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The Demography of HIV/AIDS

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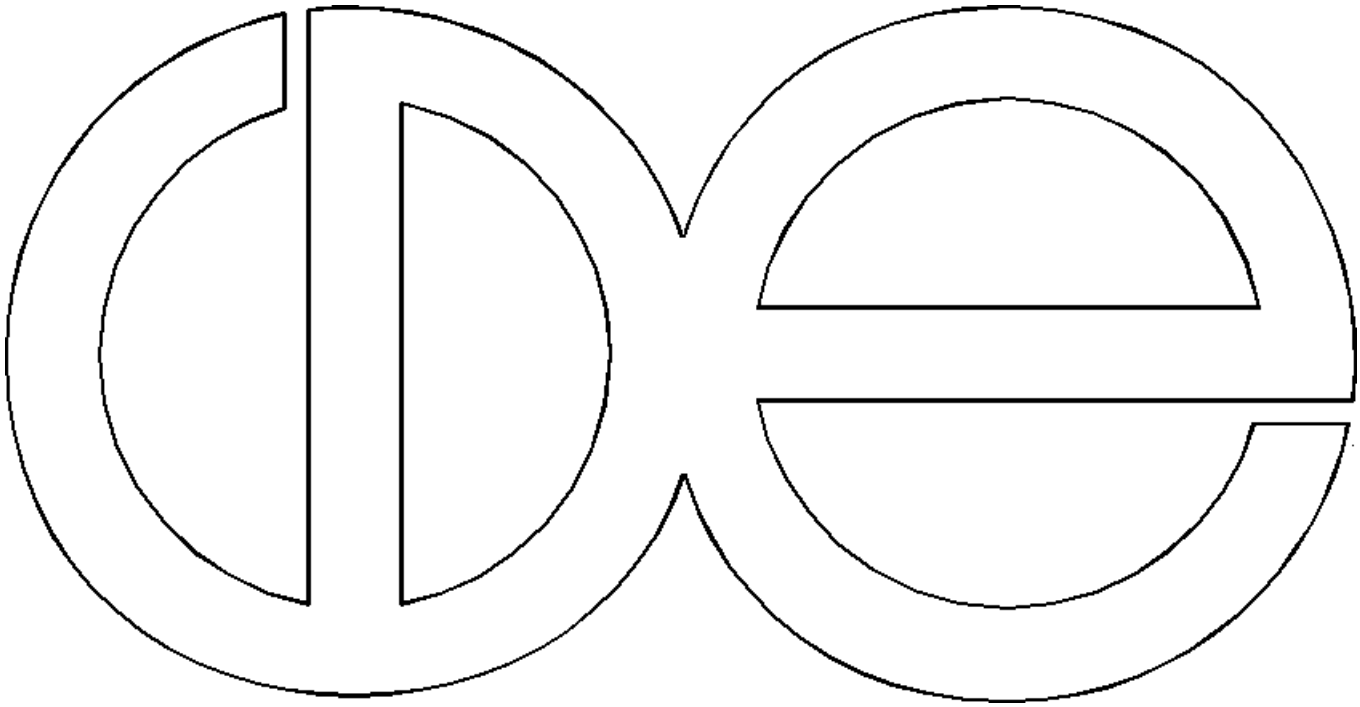

Demography of HIV/AIDS

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I. PRELIMINARIES: THE IMPACT OF HIV/AIDS ON POPULATION TRENDS AND ON DEMOGRAPHIC RESEARCH.

Approximately fourteen years ago HIV was first identified among homosexuals in the United States and Europe and soon thereafter among heterosexuals in Africa and a handful of countries in the Caribbean basin (Essex, 1994). Since then, the HIV/AIDS epidemic has spread first to the Caribbean and to South and Central America and then to Eastern Europe and to Southeast and Northeast Asia. It has left a trail of increasing numbers of deaths due to AIDS and related conditions and increased prevalence of HIV among adults and children of both sexes. Although the nature and magnitude of the problem varies according to which mode of transmission predominates, the overall effects of HIV/AIDS are of sizeable magnitude. A calculus of the total impact associated with the epidemic does not just involve the death toll directly attributable to AIDS. It also includes costs associated with increased demand for intensive health care, utilization of resources to subsidize campaigns to contain the epidemic, and the foregone value of labor inputs of infected individuals in the most productive age groups. Although these effects are neither directly visible in current morbidity and death statistics nor can they be inferred transparently from readily available data, they will surely stand out when all the accounting on the epidemic is closed and done with. Concealed from view, partly because they involve complicated lags and partly because of the ethereal quality we confer to events spread out over several generations, are intangibles such as massive disruption of family life, family organization and socialization, decline in school attendance, increased child labor force participation, and erosion of social support networks, particularly those erected to protect the elderly and the very young. In developing countries at least, the HIV/AIDS epidemic has joined forces with the economic recession of the seventies and eighties to seriously compromise the living conditions of future generations (Nicoll et al., 1994). Thus, the demography of HIV/AIDS provides very good grounds for a public image of the epidemic entirely forged from its pure negative aspects.

But hidden from view are endeavors and achievements of unprecedented dimensions. First and foremost, the identification of AIDS's aetiological agent, the Human Immunodeficiency Virus (HIV-1), and the mapping of its genetic properties must stand out as one of the most resounding successes in the annals of medical science. Although a cure or vaccine has yet to be found, the rapid pace at which knowledge about the dynamic of the disease accumulated is without precedent. Second, the establishment of large-scale efforts to mobilize the scientific community, national governments, international agencies, public health officials and, ultimately the broader public uniquely underscores how collective action organized around special interest groups and supported by centralized interventions fosters progress in public health. Third, social scientists and epidemiologists alike have ventured into areas of research that would have remained underdeveloped had it not been for the sense of urgency induced by the advancement of the epidemic. Thus, for example, research on patterns of sexual behavior, an area within the social sciences whose development has, for many years, lagged behind others, has acquired renewed impetus albeit in the midst of a great deal of controversy and not without political backlash (Laumann et al., 1994a; Carael et al., 1992). Demographers have become involved in a flurry of activities that span a remarkably broad spectrum of areas. This has occurred in response to renewed demand for short term projections of the impact of the epidemic and, more generally, as a result of the increased need for application of formal tools to represent population dynamics in the presence of HIV/AIDS. In the process of satisfying these new needs and through the use of conventional surveys, aggregate statistics and ethnographic research, intriguing regularities in the spread of the epidemic have been discovered. These regularities point to connections between patterns of spread of HIV/AIDS and social, economic, and cultural conditions that demographers and sociologists have studied for quite some time to explain fertility and mortality change. There is a growing sense that our accumulated knowledge can translate into relevant insights about the nature of HIV/AIDS patterns.

Lastly, from the point of view of demographic research alone, an important achievement is one whose potential is yet to be fully realized, namely, the strengthened interaction between the formal representation of the spread of the epidemic (modelling), on the one hand, and basic research—ethnographic and survey-based—that sheds light on behavioral assumptions underpinning formal models, on the other. The strong connections between these two normally divorced scientific activities has been recently highlighted by Anderson, one of the pioneers in the development of epidemiological models of HIV/AIDS, in a volume devoted to the exploration of sexual behavior and networking (Anderson, 1992).

This paper has two goals. First, it provides an account of the current state of the HIV/AIDS epidemic. I assign special emphasis to conditions found in the developing world (mainly Africa, South and Central America, the Caribbean and Southeast Asia) since the bulk of yearly new HIV/AIDS cases originate there and since these are regions where the effects of the epidemic are likely to be most devastating. I then evaluate forecasts and projections made during the past ten to fifteen years and, with the benefit of hindsight, assess their accuracy. I show that the record of short and long term projections is, on the whole, unsatisfactory. Since projections and forecasts represent in a condensed form the state of our knowledge about the epidemic, it behooves us to identify factors that account for their weaknesses. This leads to a second goal, namely the analysis of the relation between known characteristics of the epidemic on the one hand, and properties of the demographic and epidemiological models to represent the spread of the virus on the other. I identify the models' fault lines, along which formalization rests on fragile behavioral and epidemiological assumptions and where the impact of data collection efforts either via conventional surveys or with instruments of qualitative research are likely to have large payoffs.

The remainder of this paper is in four sections. In Section II I summarize the main characteristics of HIV and AIDS. In section III I review trends and describe the demographic profile

of HIV/AIDS in the world. This section is based on reported AIDS cases (adjusted for under-registration and delays in reporting), on syntheses of assorted seroprevalence surveys, and on a data bank maintained and organized by the International Office of the US Census Bureau. An important theme is the pattern of spread of HIV/AIDS from high risk groups to the general population and the different modalities this pattern acquires according to the transmission mechanism that dominates in a society. Section IV evaluates selected projection models and compares forecasts and projections with observed trends during a period of approximately twelve years. Section V summarizes the nature of mathematical models for the spread of HIV/AIDS and identifies linkages to conventional demographic models, formulates conditions for their fruitful application, and suggests areas where interdisciplinary research could have sizeable returns.

II. DEMOGRAPHIC AND EPIDEMIOLOGICAL FEATURES OF HIV/AIDS

a. The nature of HIV/AIDS.

AIDS results in a gradual and progressive impairment of the human immune system and the emergence and recurrence of a variety of infections and neoplasms. HIV (also known as HIV-1), the causative agent of AIDS, is a virus belonging to a family of retroviruses and a member of a class of viruses with genetical commonalities that induce breakdowns in the immune system of humans and simians. HIV was identified independently in 1983-84 by US and French scientists (Essex, 1994).

The transmission mechanisms of HIV are known to be fairly limited. These include: vaginal and anal intercourse with infected partners, injections and intravenous inoculation with contaminated needles or syringes, administration of infected blood or blood products, and perinatal transmission from mother to child (Curran, 1985; Friedland and Klein, 1987; Essex, 1994). The efficiency of viral transmission ('infectivity') is known to be highest for blood transfusion with contaminated blood and lowest for needle punctures. Infectivity of heterosexual intercourse with an infected partner is

intermediate between these two extremes but subject to high variance. A host of factors induce variability in the efficiency of infectivity associated with any transmission mechanisms. Thus, different techniques for storing blood products and different practices in the application of injections (intravenous versus intramuscular) lead to different relative risks associated with each mechanism (Francis and Quinn, 1994). Similarly, infectivity of sexual contact with an infected partner is highly variable as it depends on type of sexual intercourse (Dallabetta et al., 1990; Nzila et al., 1991; Piot et al., 1990; Piot et al., 1994), the duration of infection of the infected partner (Anderson, 1992), presence of other sexually transmitted diseases (STD), (Plummer et al., 1994) and lack of circumcision (Cook et al., 1994; Bongaarts et al., 1989). The conditional probability of being infected after receiving a blood transfusion with infected blood is estimated to be in the range of .70-1.00, whereas estimates of the probabilities of perinatal transmission range between .20 and .40 and varies widely by geographic region with Africa falling in the upper part of the range and more developed countries in the lower part (Piot et al., 1994). More uncertainty clouds the known estimates of the conditional probability of infection through sexual intercourse (otherwise known as rate of infectivity) which ranges anywhere from 1 in 1,000 to 1 in 10 per sex act per partner.

In addition to the rather limited repertoire of transmission mechanisms, HIV is characterized by a highly variable but normally quite long incubation period, the time elapsed between infection to the first appearances of AIDS symptoms. Although reliable estimates are hard to come by and those that exist change gradually as the epidemic progresses, it is believed that the median incubation time may surpass ten years (Moss and Bacchetti, 1989; Ryder and Mugerwa, 1994). This is again variable and depends on the mode of transmission, degree of exposure to other diseases and characteristics of the host, including age, health status and genetic make-up. In light of these considerations it should not be surprising to find regional variation in the distribution of incubation times. Since incubation is fairly long, individuals who are infected are not easily distinguished from susceptibles and infection

can be transmitted more readily from one to the other and over longer periods of time per infected person. Unlike other apparently new viruses such as the so-called Ebola, HIV is an 'intelligent' virus that conceals itself in the body of the host thus substantially increasing its chances of survival and reproduction.[2] If HIV led to AIDS within a very short time interval, infected individuals would, in theory at least, be easily identified shortly after infection and the number of secondary cases caused by each infected individual would be readily minimized.[3] A second dimension of the incubation process is the proportion of infected individuals who eventually develop AIDS. Although the evidence on this score is somewhat murky, it is believed that the majority (over 90 percent) of those who become infected with HIV will eventually develop AIDS.

A final characteristic of HIV/AIDS is its high lethality. Available evidence indicates that virtually all individuals who contract the virus and develop AIDS will die within a period no longer than three years after the onset of AIDS and that their deaths will be due to some of the ailments brought about by the collapse of the immune system. The causes of deaths most commonly associated with AIDS are fairly reduced but quite variable across geographic regions. Although uniformly high, the force of mortality associated with AIDS varies by age, social and economic condition and, finally, is affected by the use of medications such as AZT which retard or inhibit the full-fledged reproduction of the virus and the consequent development of symptoms.

b. The interaction of HIV and other diseases.

Although the demographic impact of HIV/AIDS and its imprints on various social groups and age categories across regions depends strongly on the relative dominance of major modes of transmission and on the nature of contacts between high and low risk groups, it is also influenced to a non-trivial extent by the interaction of HIV with other diseases. This is an important dimension of the spread and consequences of HIV/AIDS since it modulates the effects it has on human populations. Any attempt to understand the spread of HIV in different regions of the world requires

at least some knowledge of the prevalence of other diseases and of the particular ways those diseases interact with HIV and AIDS.

The relation between HIV and other diseases involves two separate issues. The first is the role played by other diseases in enhancing (inhibiting) the **infectivity** of HIV and, conversely, of the role played by HIV to increase (reduce) the chances of acquiring other diseases. At least part of the heterogeneity in levels of prevalence within Sub-Saharan Africa, for example, is due to difference in the presence (absence) of illnesses that enhance (inhibit) infectivity of HIV. The second issue is the effect that other diseases may have on the **progression** of HIV toward AIDS, that is, on the timing and degree of health impairment associated withthe transition from HIV to AIDS. This is important, for the shorter the incubation period the morecontainable becomes the transmission of the infectious agent.

b.1. HIV and other diseases: infectivity of HIV.

It is widely suspected that there is an association between HIV and at least some Sexually Transmitted Diseases (STD's). A first macro-level illustration of this relation can be obtained by examining the association of indirect measures of STD prevalence such as sterility and HIV prevalence. In a recent study, Frank (1992) shows that there is a relation, albeit somewhat weak, between levels of infertility and HIV prevalence in Sub-Saharan Africa. Since infertility is good proxy for prevalence of some STD's but particularly gonorrhea, this association is **prima facie** evidence of an association between the two. The difficulty is that the relation could reflect the operation of multiple and disparate causal mechanisms. The first and most obvious mechanism is that prevalence of STD and HIV may be spuriously associated: to the extent that both diseases advance and spread in social groups where certain types of sexual behaviors are more prevalent than others, one would expect the association to be tight. If so, the observed association between STD's and HIV is not in itself relevant as a condition for the spread of HIV. A secondmechanism is one whereby the presence

of STD's may enhance HIV infectivity of infected individuals or may exaggerate the susceptibility of susceptible individuals (Piot et al., 1994). This is more likely to occur with STD's such as chancroid, genital herpes and syphilis, all of which produce genital ulcers or other lesions that enhance tissue exposure to genital secretions or viral shedding. Conversely, HIV appears to lengthen and magnify the infectiousness of these STD's (Wasserheit, 1992; Plummer et al, 1994). Since GUD (Genital Ulcer Diseases) are less influential factors of permanent sterility than gonorrhea, there is no reason to expect that this mechanism will result in a strong relation between HIV and infertility. But if the mechanism operates, the association between STD's and HIV will influence in important ways the trajectory of the HIV epidemic.

Finally, increased susceptibility to STD's and the consequent growth of STD prevalence may simply be the by- product of immunosuppression that results from the assault of HIV on the immune system (Plummer et al., 1994). This again is a mechanism that should neither slow-down nor accelerate the dynamic of spread of HIV itself although it will definitely magnify its broader health implications.

In addition to the aggregate relation between infertility and HIV prevalence, a review of studies investigating the synergism between HIV and STD's corroborates that at least the prevalence of GUD enhances HIV infectivity. But these studies confirm only weakly the operation of the other two mechanisms (Clemetson et el, 1993; Plummer et al., 1994).

A different type of supportive evidence indirectly relating STD's and HIV/AIDS originates in studies that uncover a negative relation between prevalence of circumcision and of HIV (Bongaarts, et al., 1989; Cameron et al., 1989; Moses et al., 1990; Caldwell and Caldwell, 1994; Cook et al., 1994). Although there are single studies where the relation has not been conclusively found (Borgdorff et al., 1991) and there is ample room for artifacts and spurious relations, it is suspected that this association may, as does the association of HIV with STD's, reflect diminished

viral shedding and/or less damaging exposure to genital secretions among circumcised than among uncircumcised men (Cook et al., 1994). This would lead to both reduced infectivity of contacts between an infected man and a susceptible woman and reduced susceptibility of a susceptible man in contact with infected women. Of course, it is possible that circumcision is reflective of cultural and social patterns that independently minimize exposure to HIV through, for example, reduction in non-marital sexual relations. In a recent factor-by-factorreview of various alternative explanations for the relation, the authors conclude that this is unlikely and thatthe relation between HIV and circumcision reflects the existence of direct protective mechanisms (Caldwell and Caldwell, 1994).

If it were the case that the relation between STD's and HIV is indeed mediated by increased HIV infectivity (or susceptibility) and that STD's are a co-factor in the spread of HIV, a reduction of STD's should attenuate the speed of transmission of HIV in high riskgroups and between high risk and low risk groups by lowering the rate of HIV infectivity. We show later that interventions designed to alter infectivity by manipulating co-factors suchas STD's are effective means of slowing-down and altering the patterns of spread of the disease. By the same token, differential prevalence of co-factors may explain differential levels of HIV prevalence in regions experiencing otherwise similar conditions.

b.2. HIV and other diseases: progression to AIDS.

An intriguing question that remains poorly investigated is whether or not the progression of HIV to AIDS is altered at all by the presence of other diseases. I emphasized above that HIV is an intelligent virus since it is able to remain concealed thus lengthening the period of time during which it can be transmitted from one person to others. It follows that the dynamic of HIV transmission could be sharply altered if other diseases accelerate (or decelerate) the time during which the virus remains inactive. If, on the other hand, immunosuppression associated with HIV were to increase susceptibility and lower resistance to or decrease the capacity to recover from other diseases, the

impact of HIV on morbidity (and its ultimate effects on social and economic outcomes) would be considerably magnified **but the dynamic of HIV spread should not be altered**.

There are four groups of diseases that may affect HIV progression to AIDS. The first group is again constituted of STD's. Circumstantial evidence suggests that individuals with STD's experience shorter progression to AIDS (Piot and Colebunders, 1987; Quinn et al., 1987; N'Galy et al., 1988). In one study of prostitutes diagnosed with STD the median incubation period was as short as 34 months (Anzala, et al., 1991). A second group of diseases are caused by HTLV-1 (Human T-cell lymphotropic virus type I), the first human retrovirus to be identified. These viruses are present throughout Africa and their prevalence is highly correlated with the prevalence of HIV (Verdier et al., 1994). **In vitro** observations suggesting that HTLV tends to activate HIV and vice versa and are confirmed by clinical studies that show sharp reductions in the incubation times of HIV among individuals co-infected with HTLV (Chen and Sankale, 1994). Thus, for example, a study in Trinidad shows that the median incubation time among homosexuals who were infected with both HIV and HTLV was as short as three years whereas among those infected only with HIV less than 9 percent developed AIDS during the same period of time (De Rossi, 1991).

Perhaps the most ominous indication of disease interaction is between HIV and respiratory tuberculosis (TB). There is no question that the incidence of clinical TB has been increasing in the last ten years and has been doing so more significantly in areas where HIV is more prevalent (Sudre et al., 1992). Annual notification of TB cases have increased everywhere but particularly in Africa and Latin America, two regions of the world where TB prevalence has been traditionally high (Sudre et al., 1992). Since TB is also highly prevalent in Southeast and Northeast Asia, where roughly 20-30 percent of the adult population is (clinically and subclinicaly) infected, the spread of HIV may trigger there too an increase in notifiable TB cases.

It is known that people infected with HIV are several times more likely to develop clinical TB

and that this occurs as HIV reactivates latent infections with **Mycobacterium Tuberculosis** (MT) (rather than the latter affecting the chances of infection with HIV). It has been known for years that the reactivation of MT occurs when the immune system is under severe stress (Lunn, 1991) and hence it should not be surprising to verify that infection with HIV and the immunosuppression that follows lead to a resurgence of TB cases. Unlike less virulent opportunistic infections triggered by HIV, such as Toxoplasmosis and Pneumocystis Carinii, TB sets in early and has a high potential of being transmitted to other people who are neither infected with HIV or MT. The importance of this fact is two-fold. First, in areas where TB is prevalent (even subclinically or in latent form), individuals infected with HIV are at higher risk of reactivation and therefore more likely to experience longer periods of disease and disability while they are infected than individuals infected with HIV in areas where TB is not prevalent. Paradoxically, this could set up a perverse beneficial feedback for the population as a whole: in poor populations with low levels of nutrition the effect of TB recrudescence could accelerate progression to AIDS thus **decreasing** incubation times and therefore potentially reducing transmission opportunities. Second, reactivation of MT in HIV infected individuals will increase the degree of spread of MT in the general population. This secondary epidemic is likely to be aggravated by reportedly increasing resistance of MT to conventional antibiotic therapy (Pablos-Mendez et al., 1990; Snider and Roper, 1992) and by the faltering success of campaigns to apply chemotherapy among those with active infections (Brudney and Dobkin, 1991). In any case, due to its relation to MT, the threat that HIV presents in the larger population is by no means confined to those who are sexually active or otherwise at risk of contracting HIV.

Malaria is the fourth illness suspected to interact with HIV, but, despite the existence of early evidence supporting mutual activation, the conjecture has never been conclusively proven. Since malarial antigens trigger proliferation of T lymphocytes and reduce the CD4 lymphocyte count, there is reason to believe that infection with the malaria parasite has at least the potential to alter the course

of HIV or, conversely, that HIV immunosuppression may worsen the course of malaria (Smith et al., 1988; Morrow et al., 1989). Since malaria is so prevalent in some areas heavily affected by HIV (Africa and to a lesser extent Central America), their interaction could have important consequences for the health status of HIV-positive individuals. As in the case of TB, the interaction with malaria not only magnifies HIV's impact on the health of populations but could also alter the dynamics of transmission by modifying the progression from infection to AIDS.

III. THE SPREAD OF HIV/AIDS AND ITS DEMOGRAPHIC EFFECTS

a. Patterns and stages of the epidemic.

The relatively short history of the spread of HIV/AIDS is punctuated by stages which result from shifting combinations of patterns of infection as the virus overtakes different and at times overlapping subpopulations. The patterns of HIV/AIDS can be characterized along three possibly dependent dimensions: age and sex profile of the infected population, distribution of cases by mode of transmission, and spatial distribution. The stages highlighted below reflect a blend of patterns with a unique distribution of cases by age, sex and rural-urban concentration and a unique rate of growth of the epidemic. The **first** stage of the epidemic occurs before 1978. It is characterized by the superposition of two radically different patterns. One pattern prevailing in North America, Western Europe and parts of Oceania (Australia and New Zealand) exhibits a disproportionate contribution of infections due to homosexual contacts, intravenous drug use (IV) and blood transfusions. The age and sex profile of this pattern of infection is marked by a very high ratio of male-to-female cases, a high predominance of infections among the young and middle-aged persons with only a small proportion of cases among infants and children,and a marked urban concentration. A second pattern of infection becomes established in Sub-Saharan Africawhere HIV spreads more generally among heterosexual groups but with strong clustering within main cities, large commercial centers, and along

principal routes of transportation. The age and sex profile of the epidemic displays a male-to-female ratio of infections close to one with a sizeable fraction of cases among infants and young children. Infections among infants and children are the joint outcome of blood transfusions—widely encouraged by regional medical practices to alleviate the symptoms of malaria and anaemia—and, more importantly, of perinatal transmission from mother to child.

A second stage of the worldwide epidemic begins with the appearance of a third pattern of infection in the Caribbean and shortly thereafter, in a modified form, in South and Central America. It is characterized by a combination of concentration of cases among intravenous drug users and homosexuals, on the one hand, and bisexual men and heterosexual individuals, on the other. The sex ratio of new cases of HIV is of magnitude roughly intermediate between those in Africa and North America, Western Europe and Oceania and so is the prevalence of infection among infants and young children. During this stage, the growth of new cases in Western Europe, North America and Oceania begins to slow down as the infection saturates the highest risk groups. At least judging by the sheer relative contribution to the total new AIDS cases, the African pattern continues to predominate throughout this stage.

HIV/AIDS becomes a global phenomenon in a third stage. This is marked by the onset and rapid propagation of HIV in the large metropolis of Southeast and Northeast Asia and, at least for now, to a lesser extent Eastern Europe. Although HIV continues to heavily affect populations of homosexuals, particularly in Eastern Europe, the bulk of new cases are due to heterosexual transmission, mother-to-child transmission, and intravenous drug use. In Southeast Asia the epidemic grows at very high rates with an age-sex profile closer to that found in Sub-Saharan Africa but with the added contribution of a hefty component of transmission via IV drug use.

To illustrate the features discussed above, I first rely on examination of regional patterns of HIV/AIDS. To bolster comparability I adopt, not without some misgivings, the regional

differentiation proposed by Mann and colleagues based on the so-called General Affinity Areas (GAA) (Mann et al., 1992). As I point out later, this regionalization overlooks and conceals important within-region heterogeneity. Some of the main features of the epidemic are illustrated in Table 1 and the various panels of Table 2. Table 1 is a synthesis of the three stages of the epidemic and of the mixture of patterns corresponding to each one of them. It summarizes some features of the global spread of HIV/AIDS and highlights the presence of different sub-epidemics emerging in the regions at different times. Thus, in North America, Western Europe and Oceania, the epidemic preserves the characteristic form of spread established at onset, e.g. it is predominantly urban and concentrated among homosexuals and intravenous drug users. Similarly in Africa (especially Sub-Saharan Africa) HIV persists as a disease affecting the urban and occasionally the rural heterosexual population. In contrast, in other regions such as the Caribbean, South and Central America and, to a lesser extent Southeast Asia, the epidemic is more fluid and advances through a mixture of patterns successively encompassing homosexuals, bisexuals, heterosexuals and IV drug users. The result of these contrasting patterns, their geographic distributions and successive shifts overtime is a fair amount of regional heterogeneity in the characteristic profiles of the epidemic and, more importantly, a substantial amount of dissimilarity in the consequences of the infection for the population at large.

Table 2 (panel a) provides information about the comparative history of regional epidemics. The first three columns of the table display the total cumulative number of AIDS cases (in 1,000) for a very early period (1979-84), for 1990, and for 1993, the year with the most recent and complete information. The fourth and fifth columns show total yearly incidence AIDS rates for the period 1985-1990 and for the most recent period with complete data, 1990-1993. The next four columns of the table show estimated quantities only.[4] These columns display, respectively, cumulative number of HIV cases for 1985 and 1995, the rate of HIV incidence for 1985 and 1995, and the estimated doubling time for each HIV epidemic. In all cases the estimates are mean values of a range obtained

through the application of alternative estimation procedures. The numbers appearing in round parentheses are the standard deviations of the distribution of estimates. This panel confirms the regional heterogeneity expected in light of considerations discussed before (see Table 1). This is particularly true with respect to the relative speed with which the epidemic spreads in various areas. First note that AIDS incidence rates grow everywhere (except Oceania and the Caribbean) but that the ratio of growth is highest in Southeast Asia, Latin America and Sub-Saharan Africa in that order. Second, the estimated growth curve of HIV cases underlying the epidemic proceeds faster in Southeast Asia, Sub-Saharan Africa, Northeast Asia and Latin America. It is noteworthy that the estimated doubling time of about 8 months in Southeast Asia is identical to that Sub-Saharan Africa and very close to that of Latin America. Third, between 1985 and 1995 the estimated HIV incidence **rates** (numbers in square brackets) are on the increase everywhere except in Sub-Saharan Africa and the Caribbean. The proportionate increase in the incidence rates is largest in Latin America and Southeast Asia. Although part of the slow-down observed in Sub-Saharan Africa and the Caribbean may be an artifact of inappropriate modelling and of reporting delays in AIDS cases, it is possible that the epidemic may be temporarily subsiding, particularly in areas where it first emerged, either as a result of saturation of high risk groups or as consequence of behavioral changes. Two features are worthy of notice. First, the epidemic is gathering strength in two very important areas of the world, Latin America and Southeast Asia. Second, the figures confirm the conjecture that in areas where the epidemic grows in social groups with partially overlapping contacts (bisexuals, IV drug users, and heterosexuals) the speed of transmission is faster and the ultimate size of the epidemic is larger.[5] The most immediate consequence of this apparently faster pace is that although the epidemic with the broadest reach continues to be the one in Sub-Saharan Africa, where close to 10 million individuals will have been infected by the end of 1995, those in Latin America and Southeast Asia could reach comparable dimensions. In these countries the epidemic is still young and the worst may be yet to

come.[6]

Panels b and c of Table 2 display estimated ranges of HIV and AIDS prevalence among adults aged 15-49 and children younger than five years of age. Regional differentials in the rates of HIV (and AIDS) prevalence are a function of the timing of introduction of the epidemic and, more importantly, a result of the particular patterns of transmission that dominate in each area. Despite the fact that the estimated ranges for HIV prevalence reflect a fair amount of uncertainty about the past (particularly for Latin America and Sub-Saharan Africa) two features stand out. First, as of 1995 the size of the epidemic, as gauged by the cumulative number of AIDS cases, is largest in Sub-Saharan Africa, Latin America, North America and the Caribbean, in that order. Given the estimates of increasing HIV incidence rates shown in Table 2a we should expect that levels of AIDS and HIV prevalence will continue to mount in all these regions.

Second, between 1985 and 1995 the levels of HIV prevalence have more than doubled everywhere although the most explosive relative growth has occurred in Latin America, Southeast Asia and Sub-Saharan Africa. The overall levels of adult prevalence in 1995 do not exceed 4 percent of the adult population and are highest in Sub-Saharan Africa (about 3.2 per cent) and Latin America (1.7 percent). These are the two regions where prevalence among children younger than 5 are also highest. Since AIDS lethality is high, the estimated ranges for AIDS prevalence (panel c) are significantly lower than the HIV levels of prevalence but, as expected, the rank order of regions according to AIDS prevalence preserves the rank order by HIV prevalence. The relatively low absolute levels of HIV prevalence in the general populations can be misleading since as the epidemic penetrates different social groups very unequally and with markedly different timing, a low **total** level of HIV prevalence may signify near saturation in some social groups. How far the epidemic will reach depends on the nature of contacts between groups where the virus can spread rapidly ('high risk') and the rest of the population ('low risk'). Due to the nature of the dominant means of transmission

(see below) we suspect that the epidemic has a strong potential for future growth in Latin America, Southeast and Northeast Asia and that levels of HIV and AIDS prevalence in these regions will continue to rise in the near future.

b. Impact on mortality levels and patterns.

A population exposed to HIV/AIDS will experience changes in levels and patterns of mortality which are directly related to the prevalence of the infection. What has been the recent impact of the epidemic on mortality levels in the various regions?

Panel d of Table 2 displays estimated proportionate increases in adult and child mortality rates as well as the corresponding decrease in life expectancy at birth and mean number of years of life in adulthood (between 15 and 49) that results **from the experience with the epidemic from 1985 to 1995**. The estimated **ten-year** impact on the 1985-1995 excess deaths due to AIDS is reflected in percentage increases in adult mortality rates (age group 15-49) ranging from about 0 or 1 in Northeast Asia, Southeast Asia and the Southern Mediterranean, where the epidemic is still in its early stages, to as high as 34 and 40 percent in North America and Western Europe. Since levels of adult mortality in the world's most developed areas are very low to start with, even minor absolute increases in mortality translate into proportionately large changes. Excess adult mortality due to HIV/AIDS potentially reduces temporary life expectancy between ages 15 and 49 by as little as .02 years in Northeast Asia and by as much as 2.9 years in Western Europe.

The effects of HIV/AIDS on mortality below age 5 are contained within a range from 0 to about 4 percent and translate into losses in life expectancy at birth ranging from 0 to about 2 years during the ten year period considered in the calculations. Since child mortality is a more influential determinant of life expectancy at birth in areas with high child mortality, lower proportional changes in child mortality induced by HIV/AIDS could end up producing, as they do in Sub-Saharan Africa and Latin America, larger fractional changes in life expectancy at birth.

By and large, the excess deaths associated with HIV/AIDS are due to opportunistic infections and neoplasms that display some heterogeneity across regions. This heterogeneity appears to be linked to the nature of the incubation process, the ecology of other diseases, and the degree of access to specialized medical care.[7]

By all accounts then, the HIV/AIDS epidemic could lead to unprecedented sustained reversals in a century-long process of mortality decline. The pace of these potential losses is quite staggering: in the regions most affected by the epidemic, the combined losses in life expectancy associated with excess child and adult mortality due to HIV/AIDS could reach rates as high as .33 per year, not unlike the rates of **gains** with which some of these countries were able to overcome high levels of mortality prior to the massive adoption of medical knowledge and innovations and the widespread deployment of public health measures. To the extent that these losses may be partially or totally offset by mortality reductions associated with improvements in other illnesses, they should be considered only 'potential losses'. They are more likely to become actual losses in areas with widespread poverty and where health care delivery is difficult and of limited efficacy. Even though most systems of vital statistics are not good enough to confirm it, there are some indications that mortality has begun to increase in countries of Sub-Saharan Africa most affected by the epidemic (Sewankambo et al., 1994; Foster et al., 1995; Hunter, 1990). Already by the end of 1988, deaths due to AIDS in the age group 35-44 exceeded 12 percent of all deaths in the US and were over 5 percent of all deaths in countries of Western Europe (World Health Organization, 1991).

c. Impact on social and economic conditions: the importance of morbidity .

The effects of HIV/AIDS on mortality are only the most visible manifestations of broader implications for the health of populations. Potential increases in infant and child mortality in areas where HIV is transmitted heterosexually (or in combination with IV drug use) will be hard to avoid, particularly in countries where health budgets have been shrinking under the devastating effects of

prolonged economic crises (Dozon and Guillaume, 1994). And yet, there can be little doubt that it is increases in adult mortality and the impact on adult health that will have longer lasting consequences in many societies.

To begin with, excess adult mortality due to HIV/AIDS affects the population in the most productive age groups and strikes virtually all social and economic groups.[8] This translates into massive direct costs of increased health care met by governments and individuals alike by shifting away from consumption. It also implies large indirect costs associated with productivity losses due to illnesses and disability that punctuate the life of individuals with AIDS and the reallocation of tasks and reorganization of households that follow as coping strategies (Scitovsky, 1985; Ainsworth and Over, 1994; Ainsworth and Over, 1995). Depending on the dominant means of transmission, excess adult mortality may reverberate through social institutions causing disruption in families and households through increases in orphanhood and widowhood. Increases in orphanhood have been documented in areas such as Rakai District in Uganda (Hunter, 1990) and in Manicaland in Zimbabwe (Foster et al., 1995). These measured increases in orphanhood are broadly consistent with forecasts and projections based on estimated levels of adult HIV prevalence (Gregson et al., 1994; Palloni and Lee, 1992; Preble, 1990). We know very little about the family impact of HIV/AIDS in regions where the main modes of transmission are homosexual contact and IV drug use.

Yet for all its importance, emphasis on increases in adult and child mortality distracts attention from a possibly more disruptive effect of the HIV epidemic. At any one time, a variable fraction of individuals who are HIV positive is asymptomatic, that is, for all purposes they cannot be distinguished from uninfected persons. But, as discussed before, in social and ecological contexts where other diseases strongly interact with HIV, the 'asymptomatic period' may be characterized by more frequent episodes of illness and longer duration of disability than in other social and ecological contexts (Taelman et al., 1990; Riera et al., 1990; Melchior et al., 1990). The exacerbation of

respiratory tuberculosis in Sub-Saharan Africa, Latin Americaand Southeast Asia is just one example of this situation. Thus, the individual direct and indirect costs as well as the effects on the make-up, activities, and division of labor of families and households begin to mount **well before** the infected individual completes the incubation period. In societies where the epidemic is in its initial stages, the majority of those who are infected at any point in time were infected very recently and, therefore, the composition by duration of infection is heavily dominated by individuals who are more likely to be asymptomatic. As the epidemic progresses, however, the rate of increase of the infected population begins to slow down and the composition of the infected population by duration of infection will inevitably be dominated by those who, though free from AIDS symptoms, may be more frail and more susceptible to infectious diseases and other health impairments.I now provide two illustrations of this dimension of the impact of HIV/AIDS. The first illustration is drawn from theexperience of Latin America. The estimated course of the epidemic in this region combinedwith a median incubation time of about 10 years implies that the median duration of HIV infections will shift from .8 years in 1985, to 5.6 years in 1995 and will reach about 11 years in 2000. Thus, almost half of those infected in 2000 will belong to a category whose health could be severely impaired even if they do not yet have AIDS. A second illustration comes from the African experience. Under conditions resembling the epidemic in Sub-Saharan Africa between 22 and 38 percent of children younger than ten are expected to become orphans of one or two parents ten years into the epidemic (Palloni and Lee, 1992) and between 42 and 83 percent of children are expected to experience HIV- related illness from at least one parent. The toll in terms of child care, socialization, child education, and child labor force participation will be substantial.

d. Less abstract comparisons: country-specific patterns of HIV/AIDS

A shortcoming of the previous discussion is that it does not lead to a clean reading of peculiarities of individual countries or societies and, therefore, does not permit us to identify factors

that may explain regional divergency in patterns of spread of the epidemic. The diversity portrayed in Tables 1 and Table 2 masks deep contrasts since the regionalization scheme overlooks important within-region variation. Indeed, although in some regions described above, notably North America, Oceania and Western Europe, HIV/AIDS is fairly homogeneous, elsewhere the epidemic develops with significant within-region heterogeneity. For example, the patterns in Sub-Saharan Africa combine the explosive spread of HIV-1 among heterosexuals in Central and East Africa, the more subdued and slower spread of both HIV-1 and HIV-2 in West Africa, and the Europe-North American-like patterns that takes hold at least initially in South Africa. More perplexing is the fact that the contrast between East, Central Africa and West Africa is reproduced on a more micro scale within fairly contained subpopulations in each of these regions themselves. Thus, for example, the spread of HIV/AIDS in Uganda and Zaire follows different patterns even though both countries belong to the East-Central Sub-Saharan Africa region. By the same token, patterns in South and Central America are not equivalent to each other as the former is more European-like, while the latter is closer to the one observed in the Caribbean region. Since treatment of this heterogeneity is important as a diagnosis and forecasting tool, we now examine in more detail the contrast of HIV/AIDS regimes in countries that are typical of the class of patterns described before. I focus on three of elements that define the epidemic's profile: the relative contribution of various means of transmission, the age-sex profile of HIV prevalence, and the relative speed with which the disease spreads among various social groups.

d.1. Change and stability in modes of transmission.

Figure 1 displays estimates of the proportional contribution of different modes of transmission to the total diagnosed AIDS cases for two periods of time and for countries and subregions with available information. A remarkable feature in this figure is the sharp contrast between relative stability in US, Western Europe and Sub-Saharan Africa and persistent changes elsewhere. Note that

the bulk of cases in the US and Western Europe continues to be attributable to either intravenous drug use or homosexual behavior though, admittedly, there is an increasing contribution of the former at the expense of the latter. Instead, in South America as a whole and in Brazil and Mexico in particular, there is a substantial increase, in most cases a doubling, of cases due to heterosexual transmission and intravenous drug use and a steep decreaseof cases due to homosexual transmission. Newly released figures for Mexico, for example, suggest that whereas in 1987 74 percent of infections were attributable to homosexual contacts, in 1995 the figure had been reduced to 33 percent. Correspondingly, the contribution of infections associated with heterosexual contacts increased from about 0 percent in 1987 to about 27 percent in 1995 (Liguori, 1995).

These figures point to a definitive shift in the transmission patternsand underscores the spread of HIV/AIDS to different social groups. This shift may partially explain the fast growth of cases in Latin America identified before in Tables 1 and 2. Although the information is not available to confirm the conjecture, time series data on seroprevalence among prostitutes and IV drug users suggest that the pattern of distribution of cases by mode of transmission in Southeast Asia is similar to those observed in South and Central America and the Caribbean and that, there too, the transmission of HIV is moving increasingly toward a concentration in heterosexual groups (U.S Bureau of the Census, 1994). Once suitable time lags are accounted for, changes in the distribution of cases by modes of transmission must mirror closely the growth profile of HIV cases in various risk groups. The recent sudden changes in the relative significance of different modes of transmission in Southeast Asia and Latin America have as a counterpart an explosive growth of HIV prevalence among prostitutes (and their clients) which, in turn, resembles the growth of cases among comparable groups in Sub-Saharan Africa. As illustration,Figure 2 displays the relation between HIV prevalence among pregnant women and among prostitutes in various cities with available data. Although the plots are by region to highlight idiosyncracies, it is clear that there is a fairly general linear pattern

everywhere with slight differences in the slopes of the relation.

To summarize: the initial characteristics of the epidemic in regions most recently affected by it will not be preserved for too long. Indeed, the evidence available indicates that a shift toward a pattern dominated by heterosexual transmission is already in the making with all the implications this has for the broader spread of the virus in the general population.

Sub-Saharan Africa is at the other end of the spectrum: a very stable HIV epidemic with stationary contribution of various modes of transmission and cases persistently concentrated in the heterosexual population. To be sure, the epidemic has undergone important changes in the region but these are more closely related to patterns of geographic and social diffusion than to radical shifts in modes of transmission. Thus, while HIV has reached very high prevalence rates in some urban areas in Central Africa, in others the spread of the epidemic has been all but halted. Some of the reasons for this diversity will be discussed later but none of them involve heterogeneity in modes of transmission.

d.2. Age and sex profiles.

The most important contrast that emerges with the progression of different patterns of HIV is visible in the age-sex pattern of HIV and AIDS prevalence. In areas where the epidemic spreads mostly through homosexual contacts or among isolated pockets of IV drug users, the curves of age prevalence are heavily weighted towards adult men with little general impact on the female population or among children. Instead, where HIV is transmitted through bisexual and heterosexual contacts as well as through IV drug use, the age-sex pattern has a typical double peak during infancy and adulthood and relative parity between men and women. This contrast is apparent in the stylized representations in Figure 3 where curves of age-sex specific HIV and AIDS prevalence and incidence curves for various countries are compared. The importance of different age-sex profiles in HIV prevalence cannot be overemphasized. They reveal entrenched modes of transmission and prefigure

the contours of short run trends of the epidemicand of its demographic, social and economic effects. When the relative importance of modes of transmission changes as it has done recently in Latin America and Southeast Asia, we expect to see shifts in the age-sex profile of the epidemic, witha higher concentration of cases among females and young children. As a result we also should expect to see a convergence of the Latin American and Southeast Asian pattern toward that of Sub-Saharan Africa.

d.3. Parallel tracks: the course of the epidemic among high and low risk groups.

As suggested before, changes in the contribution of different modes of transmission and the consequent changes in the age-sex distribution of cases reflect important shifts in the underlying pattern of spread of the epidemic in the general population. The dynamics ofspread in the general population and the ultimate size of the epidemic depend on three factors. First, on the spread within high risk groups; second, on the degree of contact between high risk groups and the general population; and, third, on the main modes of transmission within the general population.[9] If contact between high and low risk groups is minimal, the epidemic may continue to grow and possibly saturate high risk groups while having few or no consequences for the general population.[10] If, on the other hand, the rate of contact between the two groups is non-trivial then the prevalence of HIV/AIDS will grow in the low risk population tracking with variable time lags the growth of HIV/AIDS in the high risk groups. This means that the correlation of HIV prevalence in these groups is in itself an indicator of the nature of the epidemic and of the potential threat facing the population at large. We now explore the nature of the relation from a comprehensive database maintained at the International Division of the US Bureau of the Census. This database includes estimates of HIV prevalence among subgroups considered representative of the total (adult) population (low risk) and among groups considered to be at high risk. To describe the relations between prevalence in high and low risk groups I chose a general purpose model with the following functional form:

$$G(\rho_{ij}(t)) = \sum_{j=1}^{j=n} \lambda_j G(\rho_{ij}(t-k)) \qquad \textbf{(1)}$$

where G(.) is a function member of the class of Box-Cox transforms, ρ_{ij}(t) refers to the estimated prevalence in country I, risk group j and time t, and λ_j is an 'effect' associated with risk group j. I consider the separate contribution of three major risk groups: homosexuals and IV drug users (combined), prostitutes (males and females combined), and STD patients. After a considerable amount of experimentation, three regularities were identified: first, the optimum transform G(.) in square and relative error terms was the logarithm of HIV prevalence; second, a consistently dominant predictor of HIV prevalence among the general population was HIV prevalence among prostitutes; and third, and as expected, the relations were stronger if prevalence among high risk groups was lagged.[11] The estimated coefficient (of the log of HIV prevalence among low risk populations during a period time on the log of HIV prevalence among high risk populations during a preceding period) is an elasticity coefficient that reflects the degree of communicability between low and high risk groups. Figure 4 shows the nature of the relations within two broad regions through two scatter plots. The fit is by no means perfect but it is quite close in both cases. The estimated elasticity is twice as much in Africa (.84) as it is in the other broad region (.31). A coefficient with a value of .84 can be interpreted as indicating that a one percent increase in the HIV prevalence among high risk groups translates, after a lag of a few years, into three-fourth of a percent increase among the general population. Although cautionary caveats regarding causality, temporal ordering, the cross sectional nature and the quality of the data rightfully apply, this model could be used to attempt an elementary sort of short run forecast which only requires simple information: indeed knowledge of estimates of prevalence among high risk groups at time t and the relations estimated before uniquely determine a range of forecast values of HIV prevalence for the general population some time later. How well

do these forecasts perform when compared to other, more elaborate ones? Figure 5 plots forecast values of adult HIV prevalence for selected countries around 1997-2000 against those obtained by macro-simulation models employed by the authors of **Aids in the World** (Mann et al., 1992). The plot reveals disagreements although, to be fair, the association between alternative forecast values is not less than what was found in the original data on prevalence by risk groups. The graph also displays a modified logistic curve adjusted to the pairs of forecast values which highlights the fact that predictions derived from the association between prevalence in various social groups (the y-axis in the graph) tends to be systematically lower when the alternative forecasts are higher and vice versa.[12] Later I show that the magnitude of the disagreement portrayed in Figure 5 is modest when compared with the gulf that separates more elaborate forecasting models.

IV. TRACKING THE DEMOGRAPHIC EFFECTS OF HIV/AIDS

Our discussion of the demographic impact of HIV/AIDS reflects uncertainty about the past, current, and future course of the epidemic. It is not that the procedures on which our estimates rest are distinctively inaccurate relative to others. Rather, **all** procedures designed to track and forecast the HIV/AIDS epidemic and its demographic effects are subject to errors of some importance. In this section we examine the origins of this uncertainty by turning on its head the problem posed at the outset: We started out by asking about the nature of HIV/AIDS effects on population outcomes and on selected social and economic processes. This demands the utilization of modest modelling efforts on our part. We now ask how demographers and epidemiologists articulate knowledge about the natural history of the virus and of the relevant social behaviors that affect its transmission into procedures, techniques or models that enable them to track the effects of the epidemic. We set out to identify aspects in model-building that translate into key uncertainties and, therefore, to identify where demographic, epidemiologic and social research is likely to have significant payoffs.

a. Simple and complex models.

Just as with population projection models, models for the spread of HIV differ considerably in terms of the complexity they are designed to capture, the data required as inputs, and in the relations between their components. Demographers are familiar with the paradoxical result that projections of (total) population perform better (in relative-error terms) when they are based on simple extrapolation of past growth rates than when they rest on the method of cohort-components and, furthermore, that this advantage is not exclusively confined to projections of the very short run (Keyfitz, 1981). Although the apparent superiority of aggregation and simple extrapolation of population figures is likely to be highly dependent on the size and complexity of the geographic and population aggregates used as units of analysis and may, in due course, be offset as cohort-component methods incorporate advances in stochastic forecasting, it is nevertheless somewhat unsettling to know that the fine-tuning embedded in a cohort-component procedure has, after all, a negative pay-off. Admittedly, a simple extrapolation of total figures cannot supply useful information on characteristics such as sex, age, or even regional composition. However, if total figures calculated with cohort-component methods are less trustworthy than those calculated with alternative procedures, it is at least awkward to place any confidence on finer breakdowns of population totals.

The tension between 'complex' and 'simple' models for the spread of HIV/AIDS is also an integral part of the development of formal epidemiological and demographic models for the spread of HIV/AIDS. As is the case for population projections, the distinction between a class of simple extrapolation models and a class of models involving complexities akin to those inherent to cohort-component methods reflects the distinction between models that minimize the information requirements for straightforward, short-term assessments and those that place high demands on levels of knowledge of behavioral and biological parameters of the epidemic for short and long term assessments. However, it is not an easy matter to design a fair comparison of their relative

performances. There are two reasons for this. First, complex models are highly dependent on the specific values assigned to a large number of parameters many of which are difficult to verify empirically. The difference between observed and projected values could thus reflect improper modelling as much as it does inadequate characterization of the parameters. The difficulty is that there is no simple way of disentangling the contribution of these two sources of error. Simpler models are less dependent on little-known behavioral parameters and the differences between observed realizations and projected outcomes are more straightforwardly associated with erroneous underlying assumptions about natural history or the dynamic of spread of HIV/AIDS.

Second, these two classes of models have very different objectives. Not only are simple models designed to produce projections only for the very short run (a few years ahead) while more complex models emphasize developments in the long run but, more importantly, the latter place heavy emphasis on the unfolding of the process of spread and on its impact on multiple subgroups. In other words, as is the case with the contrast between simple and complex models for population projections, simple and complex models for HIV/AIDS differ in terms of the sheer number of dimensions of population heterogeneity that each is designed to handle and inform.

b. The nature of models for HIV/AIDS projections.

The starting point for simple formalization of the spread of HIV/AIDS is a representation similar to that in Figure 6. At any time any individual in a population may occupy one and only one of three possible states: healthy or susceptible (Healthy), asymptomatic-infected (HIV+ or Infected) and AIDS (AIDS or ARC). The last state refers to all those stages associated with clinical manifestation of AIDS and encompasses potentially heterogeneous conditions, such as AIDS Related Complex (ARC) and full-blown AIDS.[13] In addition, each of these three states leads to an absorbing state, death, although the causes of death may be and frequently are quite different.

The flows between the three non-absorbing states, on the one hand, and these and the

absorbing state, on the other, are governed by five instantaneous rates. The first, λ, is the instantaneous transition rate from Healthy to Infected and depends on the process of infection. The second, δ, is the instantaneous transition rate from asymptomatic infected to symptomatic infected or AIDS, when individuals experience clinical conditions associated with the onset of AIDS. This passage corresponds to what is known as the incubation process. The flows between each of these three states and death are regulated by three possibly different instantaneous mortality rates, μ_1, μ_2, and μ_3 reflecting, respectively, the force of mortality among healthy or susceptibles, infected individuals and among individuals with full-blown AIDS.[14]

Stratifying the population by age and sex is conceptually straightforward and, when the corresponding age-sex specific transition rates are known, one can formulate a modestly complicated multistate (increment-decrement) life table model whose algebraic properties are well-known to demographers. The application of increment-decrement tables is greatly simplified by the fact that the flows are non-reversible, that is, that there are no backward flows among the non-absorbing states. However, it is complicated somewhat since some of the transitions are not just age but also duration dependent. An additional, and unfortunately not minor complication is that, as I show later, the force of infection, λ, is an 'endogenous' parameter since it is a function of the fraction of people already infected. Further stratification by regions of residence and social class is, of course, possible but even though they are conceptually transparent, the applications to multiple groups are computationally taxing and place high demands on information that is frequently unavailable. Not surprisingly the most important criterion of stratification, in addition to age and sex, involves classes of sexual behavior, a more mysterious and much lesser known phenomenon.

The force of infection, λ, is unarguably the most important parameter for the progression of the epidemic. It is divisible into five components associated with the five most important modes of transmission of HIV: blood transfusions, needles (mostly IV drug users), heterosexual contact

homosexual contact and vertically or mother-to-child transmission. Thus, λ is the sum of five transmission-specific forces of infection (λ_i, I=1,...,5). Since each of the forces of infection has its own dynamics and depends on fairly different conditions they normally require separate treatment.

Known also as the force of incubation, δ is the instantaneous probability that an HIV infected individual will develop clinical manifestation of AIDS. It completely determines the waiting time to AIDS or incubation period. This is an elusive parameter and there is still great uncertainty not only about its magnitude and functional form—its dependence on duration of infection—but also about the factors that affect it, including individual genetic endowment, generalized health and nutritional status, and external disease environment.[15]

Although in developed societies μ_1 and μ_2 are, by and large, quite similar, it is not known whether or not the equivalence is preserved under conditions of widespread prevalence of other infectious diseases. It is suspected, though not yet conclusively proven, that individuals who are HIV positive may be exposed to higher mortality risks than susceptibles particularly when exposed to environments where other infectious diseases are rife, where nutritional status is below normal standards, and where health services and care are poor (Piot and Colebunders, 1987; Quinn et al., 1987; Ryder and Mugerwa, 1994). In contrast to the uncertainties surrounding μ_2, the value of μ_3 is known with a great deal of accuracy: it is of high magnitude, normally implying half-lives of about one to two years and is higher in Africa than in the US (Piot, Goeman and Laga, 1994).[16]

All projection models for HIV/AIDS presuppose that a representation such as that in Figure 6 (or other approximations to it) is a good reflection of real processes. What distinguishes one model from another is the type of population and behavioral heterogeneity they are designed to handle.

c. A simple taxonomy of models.

Although there are alternative classifications of HIV/AIDS projection models (Bailey, 1988; Palloni and Glicklich, 1991; Isham, 1988), the scheme we discuss below is useful for comparing their

predictive power in a relatively fair manner. The description proceeds from the simplest to the most complex models.

c.1. Forward projections.

Simple HIV/AIDS projection models utilize the most accessible data describing the prevalence (and incidence) of AIDS. Let A(t) represent the new number of AIDS cases at time t, where t is the time elapsed (in months or years) since a suitably defined origin of the epidemic. The extrapolation procedure seeks to identify parameters of a function F(t) so that some loss function of F(t) and A(t), say $\mathcal{L}$, is minimized. At least for the initial stages of the epidemic, one of the preferred functions F(t) is an exponential (Morgan and Curran, 1986; Artzrouni, 1988). It has been argued, however, that for epidemics such as those experienced in the US and Canada, where there is a high degree of sexual heterogeneity even among the high risk groups, a cubic exponential performs much better (Colgate et al., 1988; Panjer, 1988) since it captures the transient decline of the growth rate of new AIDS cases that sets in as the epidemic saturates first the highest risk groups and then moves on among those exposed to lower risks. An important drawback of simple extrapolation procedures is the inability to resolve a fundamental identification problem: given a relatively small number of observations available at any time during the initial stages of the epidemic, many functions fit the data well but it is very difficult to tell these functions apart. Because the extrapolation procedure requires no a priori information about the transmission mechanisms, functional forms that fit the empirical data may differ substantially beyond the small window of time within which the extrapolation is based; therefore the accuracy of forecasts will decrease sharply as their time horizon is increased. This is, of course, an implausibly optimistic assessment since the reported information on AIDS prevalence or incidence is rarely accurate and timely.[17]

Figure 7 illustrates these shortcomings of the extrapolation procedure. The figure shows (the logarithm of) observed AIDS cases in the US for various years and superimposed on them the

projected values derived from exponential trends that fit the first four and the first six initial observations (curves labeled 'predict(4)' and 'predict(6)'.) The important point to note is that the projection fan is fairly broad even though it only reflects uncertainty about the shape of the trend as inferred from a small and changing number of observations and neglects all other sources of propagation of errors. An important source of uncertainty in this kind of projection is heterogeneity in sexual behavior. Population heterogeneity in sexual behavior typically generates multiple and partially overlapping epidemics that are superimposed on one another and follow each other with time lags (see below and Anderson, 1992). The observed (total) number of cases increases rapidly at the outset but slows down as the epidemic moves from higher risk to lower risk subpopulations. Note that the data for the US plotted in Figure 7 shows a leveling off new cases around 1988-1990 followed by a later resurgence. When this type of heterogeneity is present, simple extrapolation of HIV and AIDS using a handful of cases at the beginning of the epidemic can be badly off the mark even for modest projection horizons. A cubic polynomial may be a better choice, though the precision of the estimation of the parameters of the polynomial with only a handful of observations is problematic.

In sum, unlike the relatively benign experience demographers have had with simple projection population models, the highly uncertain and variable course of the HIV/AIDS epidemic is much less likely to be captured by simple functional representations of new AIDS cases.

c.2. Backward projections.

Normally we do not have access to good surveillance of new HIV cases in the general population or in selected subgroups. Despite shortcomings, reporting of new AIDS cases is more complete, more timely, and more frequent. Obviously, observed AIDS cases reflect underlying HIV cases. The question one can pose is: given an observed trend of newly reported AIDS cases, can we determine the growth curve of HIV cases? For most infectious diseases where differences between

asymptomatic infected and symptomatic infected are of little importance, the answer to the question is trivial since the number of asymptomatic individuals should equal the number of symptomatic ones. In the case of HIV/AIDS, however, where incubation times are potentially very long, the distinction between asymptomatic and symptomatic populations is crucial. The relation between new AIDS cases at time t and HIV cases in the past is as follows:

$$A(t) = \int_0^t I(t-s) \exp\left(-\int_0^s \delta(v)\,dv\right) \delta(s)\,ds \qquad \textbf{(2)}$$

Information on A(t) combined with an assumption about the functional form for instantaneous rate of incubation $\delta(s)$ reduces the problem to one of finding a representation for I(.) over the range 0 to t, the underlying (and unknown) function reflecting the number of new HIV cases.[18] Once I(t) is found, short term projections of the size of the epidemic are possible as long as mortality among HIV infected individuals is negligible.[19] Solution of the integral equation in expression (2), conventionally known as 'backward projection', is far from trivial and several robust procedures have been suggested (Brookmeyer and Gail, 1994; Colgate et al., 1988; Hyman and Stanley, 1988; Michalski and Yashin, 1989; Artzrouni, 1990). Virtually all variants are highly sensitive to assumptions about the incubation period and to the functional form chosen to represent the trajectory of A(t). Furthermore, the solutions to the equation are generally unstable in the sense that small stochastic disturbances affecting the observed values of A(t)—in the form of delays or omission in reporting—can produce sizeable changes in the estimated HIV incidence function. To illustrate some of the problematic aspects of backward projection, I will use the series of reported AIDS cases for the US with the parametric backward projection procedure suggested by Artzrouni (1990).[20] Figure 8 contains two plots. The first plot displays the observed yearly new AIDS cases for the US and the expected number that result from fitting three alternative backward projection models. The first two

sets of values (aids1 and aids2) differ only in terms of the median incubation period: aids1 reflects a median incubation time of 8 years whereas aids2 corresponds to a median incubation time of 15 years. The third set of values, aids3, corresponds to a backward projection model assuming an incubation period of 8 years but using only the first 6 observations of reported new AIDS cases. The second plot displays the estimated number of new HIV cases associated with each of the three backward projections. First, note that differences in the median incubation period produce fairly significant differences in the estimated underlying HIV epidemic and in the estimated number of AIDS cases but only at the tail end of the forecast horizon. Second, the use of a different set of observations leads to completely different HIV epidemics and forecasts of new AIDS cases. In sum, the combination of mis-identification of the correct median of the incubation function and a variable set of observations reflecting different stages of the epidemic leads to important errors both in tracking past HIV cases and in future forecasts.[21]

A variant of backward projection used by the United Nations and the World Health Organization in areas with very low quality AIDS data, requires us to estimate a yearly series of adult HIV prevalence. This information is then combined with an assumption about the probability of mother-to-infant transmission in order to generate estimates of pediatric infections. Finally, the estimated infections in any year t are allocated 'backwards' in time assuming an incubation function and a year for the start of the epidemic. Not surprisingly, this modified backward projection is also vulnerable to violations in assumptions about the incubation function and about the year of onset of the epidemic (Heuveline, 1995). And, more importantly perhaps, its accuracy is tightly connected to estimates of HIV prevalence whose accuracy is by no means assured.

c.3. Introducing population heterogeneity and infection rates: the weaknesses of complex models.

A complete formal representation of the phenomenon defined in Figure 6 consists of a system of partial differential equations. These partial differential equations will be recognized as simple

extensions of those first proposed by Verlhust (1838) and Von Foerster (1959) and then developed by Bennet and Horiuchi (1981), Preston and Coale (1982) and Arthur and Vaupel (1984) for generalized stable populations.[22] The only difference is that in this case we need to define 'compartments' within the stable population, each one of them corresponding to one of the three main states identified in Figure 6. For each compartment there is a pair of equations expressing age and time dependencies of the number of individuals in the various states, H(a,t), I(a,t) and A(a,t). The resulting total derivatives are the following:

$$\begin{aligned} \partial H(a+v,\ t+v)/\partial v &= -(\mu_1\ (a,t) + \lambda\ (a,t)\ )\ H(a,t) \\ \partial I(a+v,\ t+v)/\partial v &= -(\mu_2\ (a,t) + \delta\ (a)\ )\ I(a,t) + \lambda\ (a,t) H(a,t) \\ \partial A(a+v,\ t+v)/\partial v &= -(\mu_3\ (a,t)\ )\ A(a,t) + \delta(a)\ I(a,t) \end{aligned} \qquad \textbf{(3)}$$

In the steady state all partial derivatives with respect to time are the form $\partial F(a,t)/\partial t = r * F(a,t)$, where F stands for H, I or A, and where r is the instantaneous rate of growth in the population. To avoid cluttering, I omit reference to duration dependency which is inherent in the incubation function and neglect consideration of social heterogeneity which necessitates extensive use of subscripts. A further simplification involves dropping time dependency for $\delta(a,t)$. But even after these simplifications, an analytic solution to the system of differential equations is not readily available since the force of infection, $\lambda(a,t)$ is **endogenous** as it depends on the fraction of infected $I(a,t)=I(a,t)/(H(a,t)+I(a,t))$ (Anderson and May, 1991). To circumvent the problem, modelers have relied on numerical approximations and the various computer programs associated with these models are implementations of fine-tuned algorithms to obtain numerical solutions. These numerical solutions handle social heterogeneity by modifying the representation in (3) to include a finite number of subgroups with different sets of sexual preferences and sexual behavior. Preferences and sexual behavior affect the rates of contact between social groups and, through them, the rates of infection affecting contact between members of any two groups. The formal representation involves an

extension of (3) to one considering several subgroups requiring one system of equations per subgroup plus the equations establishing intergroup transfers and contacts. However, the precise conceptualization of social groups and of their transfers and contacts is complicated, and herein lies one of the most important factors accounting for contrasting results among various models.

Next I show that there are important differences in the results obtained by different types of numerical solutions for (3) and that this diversity is related to the conceptualization of heterogeneity of social groups and of sexual preferences and behavior. Although numerical solutions to the system of equations in (3) are not as elegant as exact solutions, they facilitate the introduction of complexities such as different social groups and different degrees of communicability between them, they make feasible the evaluation of feedback effects and, if properly implemented, they provide a precise and very effective means to assess short and long run effects of a variety of initial conditions regarding patterns of couple formation, couples' sexual behavior, incubation times, and infectivity. But since the core of all numerical solutions is a system of differential equations representing the process depicted in Figure 6,[23] one would expect a priori that, as in the case of simple population projections, there should be some concordance of results. As in the case of population projections, however, the expectation is simply not realized.

To compare results produced by alternative models I introduce three different contrasts. The first focuses on several demographic outcomes calculated from different models but under conditions for the epidemic that are **strictly comparable across models**. The second focuses on outcomes obtained from alternative models with no restrictions on the conditions of operation. The third is an attempt to compare selected outcomes from a simplified solution to the differential equations.

I. **<u>Constrained comparisons: the UN/WHO experiment with complex models</u>.**

The first contrast involves selected demographic outcomes obtained from several models using standardized inputs. This experiment was carried out in a joint exercise organized by the United

Nations (UN) and The World Health Organization (WHO) (United Nations, 1991). It was intended to generate forecasts for an HIV/AIDS epidemic with a profile very much resembling the one observed in Sub-Saharan Africa. Table 3 displays the rather disappointing results produced by the experiment. Note that there is hardly any agreement in any of the demographic outcomes selected for comparison except for the fact that all models generate no sustaining epidemic when the infectivity rate (the probability that an infected individual transmits the infection to a susceptible individual) is set to the lowest value (best scenario).

ii. **Unconstrained comparisons: results from a range of models.**

Table 4 shows a comparison of predicted life expectancies and rates of increase obtained from three different sets of models for Sub-Saharan Africa and Uganda. The models differ in terms of complexity. On the one hand, the UN model is based on the modified backward projection described before. This is not exactly a complex model in the sense we defined it above since it does not explicitly consider intergroup contacts and sexual behavior. The World Bank and the IWG models, on the other hand, are both based on macrosimulations involving several social groups and their contacts. These two models differ from each other in terms of the built-in complexity (number of parameters) and number of social groups each can handle.

The level of agreement achieved by these three models is no more encouraging that what was obtained before even though no a priori standardization ofinputs was enforced to achieve the results. Table 4 confirms once again that the business of forecasting the demographic impact of HIV/AIDS is a risky one, even when one focuses only on one region or country.

Why should results from alternative models be so different? To answer this question I show that, upon considerable simplification, the system of equations (3) yields a solution that identifies the key drivers of the epidemic. I then show that results are very sensitive to minor variations in some of the parameters and indicators selected to represent these dimensions and that it is there where we

should concentrate our research efforts.

c.4. Reducing complexity to see the forest: the power of simplicity.

Significant insights into the dynamics of the process can be gained by treating $\lambda(x,t)$ as if it were independent of time and exogenous to the system, that is, ignoring its relation to I(a,t). This is tantamount to examining the behavior of the models in a steady state. With this and other simplifications, all results from generalized stable populations follow. In particular, the set of differential equations have solutions for H(a,t), U(a,t) and A(a,t) which implies that: (a) the age distribution of the population is stable and the age distributions of the populations in the three compartments are also stable; (b) the time trajectory of the population in each compartment follows an exponential curve with a rate of growth equal to r; (c) the asymptotic age-specific prevalence and incidence rates of HIV and AIDS are time independent. Furthermore, all equalities of generalized stable populations to express populations at exactly age a and time t as functions of populations at early ages at some time in the past—the 'navigational' devices suggested by Arthur and Vaupel (1984) for the Lexis hyperplane—carry over for each and every one of the compartments in this population (Palloni, 1996). The most important consequence of the solution, however, is that an integral equation analogous to Euler-Lotka's for conventional stable populations applies. Assuming that individuals who contract AIDS are excluded fromthe childbearing process and that a proportion v of all births to HIV positive mothers become infected perinatally and do not reach adulthood, we obtain the following integral equation:

$$1 = \int_0 \exp(-(\pi+\Delta)x)S(x)m(x)\ [\Phi(x)+(1-v)\Gamma(x)]dx \qquad \textbf{(4)}$$

where π is the intrinsic rate of increase in the stable population with fertility function m(x) and survival function S(x) **in the absence of HIV/AIDS**, Δ is the change in π induced by HIV/AIDS, $\Phi(x)$ is the probability of not being infected by age x and, finally, $\Gamma(x)$ is a function of incubation

times and approximately equal to (1-I(x)). This equation contains a number of useful properties that we examine elsewhere (Palloni, 1996). It is important to note, however, that the integral equation depends on age specific rates of infection (through I(x)), on the incubation function (through $\Phi(x)$), and on the probability of mother-to-infant transmission (through v). Accordingly, the asymptotic rate of growth of a population affected by HIV/AIDS—the term $(\pi+\Delta)$—must be a function of these quantities and of the net maternity function only.

Although this result is fairly general and enables us to focus on the effects of HIV/AIDS on any demographic outcome we wish to consider, we will establish a straightforward relational metric regarding the rate of natural increase only. The metric we use here provides an answer to the following question: what is the relation between levels of HIV prevalence in the adult population and changes on the rate of natural increase? Besides its appealing simplicity, this metric suggests an immediate answer to the intriguing question about the 'efficiency of the HIV/AIDS epidemic' as a mechanism of control of population size and natural growth.[24]

To obtain a relation between adult HIV prevalence and associated changes in r in the simplified model (4) we examine two possible cases.

I. **Case I: v is identically equal to one.**

In this case every infant born to an infected mother becomes infected (and, by our previous assumption, never survives beyond childhood). The integral equation becomes a fairly well-known expression which can be used to determine the magnitude of Δ as a function of a schedule of changes in the force of mortality identical to ln I(x). It can be shown (see Preston, 1974) that Δ can be approximated by:

$$\Delta \approx -\left(\int_0^{A_m} \lambda(v)\,dv\right) \div A_m \qquad \textbf{(5)}$$

where A_m is the mean age at childbearing **in the absence of HIV**. When the schedules of fertility and mortality are constant, adult HIV prevalence, $\Phi(x)$, is approximately equal to (1-I(x)). It follows that Δ is a function of adult HIV prevalence only:

$$\Delta \approx (1/A_m)[\ln(1-I(A_m))] \qquad \textbf{(6)}$$

Since A_m changes mostly with the curvature of the fertility function, delta will be largely unrelated to mortality and incubation and only responsive to the rate of infection.[25]

ii. **Case II: v is smaller than one.**

Using the mean value theorem around A_m in the integral equation we can approximate Δ as:

$$\Delta = (1/A_m)\ \ln\ (\Phi(A_m) + (1-v)\Gamma(A_m)) \qquad \textbf{(7)}$$

The first part of the expression is simply the change in π that follows if HIV leads inevitably to vertical transmission and is identical to what we obtained before setting v=1. The second part reflects the 'additions'—relative to Case I—to the rate of increase attributable to children of infected mothers who are not infected themselves and who can, therefore, contribute to population growth.

Unlike Case I, the expression for Δ involves a dependency on the incubation function since $\Gamma(A_m)$ is simply an approximation to the distribution function of the incubation times. More concretely, the longer the incubation times, the higher the values of HIV prevalence and the more significant the offset due to the second component of the expression for Δ. However, changes in the form of the distribution function for incubation times have only minor effects on the magnitude of Δ (Palloni, 1996).

Having established these equivalences we now turn to describing the relation between rates of natural increase and adult HIV prevalence in the simplified and in the more complex models. From the complex models reviewed before we can establish the association between changes in the natural

rate of increase and alternative levels of adult HIV prevalence. If cross-model concordance were high, the relation between prevalence and change in rate of growth should be described by a set of well aligned points summarized perhaps by a straight line or a quadratic function. Figure 9 plots the relation between the two variables extracted from the simulation models studied before plus those derived from Zaba's model (Zaba, 1994). With one exception each model contributes two observations which are linked by an arrow. The exception is the single point obtained from John's model (1991). As expected from our discussion of Table 3, concordance across various models is at best quite minor. To establish a relation between adult prevalence and changes in r in the simplified stable model, it suffices to calculate the values of Δ corresponding to $\Phi(A_m)$ and $\Gamma(A_m)$. To do so we choose a stable population identical to the baseline stable population used by all models represented in the figure. We then evaluate the expression relating Δ and alternative values of v, Φ, and Γ. The points corresponding to these relations are plotted in the figure using the symbols "stab1", "stab2" and "stab3" respectively representing the cases when v is set equal to 1, .5 and .10. In all cases they imply an approximately linear relation the slope of which is proportional to the value of v. Note that the simplified relation that obtains when v=1 summarizes quite well the results obtained by a majority of models but that as v declines toward lower and more plausible values, it represents less and less the cloud of points generated by model's forecasts. A similar relation between changes in life expectancy and adult HIV prevalence (not shown) can also be estimated.[26]

V. THE NATURE OF $\lambda(a,t)$

The simple model introduced above may not be an optimal tool for making precise forecasts. Not only does it rest on algebraic simplifications that ignore higher order moments of the distributions involved but it depends on a modelling scaffold that overlooks the two sex-problem. Furthermore, the relations only hold in a steady state and do not reflect the magnitude of the **initial** impact of HIV on demographic parameters.

Despite these shortcomings, however, the simplified model has one distinctive advantage: it isolates the role of three crucial aspects of the dynamic of the infection, namely, the probability of mother-to-infant transmission, the distribution of incubation times, and the force of adult infection. In particular, it locates precisely the route through which the effects of $\lambda(a)$ or, if time dependency is allowed, $\lambda(a,t)$, have an impact on population structure. In the simplified world of the stripped model one could construct **model stable populations with HIV** since a combination of $\lambda(a)$, $m(a)$, $\mu(a)$, $\delta(a)$ and v uniquely determines the intrinsic rate of growth r. Since $m(a)$ and $\mu(a)$ may be known with some accuracy and since $\delta(a)$ and v can be approximated without great loss, the only function that stands in the way of a conventional calculus of model stable populations is $\lambda(a)$. We now turn to explore the nature of this function.

$\lambda(a)$ has been studied extensively by Dietz (1988) and by Anderson and May (1991). In the discussion that follows I will only refer to a population with heterosexual transmissions since this is unarguably the most complicated of the two cases.

There are four different elements that enter in the definition of $\lambda(a)$: population heterogeneity according to age-specific sexual behavior, infectivity per contact and its potential dependence on duration of infection, effective frequency of partners and, finally, the age and sex-specific distribution of preferences regarding sexual behavior. Consider the case when there are k groups, each defined according to sexual preferences. Membership in a group determines the distribution of number of sexual partners per year per member of the group (mean N_i and variance S_i); the preferred type of sexual practices between members of group i and group j which determines the level of infectivity of each sex act between a susceptible member of group i with a randomly selected infected member from group j who has been infected for d years, $B_{ij}(d)$; and, finally, a preference function defining the probability that a member of group i aged x at time t will choose a member of group j of age y $P_{ij}(x,y)$. A very general definition of the force of infection during year t for an individual aged x

belonging to group i, $\lambda_i(x,t)$ is, to a close approximation:[27]

$$\lambda(x,t) = \sum_{j \in J} \left(p_{ij} C_{ij} \int_{y \in Y} \left(P_{ij}(x,y) \int_0^{\infty} B_j(d)\, u_j(y,d,t)\, dd \right) dy \right) \qquad \textbf{(8)}$$

where $C_{ij}(x)$ is a function of $N_{ij}(x)$ and $S_{ij}(x)$,[28] the mean and variance, respectively, of the number of partners from group j for individuals aged x from group i, p_{ij} is the probability that an individual of group i will choose group j as a source of partners, $P_{ij}(x,y)$ is the probability that an individual aged x and in group i will choose as partner an individual aged y in group j (the sum of all $P_{ij}(x,y)$ over y should equal 1.0), $u_j(y,d,t)$ is the fraction of individuals aged y in group j who at time t have been infected for d years, and $B_j(d)$ is the infectivity of an individual belonging to group j who has been infected for d years. Finally, J is the set of labels referring to the various groups and Y is the set of labels for ages of partners that are permissible for individuals aged x.

As a first approximation we remove duration dependency and ignore preferences other than those associated with age difference. This leads to the most primitive but still realistic expression for $\lambda(x, t)$:

$$\lambda(x,t) = CB \int_{y \in Y} P(x,y)\, u(y,t)\, dy \qquad \textbf{(9)}$$

This last expression is sufficient to identify a few basic drivers of the HIV/AIDS epidemic where the main mode of transmission is heterosexual contact: mean and variance of number of partners (determining C), infectivity per sex act (determining B), and age preferences as reflected in P(x,y). A similar expression applies whenever HIV/AIDS is transmitted mainly via homosexual contacts.

a. The impact of age preferences among sexual partners.

A simple thought experiment is useful as expedient to assess the importance of age differences among partners (Palloni and Lamas, 1991). Consider what the course of the epidemic would be if (I)

HIV were transmitted only heterosexually and perinatally, (ii) infants who are HIV positive did not survive beyond say age 13 or 14, and (iii) all sexual partners had identical age (that is, the mean of the distribution of age differences between partners is zero with variance zero or, alternatively P(x,y)=1 when y=x but 0 otherwise). To assess the spread of the epidemic T_o years after its onset it is convenient to use the curve of age-specific HIV prevalence, I(x,t). It is not difficult to see that in this very peculiar population the sequence of values $\{I(x,To+k), k \geq 0)\}$ will be zero for $x \leq 50$ after k attains a value of 50. Beyond this point, HIV will literally disappear.This suggests that the epidemic may be a lot worse, **ceteris paribus**, when the distribution of age preferences involve high means and variances of age differences between partners. There are two ways to illustrate this. The first is to examine simulation results to contrast the cases when the age differences between partners are non-zero to those when they are strictly zero. The second is to examine the empirical association between age differences of partners and adult HIV prevalence.

Figure 10 displays yearly new HIV cases in two simulated epidemic regimes, the first where the mean age difference between a male and his female partner approximate a normal distribution with a mean of 5 years and a standard deviation of 2. The second regime is identical to the first except for the fact that no male-female partnerships are formed unless partners have the same completed age. The differences are as expected: whereas the first regime implies a profile of growing HIV cases, with bumps and oscillations reflecting initial conditions and converging to some steady state trajectory the second regime leads to a downward trend of cases right before fifty years have elapsed.[29]

This conjecture suggests that, **ceteris paribus**, in societies where traditional marriage and sexual partnership formation rules dictate that males be much older than women, the spread of HIV/AIDS will be more strongly entrenched and the epidemic will last longer. If so, a simple correlation between the mean or variance of the age differences between partners and adult HIV prevalence should capture the relation, provided we are able to control for relevant covariates.[30]

Unfortunately, the information available is normally about age differences of **married** partners or partners in a **union** and **not about age differences between partners in all sexual unions**. Information about age differences between casual and steady partners has not been normally collected in sex surveys (for an exception see Laumann et al., 1994b) but the study of its determinants should be high on our research agenda.[31]

A paradoxical feature of the epidemic is that two types of feedback related to age differences between partners could reproduce its presence in countries where the infection spreads heterosexually. First, increases in adult mortality may force some families to seek early sexual unions for their daughters as a way to compensate for financial losses or reduction of assets (Palloni and Lee, 1992). Under these conditions, the epidemic is ensured a self-feeding mechanism that magnifies its permanence and spread in the population. Second, it is believed that in the face of the epidemic, males seeking extramarital partners tend to seek younger women to minimize the risk of encountering an HIV-positive person. If this is so, the epidemic itself will generate behavioral changes that boost its reproduction in the population.

b. The influence of co-factors.

In our discussion of co-factors that either enhance or inhibit infectivity we identified the potential role of STD's and circumcision. Their effects increase or decrease B and affect directly the speed with which HIV spreads and the ultimate size of the epidemic. When B is too low the epidemic may not be self-sustainable whereas when B is large it may lead to total extinction of the population. The effects of differences in infectivity that may be explained by co-factors are illustrated in Figure 12 which displays the course of two epidemics, one of which involves a co-factor that increases infectivity of sexual contacts by 25 percent.[32] The differences in the levels and patterns of the epidemics are large enough to think that the existence of co-factors, particularly circumcision and STD, may go a long way to explaining some of the rather puzzling variability of HIV prevalence in

Sub-Saharan Africa (Caldwell and Caldwell, 1994; Bongaarts et al., 1989). To demonstrate that this is in fact the case is a difficult enterprise since in order to assess the role of co-factors it would be necessary to control for other social and epidemiological factors that co-vary with co-factors, for the time of onset of the epidemic, and, finally, would require us to identify a suitable unit of analysis which may not necessarily be a country or region but ethnic groups. As shown in Figure 11, however, the role of co-factors and HIV prevalence is suggestive enough to deserve more attention than it has received so far in the literature (Caldwell and Caldwell, 1994).

c. Patterns of sexual partnerships.

Consider now the addition of an extra layer of complexity to equation (9). Rather than focussing on the factors affecting intra-group transmission—as equation (9) does—we introduce consideration of several groups and their contacts. This involves a return to equation (8) and consideration of the effects of the parameter regulating the probability of contacts between groups, p_{ij}.

The effects of mean and variance on number of sexual partners, C in expression (9), is analogous to the effects of co-factors: it multiplicatively boosts the rate of infection: In societies where complete monogamy prevails, the HIV/AIDS epidemic cannot become self sustained. By contrast, in societies where partner exchange is commonly practiced, the epidemic can become endemic or explode. But it should be clear from expression (8) that there is another dimension to sexual exchange that matters: if there are several social groups characterized by different partner exchange rates that do not mix, the epidemic cannot spread from one group to the other and will remain contained within each group at levels commensurate with the parameter C_{ij}. The spread within the society as a whole, however, depends on the composition of the population by type of groups, on the degree to which these group-specific patterns of behaviors are reproduced over time and, finally, on the degree of contact between groups.

The importance of the type of mixture between groups is illustrated using simulated data in Figure 12. This figure plots levels of HIV prevalence for two simulated populations. In the first, sexual relations are controlled by random mating within and across two groups, a monogamous one and a non-monogamous one. In the second, partners are selected only within one's own group (communication between groups is constrained so that $p_{ij} = 0$ if j different from I). Other than differences in the probability of contacts between groups the simulated populations are identical. As suspected, random mixture yields by far the worst epidemic. The observed effects are exaggerated since we forced them to appear very soon after the onset of the epidemic by setting high values of C. If C had been set at lower levels the divergence of the two epidemics would have taken longer to be established but would have inevitably appeared and been as overwhelming as it is in Figure 11.

Except for information recently collected in the US and in some regions of developing countries (Laumann, et al., 1994b), we know very little about the prevalence of different types of sexual partnerships and how they lead to blends of different groups. Laumann and colleagues report that in the US, monogamy dominates and that the existence of multiple partners is fairly contained and does not appear to vary with age or cohort. By contrast, in a series of articles Carael and colleagues (Carael et al., 1992) examine the data from surveys fielded in several settings in the developing world and identify the existence of a fairly substantial amount of variability and clustering of sexual behaviors. Table 5 displays side by side the prevalence of male sexual behaviors that lead to high rates of infections (multiple partners and contacts with prostitutes) estimated by Carael and colleagues. Although it is difficult to posit a relation with HIV prevalence since we lack information on sexual partnerships for all countries or localities with very low levels of HIV prevalence and since the timing of introduction of HIV differs, it is worth noting that at least in Africa the countries (cities) with the highest HIV prevalence are also those with the highest proportion of males with extra partners or highest frequency of commercial sex. Table 5 should serve also to reinforce our earlier

impressions about current trends in some regions where HIV is incipient: two of the countries where HIV has been growing rapidly, Brazil and Thailand, share to some extent the high levels of prevalence of male behavior involving extramarital partners and commercial sex.[33]

Finally, I raise an issue that has received very little attention but that controls the epidemic in the medium to long run. This regards the mechanism of reproduction of each of the social groups involved in the epidemic. If sexual behavior is only partially transmitted from parents to offsprings and depends also on learned behavior that changes as individuals mature, the spread of the epidemic will depend crucially on the degree to which social groups experience intergroup mobility and not just on intergroup differentials in their natural rate of increase. To the extent that educational campaigns succeed in increasing the rate of mobility from groups with high rates of partner exchange to those with low rates or to monogamy or, alternatively, to shut off communicability between groups, the epidemic will inevitably slow down.

d. The plausibility of model-patterns of HIV/AIDS.

The considerations derived above from a simplified model of HIV/AIDS and from our knowledge of HIV suggest that it is possible, though admittedly complex, to generate **model-patterns of populations affected by HIV**. If we ignore for a moment the variability induced by differences in v and in $\delta(x)$, the model-patterns could be based on the following characteristics: a) main mode (or combination of modes) of transmission; b) age differences in sexual partnerships; c) patterns of sexual partnership formation; and, d) types of co-factors. To account for empirical variability we reduce complexity by conceiving of (b) and (c) as simple dichotomic properties. Thus, if we focus only on regimes of HIV where heterosexual transmission predominates, we will obtain four possible model patterns. Within each of these models additional variability can be generated by varying the level of the force of infection either through changes in C (mean and variance of the distribution of partners) or B (altering co-factors).

The usefulness of this scheme is potentially far from trivial. This is because within each of the model-patterns relations between parameters reflecting the epidemic should be fairly regular and reflected in simple expressions. For example, the effects on life expectancy ten years after the onset of the epidemic (or even in the steady state) could be represented by a regression equation relating life expectancy and levels of HIV adult prevalence **within each model-pattern**. The advantage of this representation is that the effect that different types of epidemic can exert on the relation between the two parameters is accounted for by the model-pattern. A scheme such as this enables us to deploy tools for the measurement of quantities in indirect ways. Thus, for example, suppose that surveillance data gathered in urban areas identifies the level of adult prevalence and, in addition, we obtain an independent estimate of the approximate number of years since the onset of HIV. From a relation between adult prevalence and other parameters of the epidemic calculated for each from model-patterns it should be possible to calculate estimates of several markers of the underlying epidemic including the levels of $\lambda(x,t)$, one of its main drivers.

The most important advantage of the utilization of model-patterns is not just that it offers a shortcut to estimate quantities we may be interested in, but rather that, through it, all calculations and manipulations are done respecting the different modalities of the epidemic which are dictated by the impact that different social behaviors have on the force of infection.

FINAL CONSIDERATIONS

We have come full circle. This paper began by marshalling the evidence available to retrieve indications and traces left by the HIV/AIDS epidemic. In particular we emphasized issues of timing, those related to the development of different stages and, more importantly, the emergence of radically different epidemics in various regions of the world. In particular we devoted some attention to the lessons one can learn from the epidemic in regions where it is established early on during the seventies and eighties. The events in Latin America and Asia, two of the regions where HIV/AIDS is growing fastest, should produce concern since the indications we have is that HIV/AIDS in these regions is progressing toward the general population through the same mechanisms it did so in Sub-Saharan Africa. Unless something is done quickly, it is likely that the general impact will also be the same.

The demographic impact of the epidemic is tightly connected to the dominant modes of transmission. Although the death toll is staggering anywhere due to the high lethality of the disease, the social and economic consequences of massive health impairment as well as higher mortality are likely to be overwhelming in areas where the disease is spread heterosexually since it is there where it will swept not just one but several generations in one single, swift movement. Furthermore, behavioral responses and coping mechanisms alike could trigger the emergence of feedback mechanisms that will delay the moment when the disease will retrench and could even accelerate its progress.

But the impact of HIV/AIDS is not just purely demographic, economic, social or even ideological. Its effects are also felt in the way researchers conceptualize the disease through the formulation of models, methods and techniques designed to trace the effects of the epidemic. Progress in this area has been fast and impressive but the distance to cover before we are able to produce reliable models is still fairly long. Contrasting complex and simple models and extracting from the latter the most fundamental features that drive an HIV/AIDS epidemic led to identification of a

selected social and behavioral dimensions which need to be studied in depth in order to leap toward the goal of having a better understanding of the demographic effects of HIV/AIDS.

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Table 1: Stages and Patterns of Global HIV Transmission

STAGE	PERIOD	DOMINANT MODE	MA/FE	AREA
I	>1980	Heterosexual	≈1.0	Sub Saharan Africa
		Homosexual	≈10.0	North America Western Europe Oceania
II	1980-1985	Heterosexual	≈1.0	Sub Saharan Africa
		Homosexual and IV drug users	≈10.0	North America Western Europe Oceania
		Homosexual and Bisexual; Heterosexual	≈3.0	Caribbean
		Homosexual and Bisexual; Heterosexual; IV drug users	≈3.0	South and Central America
III	>1985	Heterosexual	≈1.0	Sub Saharan Africa
		Homosexual and IV Drug Users	≈10.0	North America Western Europe Oceania
		Homosexual and IV Drug Users	≈8.0	Eastern Europe
		Homosexual and Bisexual; Heterosexual; IV drug Users	≈3.0	Caribbean South and Central America
		same as above	≈3.0	South East Asia North East Asia

Table 2a: Indicators of the HIV/AIDS Epidemic
(Observed data)

Area	Cumulated AIDS cases (in 1,000)[1]			AIDS Incidence Rate[2] (per 100,000)	
	1984	1990	1993	1985-90	1990-93
NA	12.6	210.9	492.9	71.9	102.2
WE	1.5	59.2	128.6	13.3	16.0
OC	.1	3.1	5.9	11.4	10.7
LA	1.1	101.4	313.7	24.2	51.2
SSA	2.9	736.1	2,084.0	139.5	255.7
CAR	1.5	19.0	35.9	52.0	50.8
EE	.0	2.1	4.5	.5	.5
SEM	.0	3.6	8.5	1.2	1.2
NEA	.0	1.3	3.3	.1	.2
SEA	.0.0	1.0	23.7	.12	1.7

[1] Reported number of AIDS cases adjusted for completeness. (See Appendix).

[2] Ratio of adjusted new AIDS cases during the corresponding periods to the estimated midperiod population. Estimates of midperiod calculated from United Nations Yearbook (several years).

Table 2a (cont): Indicators of the HIV/AIDS Epidemic
(Estimated data)

AREA	Cumulated HIV Cases (in 1,000)[1]		New HIV Cases and Incidence Rate (per 1,000)[2]		Doubling Time of Epidemic
	1985	1995	1985	1995	(months)
NA	338(51)	2,138(208)	94(12) [.34]	123(14) [.45]	23(1)
WE	87(5)	398(104)	30(6) [.07]	15(15) [.14]	18(7)
OC	4(1)	24(7)	2(1) [.08]	684(667) [.12]	16(5)
LA	88(11)	3873(2562)	39(8) [.09]	684(667) [1.58]	13(2)
SSA	457(253)	10,948 (1,215)	306(192) [.58]	128(127) [.243]	8(2)
CAR	40(17)	136(26)	15(70 [.03]	2(1) [.01]	12(3)
EE	*	*	*	*	*
SEM	5(2)	23(0)	1(0) [.003]	2(1) [.00]	42(2)
NEA	1(0)	38(16)	1(0) [.00]	8(2) [.01]	11(6)
SEA	1(0)	2195(1379)	1(0) [0]	768(542) [.56]	8(2)

[1] The values in each columns are averages of a range of estimates defined by Methods I and II (see Appendix).

[2] The values in round parentheses are the estimated widths of the range of estimates. The number in squared brackets are rates expressed per 1,000 population. The denominator of these rates are the estimated midyear populations.

* Estimates for Eastern Europe (EE) were unstable and unreliable due to small number of cases.

Table 2b: Estimates of HIV prevalence[1,2]

	1995			1985		
AREA	Total	Adult	Children	Total	Adult	Children
NA	1529-1841 [6-7]	1508-1816 [12-14]	21-25	211-284 [.8-1]	209-280 [.8-2]	3-4
WE	198-378 [.5-.9]	196-374 [1-2]	2-4	60-67 [.1-.2]	59-66 [.2-.3]	1-2
OC	12-24 [.5-.9]	12-23 [1-2]	0-1	3-4 [.1-.2]	3-4 [.25]	0
LA	1080-5966 [3-14]	1.38-5,733 [5-29]	42-233	56-135 [.1-.3]	54-130 [.3-.7]	2-5
SSA	6577-10654 [13-20]	5851-9477 [24-39]	726-1177	151-521 [.3-1]	134-463 [.6-2]	17-58
CAR	66-124 [2-4]	63-118 [4-8]	3-6	15-41 [.5-1]	14-39 [.9-1]	1-2
EE	*	*	*	*	*	*
SEM	16-17 [.05]	16 [.09]	0-1	3-5 [.01]	3-5 [.03]	0
NEA	18-48 [.01-.03]	18-47 [.03-.07]	0-1	0-1 [0]	0 [0]	0 [0]
SEA	780-3496 [.6-3]	755-3376 [1-5]	25-120	0-1 [0]	0-1 [0]	0

[1] Absolute numbers are the range for the number of infected individuals in each year in 1,000.

[2] Numbers in square brackets are the ranges of prevalence rates (per 1,000) or the absolute numbers of HIV cases at the boundaries of the range divided by an estimate of the midyear (adult or total) population (times 1,000). All numbers are rounded to preserve space and avoid cluttering. When only one number appears in square brackets it means that rounding led to only one estimate of prevalence rates.

* Estimates for Eastern Europe were unstable and unreliable.

Table 2c: Estimates of AIDS prevalence[1,2]

	1985		1995	
AREA	Absolute Numbers (in 1,000)	Prevalence (per 1,000)	Absolute Numbers (in 1,000)	Prevalence (per 1,000)
NA	22-31	.08-.12	243-295	.87-1.06
WE	4-8	.01-.02	48-64	.11-.15
OC	.0-1	.0-.04	3-4	.12-.15
LA	3-5	.01-.01	180-513	.43-1.23
SSA	11-24	.02-.05	1496-1565	2.84-3.0
CAR	1-3	.03-.09	18-23	.55-.70
EE	*	*	*	*
SEM	0-1	.0-.0	3-4	.0-.01
NEA	0-1	.0-.0	3-5	.0-.01
SEA	0-1	.0-.0	52-181	.04-.13

[1] Columns labelled 'Absolute numbers' contain range of estimates for the absolute number of individuals affected by AIDS for each year.

[2] Columns labelled 'Prevalence' contain range of estimates for the AIDS prevalence rates. These were obtained by dividing the absolute number of cases by estimates of the midyear population.

* Estimates for Eastern Europe are unstable and unreliable.

Table 2d: Estimated Proportionate Changes in Mortality and Losses in Life Expectancy[1,2]

	Proportionate Changes in Mortality Rates Between 1985 and 1995		Losses in Years of Life Between 1985 and 1995	
AREA	${}_{35}M_{15}$	${}_{5}M_{0}$	${}^{o}e_{o}$	${}_{35}e_{15}$
NA	.34	.033	.46	2.5
WE	.40	.010	.13	2.9
OC	.063	.016	.20	.50
LA	.082	.036	1.64	1.6
SSA	.12	.025	2.07	4.0
CAR	.031	.004	.33	1.1
EE	*	*	*	*
SEM	.002	.00	.00	.03
NEA	.00	.00	.00	.02
SEA	.008	.010	.45	.16

[1] ${}_{x}M_{y}$ refers to the mortality rate between ages y and y+x; ${}_{x}e_{y}$ refers to mean numbers of years lived in the interval y to y+x and ${}^{o}e_{o}$ refers to the life expectancy at birth.

[2] The numbers in the first two columns are the proportionate changes in the corresponding rates induced by he presence of HIV/AIDS. The numbers in the last two columns are the absolute losses in years of life associated with each of the corresponding life expectancies.

* Estimates for Eastern Europe are unstable and unreliable.

Table 3: A Comparison of Forecasts from Constrained Models[a]

		DEMOGRAPHIC OUTCOME			
Model	Scenario	HIV prev. (in %)	AIDS prev. (in %)	Life expectancy (in years)	Natural increase (in %)
	Best	0	0	60	3.0
Model 1	Medium	31.0	3.0	26	.5
	Worst	55.0	5.8	16	-2.0
	Best	.2	0	[b]	3.3
Model 2	Medium	15.0	1.9	[b]	2.4
	Worst	55.0	7.0	[b]	.16
	Best	.3	0	61	3.0
Model 3	Medium	39.5	4.5	47	2.5
	Worst	57.5	4.4	45	2.4
	Best	0	0	61	3.2
Model 4	Medium	21.2	1.8	36	1.5
	Worst	43.9	4.7	24	-.7
	Best	0	0	65	2.9
Model 5	Medium	3.5	.8	58	2.6
	Worst	42.4	12.0	28	-2.5
	Best	.1	0	60	3.1
Model 6	Medium	2.8	.4	42	2.8
	Worst	30.3	5.2	22	-.00

[a] Source: United Nations, 1991

[b] Outcome not available as output of model.

Table 4: A Comparison of Forecasts from Unconstrained Models

A. Sub-Saharan Africa

Forecasting Entity

Year	World Bank	IWG	United Nations
Differences in natural rate of increase (%)			
2000	-7.2	-44.0	-9.4
2005	-5.8	-55.0	-8.9
Differences in life expectancy (%)			
2000	-6.5	-	-10.6
2005	-5.4	29.7	-11.3

B. Uganda

Forecasting Entity

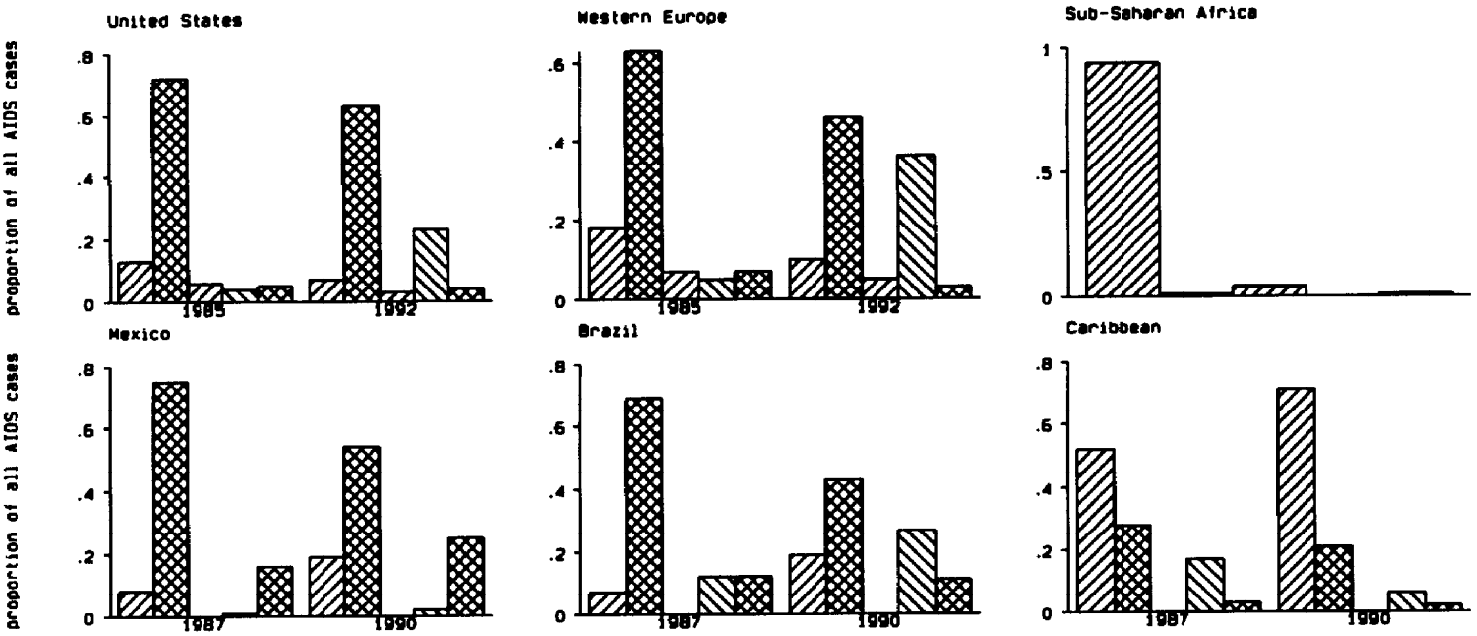
Year	World Bank	IWG	United Nations
Differences in natural rate of increase (%)			
2000	-24.2	--	-21.0
2005	-19.2	-57.1	-18.0
Differences in life expectancy (%)			
2000	-17.0	-	-26.0
2005	-16.0	50.00	27.0

Sources:
a) World Bank: from Eduard Bos, 1994: 'Incorporating mortality in population projections: the World Bank approach'
b) IWG: from Peter Way, 1994: 'Incorporating AIDS-related mortality into the U.S. Census Bureau's International Population Projections'
c) United Nations: from United Nations, 1994: 'AIDS and the demography of Africa'.
All three papers were presented at the Population Association of America Meetings, Miami, 1994

Table 5: Frequency of extramarital partners and commercial sex: selected areas

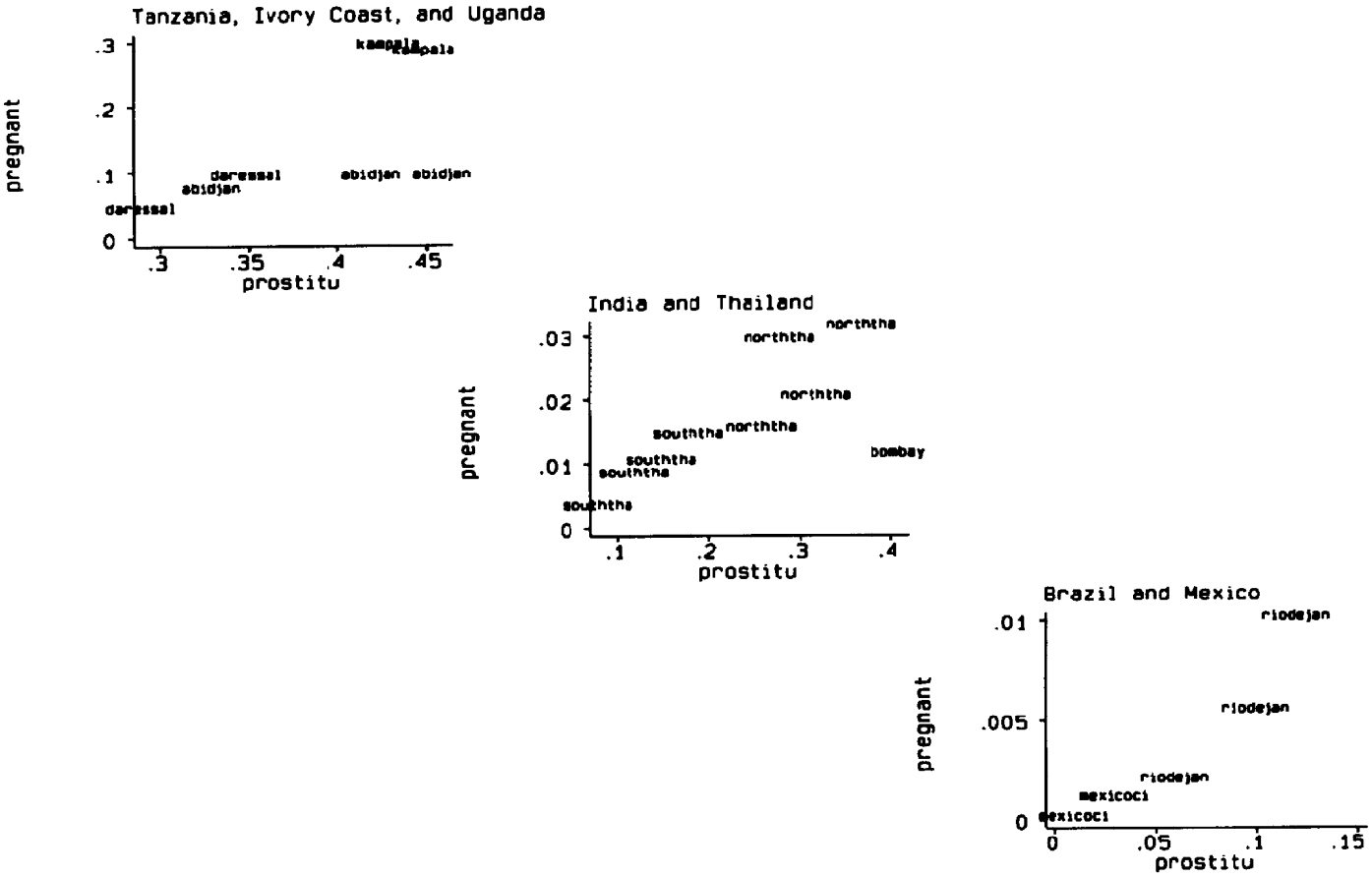
Area or Region	Proportion of males&females (aged 15-49) who report a least one extramarital partner in last 12 months	Proportion of males &females who report more that five extramarital partners in last 12 months	Proportion of males&females who report commercial sex in last 12 months
Central African Republic	.14	.02	.12
Ivory Coast	.49	.09	.10
Guinea Bissau	.42	.01	.11
Togo	.21	.02	.08
Burundi	.08	.02	.05
Kenya	.21	.04	.10
Lesotho	.46	.07	.15
Tanzania	.21	.05	.25
Lusaka (Zaire)	.35	.065	.09
Manila	.08	.065	.05
Singapore	.06	.025	.06
Sri Lanka	.05	.0	.01
Thailand	.17	.11	.25
Rio de Janeiro	.31	.10	.08

Source: Carael et al., 1992



(order of bars:heterosex., bi&homosex., transfus., IV drug, other)

Figure 1: AIDS cases by mode of transmission



(proportion HIV+ among pregnant women and prostitutes)

Figure 2: HIV prevalence in low and high risk groups

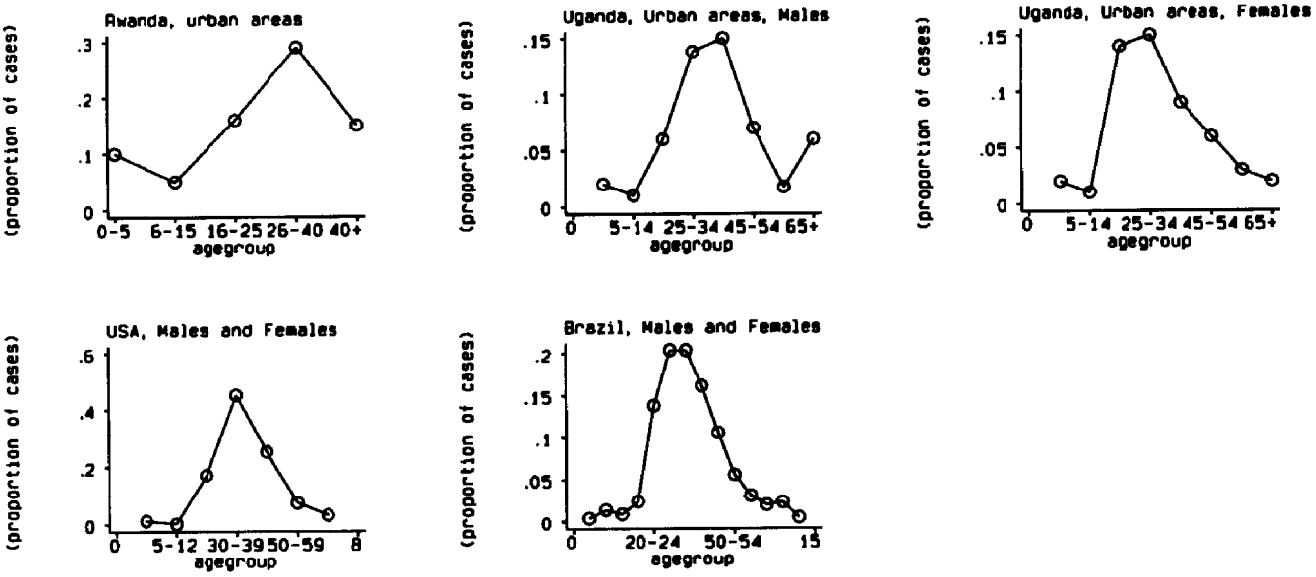


Figure 3: Stylized Age-Sex Distribution of HIV Cases

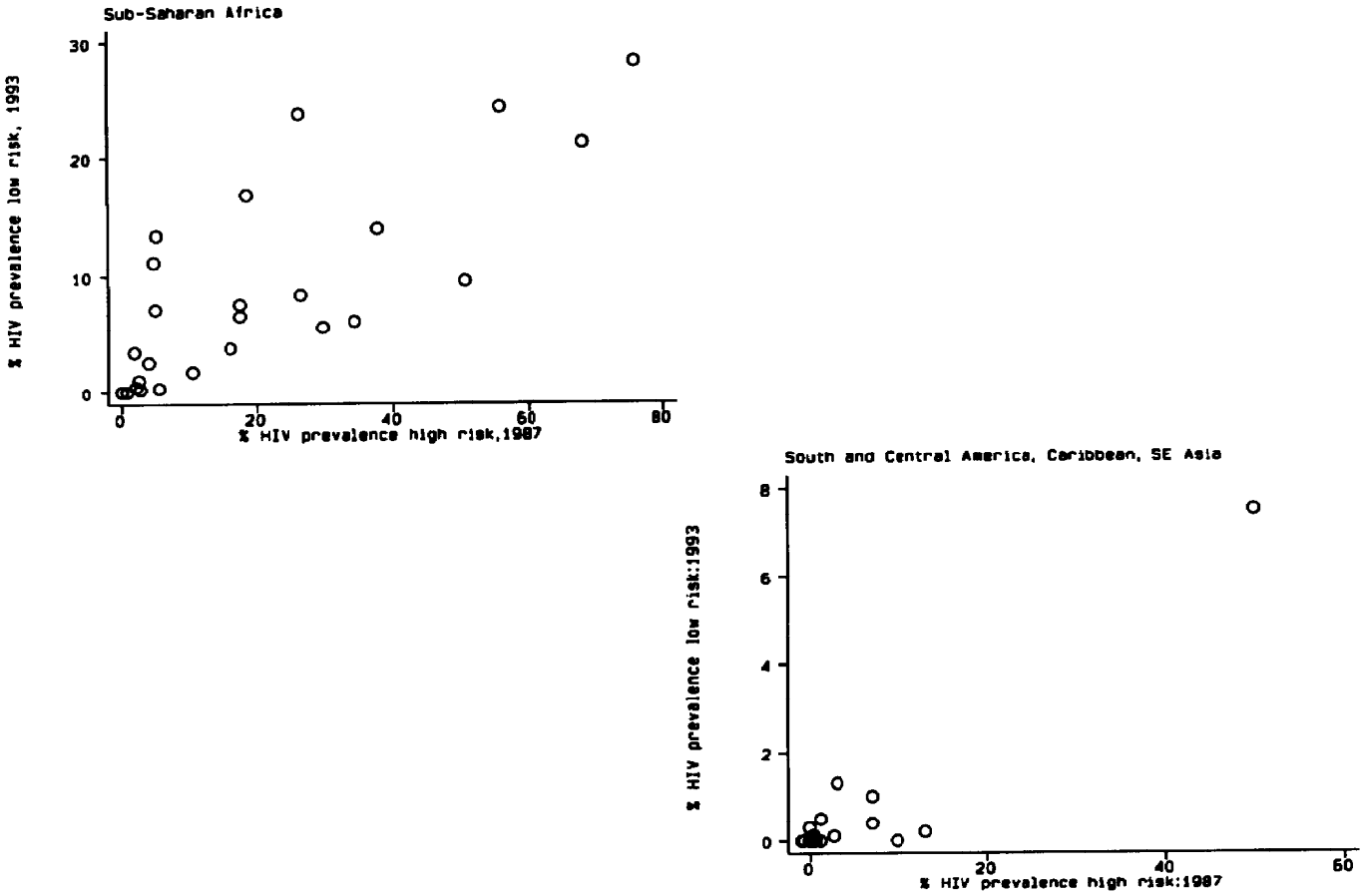


Figure 4: HIV prevalence in high and low risk groups

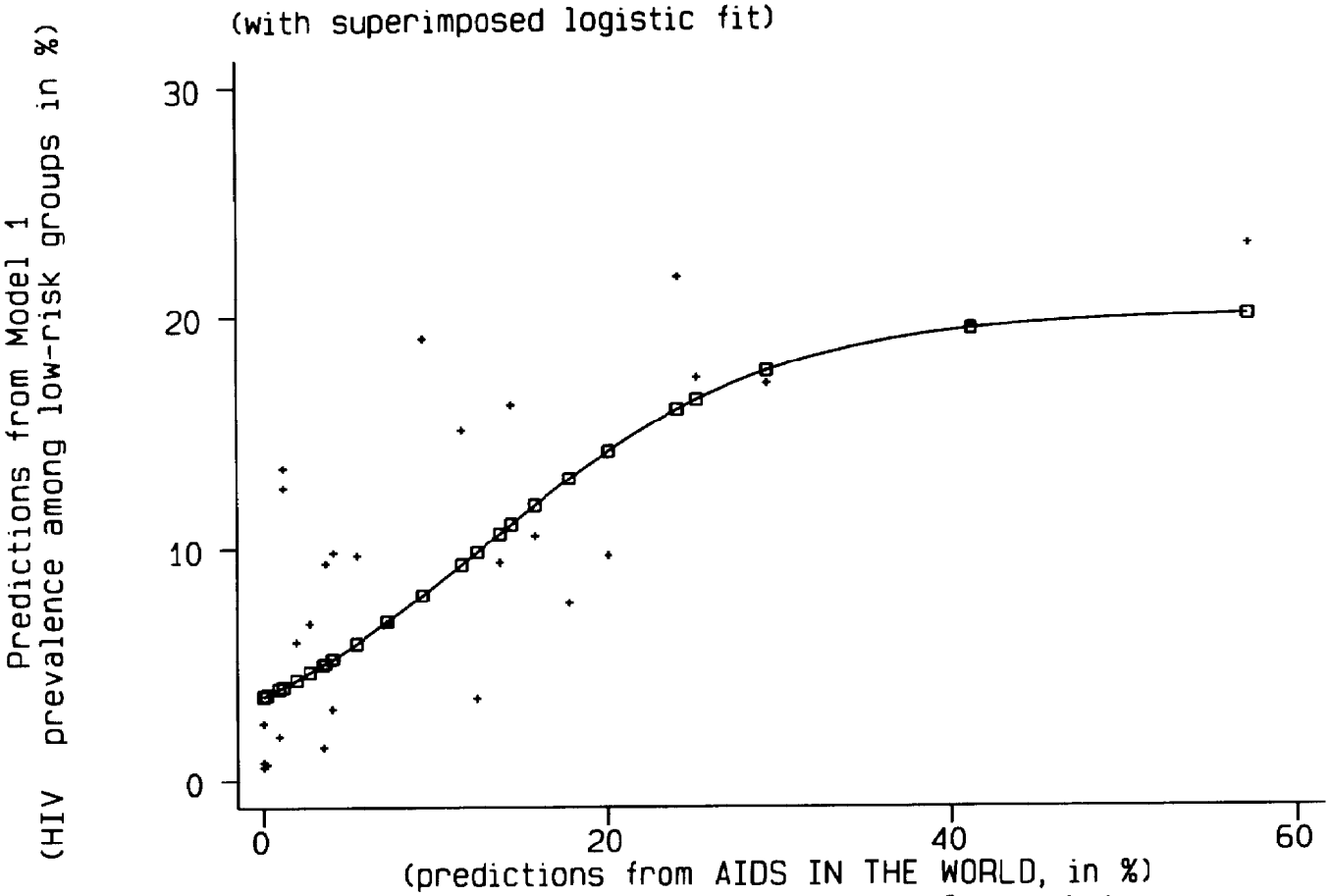
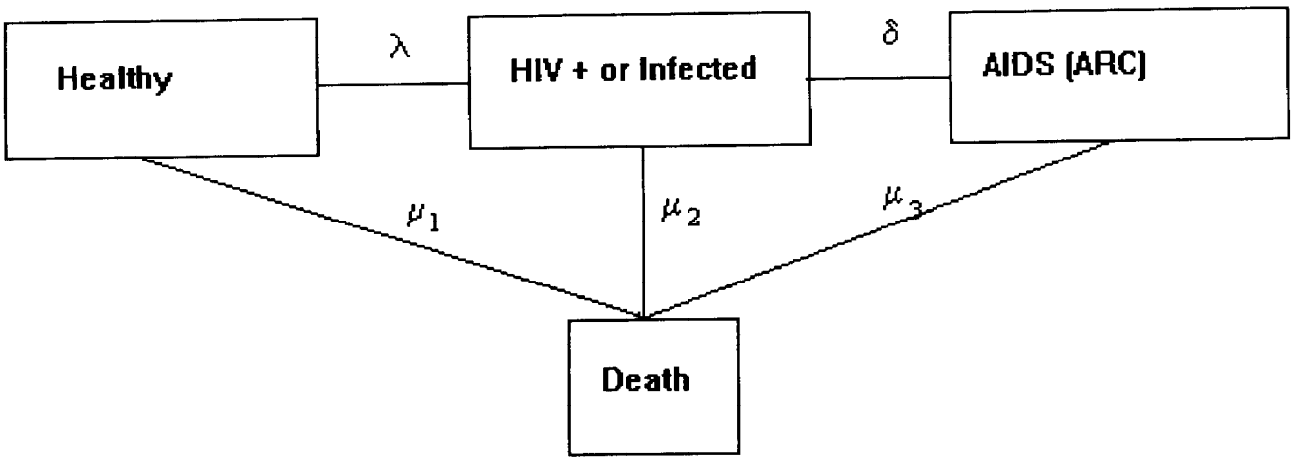


Figure 5: Forecasts HIV prevalence in low-risk groups

Figure 6: A multistate representation of HIV/AIDS



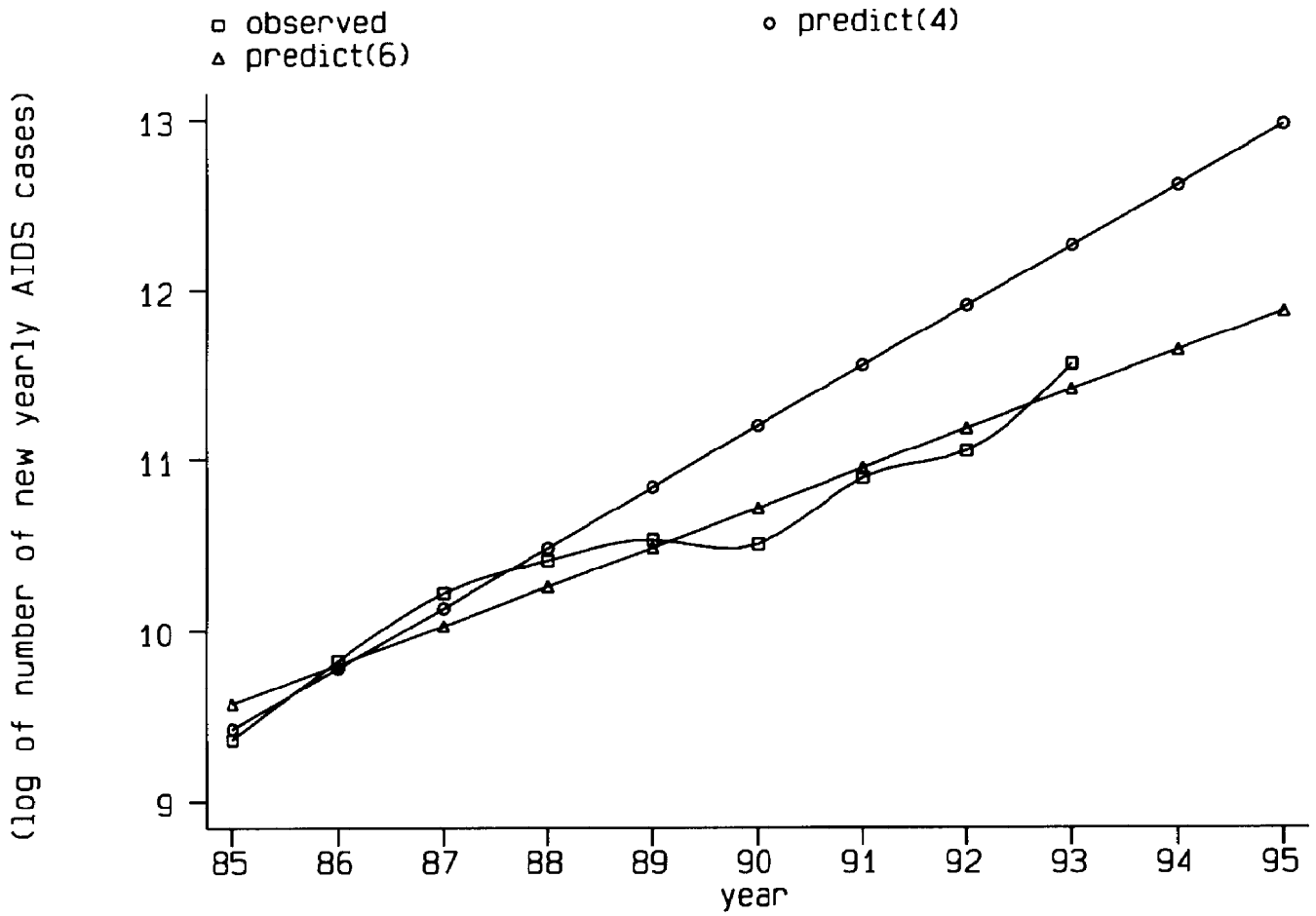


Figure 7: Log of AIDS cases in the US: 1985-1995

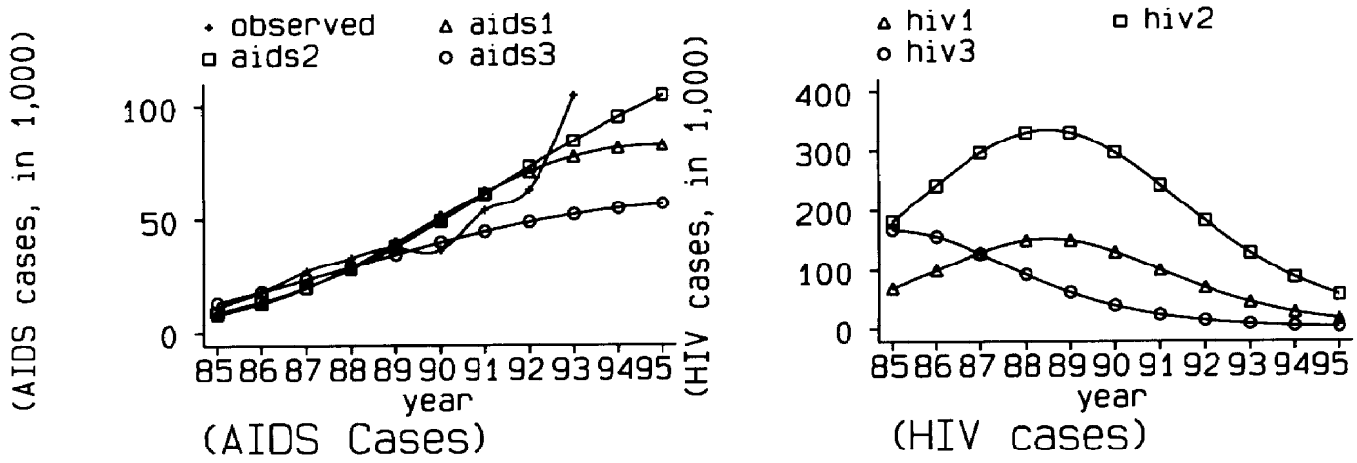


Figure 8:Alternative Backward Projections, USA:1985-1990

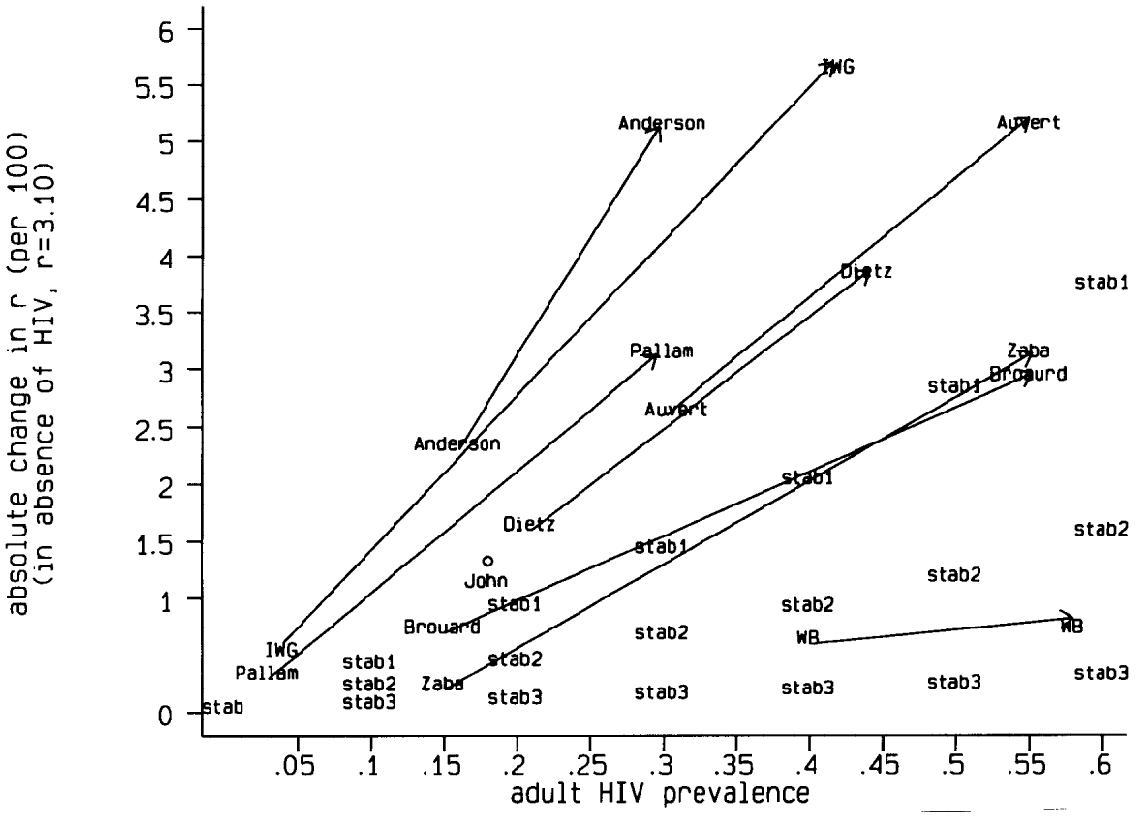


Figure 9: HIV prevalence and changes in intrinsic rate of growth

Note to Figure 9:

The labels for each point in the graph correspond to the identification of each simulation model considered in this paper. With the exception of the model by John, each model contributes two points joined by arrows. The correspondence between points and labels is as follows:

Anderson: Anderson and May model. Source: United Nations, 1991.
IWG:Inter-Agency Working Group model. Source: United Nations, 1991.
Pallam: Palloni and Lamas model. Source: United Nations, 1991.
WB: World Bank model. Source: United Nations, 1991.
Brouard: Brouard model. Source: United Nations, 1991.
Auvert: Auvert model. Source: United Nations, 1991.
Dietz: Dietz model. Source: United Nations, 1991.
Zaba: Zaba model. Source: Zaba(1994)
John: John model. Source: John (1991)

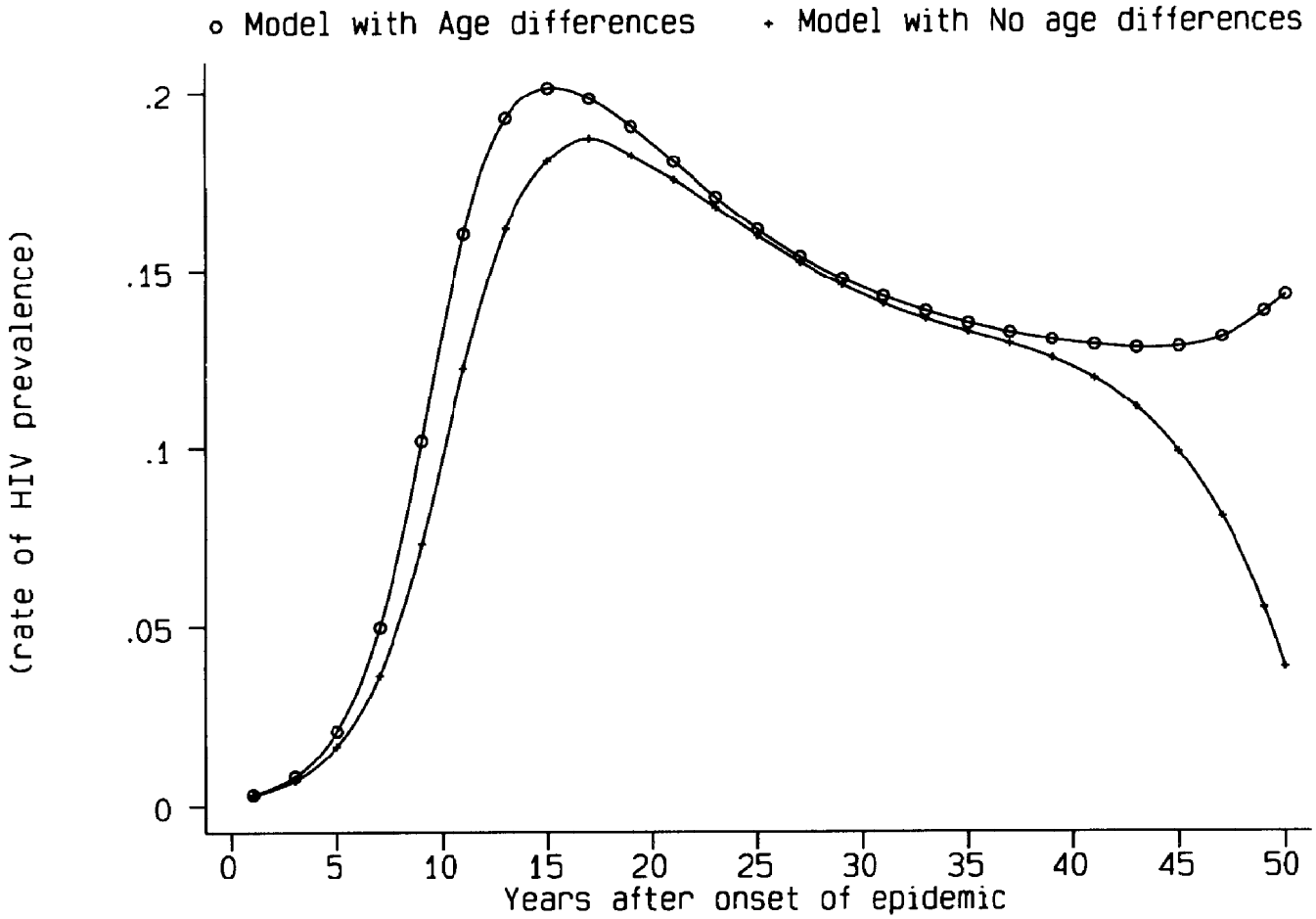


Figure 10: The effects of partners' age differences

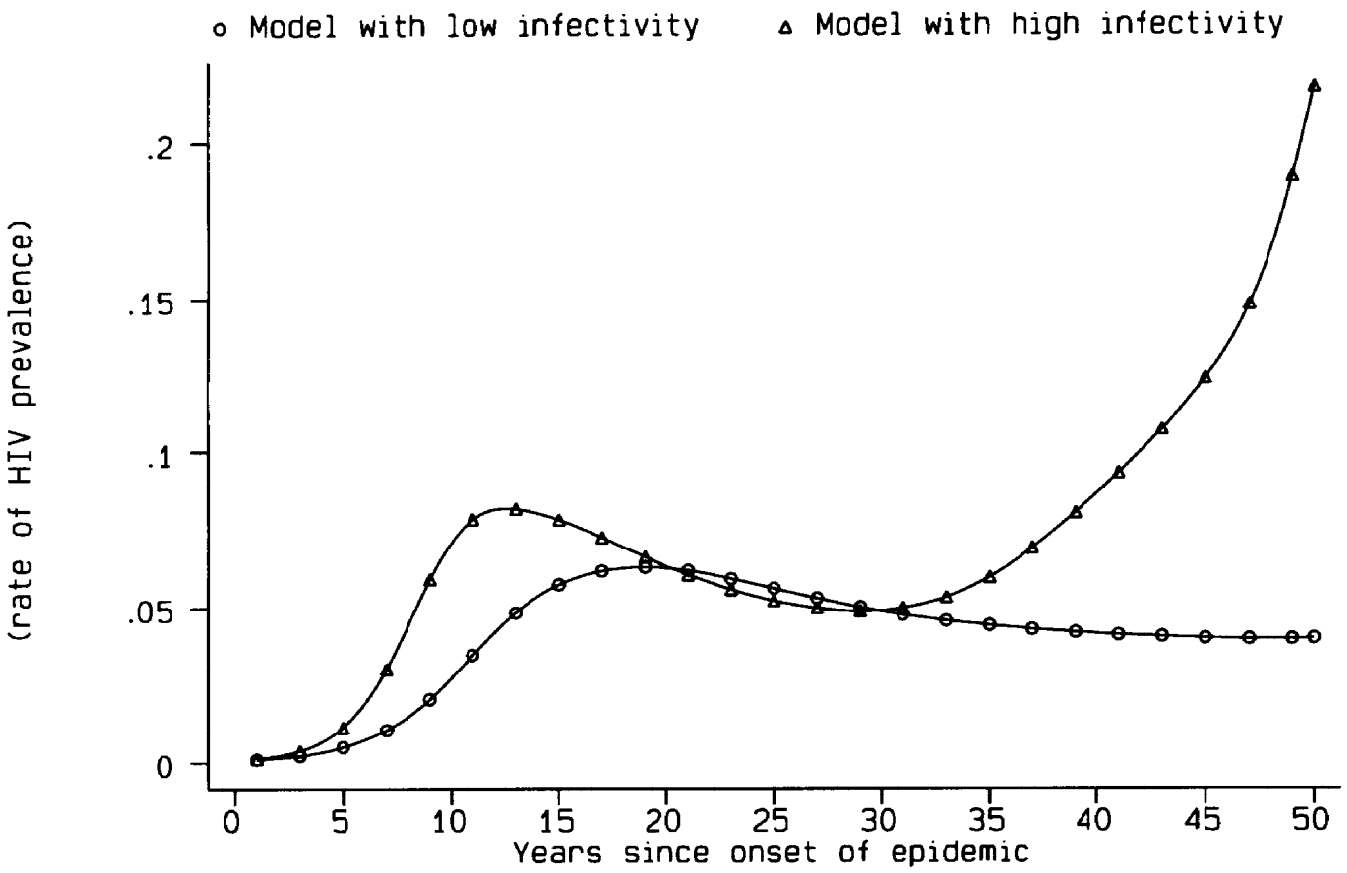


Figure 11: The effects of cofactors

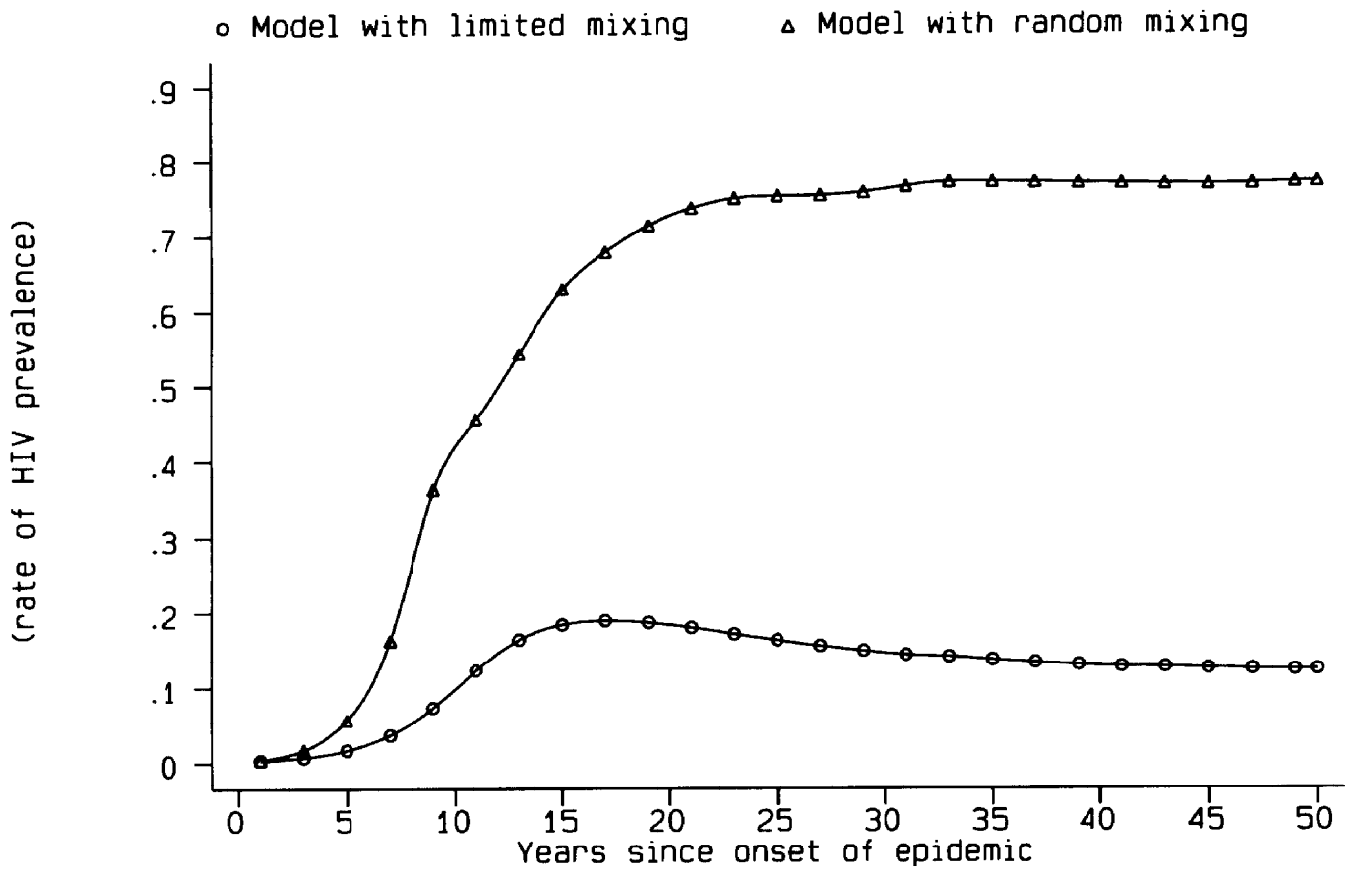


Figure 12: The effects of mixing rules

Appendix

Estimates of HIV/AIDS prevalence and the effects on mortality

This appendix describes the tedious but very simple operations employed to estimate ranges of values for HIV and AIDS prevalence and their effects on levels of children and adult mortality.

1. **Estimation of HIV and AIDS prevalence.**

Reported annual new cases for each country in each region were for several years. For some regions these reports extend back to 1978 (NA, WE, OC) but for others they start after 1982. These data on new AIDS cases were obtained from several reports ('The Current Global Situation of the HIV/AIDS Pandemic') issued by the Global Programme on AIDS (GPA) from The World Health Organization.

The reported number of cases were then adjusted for completeness and delays using completeness factors estimated as the mid-point of a range established by Delphi surveys (Mann et al., 1992). The adjusted annual figure of new AIDS cases were then cumulated to obtain cumulated cases by year.

These data were smoothed using two alternative functions: (a) a fourth-degree polynomial and b) a double exponential function. The results obtained from these two different smoothing procedures are referred to below as estimates from Method I and Method II respectively. The predicted values derived from the smoothing functions were used as input data to do a backward projection of cases to retrieve the underlying HIV incidence function, that is, an estimated yearly series of new HIV cases. The procedure used and the algorithm implemented were suggested and designed by Artzrouni (1990). To identify the unknown function describing new HIV cases it is necessary to assume that the incubation function is a Weibull. In order to make room for additional uncertainty, I assumed two alternative Weibull distribution functions differing in the degree of symmetry only. The median

incubation time was assumed to be about 10 years but the shape parameter of the Weibull was assumed to be between 1.25 and 2.50. The Weibull distribution functions contained in this range change from least to most symmetric.

Combining methods I and II with the two alternative assumptions about the incubation function leads to a range of estimated HIV cases. The lower and upper bound of the epidemic were then identified and used as the lower and upper bound of the ranges displayed in Tables 2a through 2d.

I then allocated total number of cases among children (younger than 5) and adults (over 15) using ratios of children to adult HIV cases for each region. These ratios were obtained from estimates made by GPA (WHO) according to the typical patterns of HIV prevailing in the region. Although it introduces mild errors, particularly where the epidemic has been changing rapidly, the child/adult ratios that I utilize are invariant over time. To obtain the number of individuals with AIDS at time t, a(t), I used the following expression:

$$a(t)= \int_o A(t-d)\, \Gamma(d)\, dd$$

where A(t-d) is the number of individuals who are diagnosed with AIDS at time t-d and $\Gamma(d)$ is the survival function evaluated at d years for those with diagnosed AIDS. This survival function is estimated assuming that the force of mortality for an individual with AIDS is constant, independent of age, and determining a half life of 2 years.

To obtain estimates for the number of individuals infected with HIV at time t, I(t), I use the following expression:

$$I(t)= \int_o I(t-d)\, \Pi(d)\, dd$$

where I(t) is the number of individuals who become infected at time t-d and $\Pi(d)$ is the survival function associated with the combined risks of incubation and mortality. I assume that the incubation function is a Weibull with a Median of 10 years and shape parameter equal to 2 (the midpoint of the

range used before). For adults the force of mortality was set equal to the average risk between ages 15 to 50 in a life table from the West model of the Coale and Demeny system with a life expectancy equal to those estimated by the United Nations for each country. To introduce mortality it was assumed that all individuals are infected at around 25, the mean age of several HIV prevalence functions. To obtain estimates of HIV and AIDS prevalence, the estimates of a(t) and I(t) were divided by estimates of the adult population (15-49) and of the number of children younger than 5 years.

2. **Estimates of excess mortality.**

The cumulated number of deaths associated with AIDS were obtained separately for children and adults for each country in each region. For children we assumed that all those infected with HIV became AIDS and died before reaching age 5. This quantity multiplied by the probability of surviving between 0 and 5 in the life table (without AIDS) for the corresponding country leads to an estimate of the **excess child deaths** due to AIDS.

For adults we calculated the number of deaths associated with AIDS at time t as follows:

$$d(t)= \int_o A(t-d)\, \Gamma(d)\, \mu_3(d)\, dd$$

where A(t-d) is the number of new AIDS cases in year t-d, $\Pi(d)$ is the probability of surviving d years with AIDS and $\mu_3(d)$ is the force of mortality due to AIDS. To estimate the number of **excess** deaths due to AIDS we multiplied $D_a(t)$ by ${}_{35}P_{15}$, the probability of surviving between ages 15 and 50 in the life table (without AIDS) for each country.

These calculations for children and adult were performed for each year between 1985 and 1995 and the estimated excess deaths cumulated over time. We then divided the cumulated excess deaths among children and adults by the estimated mid-period population in the age groups 0-4 and 15-49 to arrive at the estimates of proportionate changes in child and adult mortality rates and of absolute changes in life expectancies displayed in Table 2d.

ENDNOTES

1. Throughout this essay and unless explicitly stated otherwise we only focus on HIV-1, the dominant form of the HIV virus. Other forms of the virus, such as HIV-2, are also present in various parts of the world but are much less prevalent.

2. The exact nature of the incubation process is also under scrutiny. Until recently it was believed that long post-infection periods of low HIV blood counts suggested that shortly after infection (after the first two weeks) HIV was dormant and concealed, neither attacking the body's immune system nor reproducing in massive scale. However, new research indicates that HIV infects and destroys CD4 cells from the moment it enters the body. The explanation for low HIV bloodstream counts, except immediately after infection and at the onset of ARC or AIDS, appears to be that CD4 infected cells and free HIV particles do not circulate freely in the blood but hide in lymph nodes, tonsils, spleen and other lymphoid tissues, at least until these tissues have been destroyed by the virus (NIH, 1993).

3. Recent experience with the outbreak of Ebola virus in Zaire is an example of the operation of an infectious agent which combines high levels of lethality with a 'non-intelligent' reproduction strategy that relies on incubation periods shorter than two weeks. With such short intervals between contraction of infection and the nearly always fatal and dramatic collapse of the human host associated with Ebola, containment of the spread of the virus is not difficult if proper quarantine measures are applied in a timely way.

4. See the Appendix for a summary of procedures used to obtain the estimates.

5. An alternative explanation is that more recent epidemics (roughly those that appear to proceed at faster speed) are driven by an HIV-1 type of virus that is more easily transmittable than the original one. Since HIV-1 is a highly adaptable and mutable virus (Temin, 1989), this remains a distinct possibility but as yet there is no evidence to confirm the conjecture. A further possibility is that the more recent spread is fueled by higher infectivity attributable to shifts in the transmission mechanism (Mastro et al., 1994).

6. The estimates in the various panels of Table 2 should be interpreted cautiously since they are based on assumptions that are difficult to verify. Indeed, alternative estimates of the underlying HIV epidemic do exist and although there are broad similarities with those presented here, there are also important differences (Mann et al., 1992). The standard errors and ranges associated with our estimates should provide an idea of the magnitude of uncertainty we are dealing with in Table 2.

7. It is important to calibrate the estimates of the impact of HIV/AIDS on mortality presented in Table 2d with independent estimates. First, we can compare the number of cumulative deaths associated with AIDS that underlie the figures displayed in Table 2d with similar quantities calculated by Mann and collaborators and by the World Health Organization for similar regions. These are shown below for selected regions:

Cumulative number of deaths due to AIDS: 1985-1995 (in 1,000s) in different regions:

	North America	Western Europe	Latin America	Sub-Saharan Africa	Southeast Asia
Table 2d	543	103	580	2,555	195
AIDSIW (Mann)	291	107	243	2,369	92
WHO	400	100	300	1,900	100

All figures refer to cumulative deaths to the end of 1994. AIDSIW refers to figures estimated in Mann et al. (1992). The WHO figures for Southeast Asia refer to Asia, not just Southeast Asia as the other estimates do. Second, cruder alternative estimate of the proportionate increase in child mortality induced by HIV/AIDS can be derived from first principles and calculated by way of contrast. This estimate is only applicable where HIV is transmitted heterosexually and requires knowledge of adult HIV prevalence and the probability of mother-to-infant transmission. When adult prevalence is about .03 and the probability of vertical transmission hovers around .30, the proportionate increase in child mortality should be between .01 and .10 when, as it occurs in Sub-Saharan Africa, Latin America and the Caribbean, the death rate in the age group 0-4 is within the range .0050-.0350. The point estimates in Table 2d fall within this range.

8. An important feature of the epidemic is that it is rarely confined within the narrow boundaries of a particular social class. Instead, HIV may affect members of the elite and the lower classes alike. Although the evidence regarding the association between HIV prevalence and social class is patchy at best, in Africa there are indications of a positive association (Wawer et al., 1991; Serwadda et al., 1992; Ryder et al., 1990; Bugingo et al., 1988; Melbye et al., 1986). The distribution of cases by social classes is obviously a function of the predominant mode(s) of transmission and the association between relevant sexual behaviors and social class membership.

9. By ‘low’ and ‘high’ risk group I refer to groups whose members engage in behaviors that are less (more) likely to lead to contact with one or more means of transmission. Intravenous drug users (IV users) prostitutes, homosexuals and bisexuals are all high risk groups. Members of the general population such as blood donors (voluntary or not) and pregnant women are considered to be low risk groups. Patients in STD clinics have ambiguous status although they are more likely to resemble high risk groups either because STD’s reflect high risk behavior or because STD’s are themselves a co-factor in the contraction of the infection. In order to draw more robust inferences, I treat these three groups (high and low risk and STD patients) separately.

10. ‘Contact’ refers very generally to any type of interaction between the groups, be it direct (such as sexual contact) or indirect (such as blood transfusion or needle punctures). For the most part, however, direct contact is the most relevant of the two.

11. Exploration of these data involved resolution of a series of fairly delicate issues. I will mention only two of them. The first and most important is that for each risk group the database includes multiple observations for each year. After excluding the estimates corresponding to samples with less than 100 observations and with unknown or uncertain date, I calculated the average and standard deviation of the remaining estimates corresponding to a calendar year. The regressions are weighted regressions of these averages where the weights are the reciprocal of the variances associated with each mean. The second problem is to decide on a reasonable regionalization. Sub-Saharan Africa includes all countries from East and West Africa traditionally known to be in Sub-

Saharan Africa. It excludes South Africa. There were only two observations for Southeast Asia (Thailand and India) that I considered reliable enough for inclusion. These two observations were added to those from South and Central America to constitute a second region on the grounds that the corresponding epidemics have similar patterns. The last region was the Caribbean. The third problem is to determine the time points that can be safely analyzed. Although for some countries at least, one can construct a time series from the early 1980's until the mid 1990's, the reliability of the information is probably not high. Furthermore, only a few countries have complete information. To solve this, I pooled all the information pertaining to years 1987 and earlier and the information pertaining to years after 1987. This was done in the belief that an average over time points would produce a more robust representation of prevalence for the corresponding period. The cutting point is admittedly arbitrary though it was chosen to coincide with the timing of stages described in Table 1.

12. Other regression models were are also tried but on the whole they were less successful. The most important among them is one where only current prevalence is taken into account (no lags are involved) and where the predictors are the prevalence estimates in all three high risk groups. Although the fit changes, none of the additional effects is statistically significant.

13. The clinical definition of AIDS has changed over time and is slightly different in various regions of the world.

14. An important set of parameters that are not explicitly introduced in the model in Figure 1 refer to the ultimate proportion of individuals who will experience a given transition. While this is not problematic in relation to the transitions to the absorbing state, it causes a not insignificant amount of trouble in relation to the flows from Healthy to Infected and from this to AIDS. First, our knowledge about the dynamics of HIV transmission and of its natural history does not enable us to state unequivocally whether or not anybody can be spared infection even if exposed to it. Second, the assumption is frequently made that once an individual is infected he or she will always develop full-blown AIDS. Over time this assumption has been subject to assault since there is growing evidence indicating that a minority of infected individuals at least may never develop AIDS. The results of models are sensitive to specification of these quantities but we have little knowledge to even hazard a guess about them.

15. Although for simplicity our description does not suggest it, there are some dependencies between the parameters. For example, the incubation process is strongly related to the mechanism of infection.

16. μ_3 may also depend on individual characteristics, environmental conditions and chemotherapeutic interventions. However, the variability of μ_3 is known to be fairly minor and inconsequential for the purpose of population projection.

17. Imperfections of the data are quite widespread. A considerable amount of effort has been invested, for example, to develop procedures for dealing with delays in reporting while very little, beyond informal appraisals, can be done to correct for systematic under-reporting. More tractable but cumbersome are procedures to handle changes in definitions of AIDS over time.

18. Note that equation (2) can, if we so desire, be specialized to selected age groups.

19. If the assumption is inappropriate it is not difficult to introduce mortality in the model but at the expense of some heavy-handed assumptions about the age distribution of infected individuals

and the actual level and age patterns of the force of mortality that applies to asymptomatic infected individuals.

20. This exercise was also performed with observed data for Sub-Saharan Africa and Western Europe. Although the contrasts are much the same as those obtained with simulated data, their overall profile is less sharp since the data are affected by severely changing coverage levels of reporting.

21. Non-parametric procedures, such as those suggested by Michalski and Yashin or by Brookmeyer and Gail, have the potential to reduce errors due to misidentification of the underlying trend in new HIV cases but they are sensitive to mis-identification of the underlying incubation function.

22. An alternative formulation has been presented, discussed and mathematically justified, first by May, Anderson and McLean (1988) and then more thoroughly by Anderson and May (1991). These authors pioneered the mathematical investigation of long- (asymptotic) and short-term population consequences of HIV/AIDS in populations where HIV/AIDS is transmitted either heterosexually or homosexually.

23. This is true whether one deals with models that are deterministic or probabilistic. The bulk of models formulated so far are of a deterministic kind. Some (for example, the model by Auvert and colleagues (Auvert, 1994)) are probabilistic in the sense that they rely on Monte Carlo microsimulations.

24. Basia Zaba was first to introduce explicitly this metric in a very interesting paper reviewing possible extensions of demographic models for HIV/AIDS (Zaba, 1994).

25. Throughout, I assume that HIV affects mortality only by increasing the force of mortality among those who become AIDS victims. I neglect all effects that HIV may have on fertility. As argued by Zaba (1994), this is an unrealistic assumption.

26. To be sure, the existence of simplifying relations such as the one derived here is not new. Bongaarts and Way (1989) have already pointed out to a very close relation between levels of adult prevalence and changes in the crude death rate. What is new in this presentation is that the relation is derived from first principles of a generalized stable population.

27. This approximation rests on the simplifying notion that (1-exp(n*q)), the probability of not observing an event in n binomial trial with probability of success q is approximately equal to n*q when q is very small. In our case n is C_{ij} and q is B_j.

28. Anderson and colleagues (Anderson et al., 1986) showed that C can be approximated by $m+\sigma^2/m$ where m and σ are the mean and standard deviation of the distribution by number of partners.

29. Anderson (1992) reported similar results, though in his simulation it does not appear to be the case that the epidemic withers away once the age differences between partners becomes 0. It is possible that this occurs because in his models there are sources of infection other than heterosexual contacts or, alternatively, because children who are born infected survive past age 15. Similar results have been reported by Brouard (1994).

30. Societies that practice polygyny, for example, may have larger age differences between partners, but the effect that this has on the HIV/AIDS epidemic could be offset in part by reduced frequency of extramarital partners or by lower availability of young female partners for young males.

31. Casterline et al. (1986) studied the patterns of age differences between partners in union in several countries. The information for Sub-Saharan Africa reported there, however, is insufficient to verify the conjecture about a more modest HIV/AIDS epidemic when age differences are of lower magnitude.

32. Although not always conceptualized as a co-factor, condom use also operates on $\lambda(x,t)$ by directly reducing B. The amount of the reduction depends on the effectiveness of usage.

33. An important element to characterize patterns of sexual behavior is to emphasize the contrast between social groups who deviate from monogamy by recruiting sexual partners among commercial sex workers and social groups who recruit additional partners from other groups. This distinction is helpful in Africa where contacts with prostitutes among urban males is widespread in some societies of the Eastern belt of Sub-Saharan Africa (Caldwell and Caldwell, 1994; Larson, 1989).