection to the occurrence of these STDs have also been hypothesized by different workers. GUDs have been implicated in greatly increasing the infectivity of seropositive people due to the disruption of integrity of the mucosal epithelium which allows easier penetration by the virus (Kreiss et al., 1985) and cervical ectopy, which is a normal physiologic event in the female genital tract and which is known to increase the risk of gonoccocal infection is said to increase the susceptibility of women to HIV infection (Laga et al., 1990; Moss et al., 1990). Simonson et al. (1988), suggested a causal association of HIV and genital ulcers. He explained that genital ulcers could alter a man's susceptibility to HIV by providing a portal of entry for the virus and their occurrence in females

infected with HIV could render the females more infectious; thus, cotransmission of HIV and GUDs could explain the association. These possible explanations also reveal that the above discussed STDs might have facilitated heterosexual transmission of HIV in our student population and show that heterosexual intercourse might have been the only likely route by which the students acquired HIV infection.

The influence of confounding variables was indicated by stratifying the data for some risk variables. Analysis for syphilis showed that the association between HIV and syphilis in the whole student population arose because the confounding factors such as amount of sexual activity, condom use and a history of STDs were risk factors for both syphilis and HIV infection. In this work the confounding effects of each variable were not further stratified and the analysis not conducted. Even if it is rarely possible to measure confounding factors so precisely and is not possible to remove them entirely (Mertens et al., 1990), the need for detailed further studies is obvious.

This study has certain limitations. First, the survey was not a random sample of all highschools and colleges in Addis Ababa but it was based on presenting samples of students. These presenting samples who attended health centers and have blood drawn may not be representative of all students in Addis Ababa. In addition, the cross-sectional study by which we investigated the association

between HIV and STDs is not capable of determining the sequence of events.

Finally, from the results obtained in this study, the following recommendations may be put forward:

1. The results obtained in this study point out the need for preventing HIV infection through education by providing effective AIDS awareness opportunities at secondary schools and colleges. Therefore, effort in health education should include training instructors to include preventive measures against the disease as part of their subject matter and the establishment of an interdisciplinary health committee in schools and colleges for the prevention and control of AIDS by providing educational programmes focusing on behaviour modification.

2. Chancroid and syphilis were found to predispose to heterosexual transmission of HIV among students. Therefore, the control of these diseases could prove to be an important focus of intervention. More extensive programmes for control of these STDs among the population are recommendable.

3. Every attempt should be made to prevent the spread of HIV by non-sexual routes such as parenteral

transmission which occurrs via reuse of inadequately sterilized infection needles at some health centers that lack adequate medical resources. Therefore, institutionalizing of more precautious and preventive procedures at health centers is recommended.

4. Studies on knowledge, attitude, practice and behaviour of students for appropriate assessment and evaluation of methods of intervention in HIV prevention and control are recommended. Efforts in this area could be instrumental in overcoming persisting problems in the selection and application of appropriate and adequate strategies in the monitoring and evaluation of AIDS health education programmes.

5. A more detailed and comprehensive study on a large sample of students of highschools and colleges which is representative of all students is recommended for the assessment of the prevalence of HIV in this population.

ANNEX I

QUESTIONNAIRE

1. Study number _____

2. Clinic _____

3. Age _____ / / High School / / College

4. Sex Female / / Male / /

5. Ethnic _____

6. Religion _____

7. Marrital Status / / Single / / Married / / Divorced

8. Complaint in the last twelve monthes / / Yes / / No

   Symptoms / / Fever / / diarrhoea / / cough

   / / wt. loss others _____

9. Present complaints / / fever / / diarrhoea / / cough

   wt. loss / / others _____

10. Blood transfusion in the past or at present

    / / yes / / No year _____

11. Blood donation in the past or at present

    / / yes / / No Year _____

12. Have you ever practiced sexual intercourse?

    / / yes, so many times

    / / yes, beginner

    / / No, not at all

13. Sexual contact with more than one partners

    / / yes / / No

14. Sexual contact with a prostitute

/ / yes / / No

15. Do you or your partner(s) use condom?

/ / yes, always

/ / yes, sometimes

/ / rarely

/ / no, not at all

16. STD in the past five years

/ / yes / / No

17. If yes, which STD? How many times?

/ / gonorrhoea _____

/ / Syphilis _____

/ / Chancroid _____

/ / other, specify

18. How was it treated?

/ / injection at clinic

/ / injection at home

/ / antibiotics prescribed by self or a friend

19. STD current diagnosis _____

ANNEX II

STATISTICAL FORMULAE

1/ 95% Confidence Limits for Sample Proportions:

[P-1.96*SQR(P(1-P)/N) , P+1.96*SQR(P(1-P)/N)]

Where , P = proportion

N = Sample size

SQR = Square root

2/ The Chi-square ($\underline{X}^2$) test :

using a general 2x2 contingency table:-

	I	II	Total
A	a	b	n_A
B	c	d	n_B
Total	n_1	n_2	n

$$X^2 = \frac{n(ad - bc)^2}{n_1 n_2 n_A n_B}$$

Where,

A= Exposure +
B= Exposure -
I= Infection +
II= Infection -
n_1= a+c
n_2= b+d
n_A= a+b
n_B= c+d
n= a+b+c+d

3. Yates correction is made when the number of degree of freedom is one.

$$X^2 \text{ (corrected)} = \frac{n(/ad-bc/-0.5n)^2}{n_1 n_2 n_A n_B}$$

4. Odds ratio calculated based on the above 2x2 table:

$$\text{Odds Ratio} = \frac{ad}{bc}$$

5. Confidence limit for the odds ratio:

[OR - 1.96 SQR (1/a+1/b+1/c+1/d) , OR + 1.96 SQR (1/a+1/b+1/c+1/d)]

6. Fisher's exact test :
In this test the P value is calculated directly when ever the expected freqency in any of the cells in a 2x2 table falls below 5.

$$P = \frac{n_1!n_2!n_A!n_B!}{n!a!b!c!d!}$$

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