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Alberto Palloni
Etienne Van de Walle
Luis Lamas
George Kephart
John Marcotte

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Alberto Palloni
Center for Demography and Ecology
University of Wisconsin

Etienne Van de Walle
Population Studies Center
University of Pennsylvania

Luis Lamas
Facultad de Economicas
University of Santiago (Spain)

George Kephart
Center for Demography and Ecology
University of Wisconsin

John Marcotte
Center for Demography and Ecology
University of Wisconsin

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INTRODUCTION.

Very little is known about the numerical impact of the Acquired Immunodeficiency Syndrome (AIDS) epidemic in Africa. By and large, the speed of its diffusion appears to be quite rapid. In fact, whereas the disease was probably of minor import before 1980, it has been estimated by the World Health Organization that one to two million people are infected in Africa, and that a minimum of 10,000 cases annually may be occurring there (Population Reports, 1986). Weekly magazines such as Newsweek and Time have reported that the cumulated number of deaths since the epidemic began in earnest exceeds 50,000. Any future projection of the population of Sub-Saharan Africa will have to reckon with the epidemic.

In this paper we examine two alternative ways of projecting populations with characteristics similar to those of Eastern Africa (United Nations, 1986). In both cases the projection models are based on preliminary reviews of the situation in Africa (Population Reports, 1986; Nunn, 1987; Piot and Carael, 1988; Quinn, Mann, et al., 1986; Brunet and Ancelle, 1985) and on information on probabilities of infection and latency times provided by various other sources (May and Anderson, 1987; Darrow, Echenberg, et al., 1987). Since the estimates of most of the parameters that we use in our models have large variances attached

to them, the projection exercises that we undertake are not designed to predict the future course of populations exposed to the epidemic. Instead our goal is to identify sets of strategic parameters, those which appear to be most important in determining the future trajectory of African populations. This type of exercise is useful not only because it will suggest where to concentrate our measuring efforts but also because it will support the task of designing targets for efficient public health policies. The models also rely on a set of simplifying behavioral assumptions. The realism of these assumptions may be questioned but their value is that they are explicit and can be changed as we accumulate information about the onset and progress of the disease in individuals and about the behaviors that foster its transmission in various subgroups of the population.

The main difference between the two projection models is the nature of the mechanism determining the diffusion of the epidemic. In the first model we assume that the incidence rate of HIV grows linearly over time and we postulate nothing about the underlying mechanisms that may lead to such linear progression. In the second model, we hypothesize three mechanisms of transmission of HIV at the individual level, each of them having its own dynamic. Here the progression of the disease over time is dependent on the operation of these mechanisms rather than being defined exogenously as it was in the first model. Preliminary results

obtained from this second model reveal that under a variety of quite realistic assumptions about the mechanisms of transmission, the incidence of the disease during the first 50 years of the projection will grow approximately as an exponential curve having a doubling time of about 35 years. The prevalence of the disease during the first 50 years of projection also grows exponentially with a doubling time of less than 35 years. The results of the second model reveal that the high lethality of AIDS diminishes the importance of sexual transmission relative to that of injections and transfusions. This is due to the fact that the infectiousness of successive generations takes a toll BEFORE they can enter the reproductive period. Both projection models, however, indicate that the short run impact (first 50 years) on the total rates of growth will be rather modest, implying in all but the most catastrophic scenarios relative reductions not larger than 35 percent and growth foregone not larger than 20 percent.

A SIMPLE MODEL.

a) Basic assumptions.

The projection is carried out by single years and separately for the non-infected and the infected populations. The non-infected births of the infected women are integrated in the non-infected population. The most important assumptions are as follows:

1. A growing incidence of HIV is infecting a part of the population which is then at risk of contracting the disease. We

assume that HIV yearly rate of infection (i.e., the proportion of new cases with respect to the total population) was equal to zero in 1980 and that it will grow linearly to reach 1 per cent in the year 2010. This assumption is in lieu of assumptions about mechanisms of transmission. It may be unrealistic to model the spread of this disease (as well as other infectious) but it permits us to implement a simple procedure to assess the magnitude of the impact on the rate of growth of the population experiencing the epidemic.

2. The new infected cases are distributed by age according to age-sex specific curves empirically estimated. These distributions show that the young, sexually active adults, particularly females, are at highest risk. They do not reflect, however, the bimodality of observed curves of HIV seroprevalence whereby very young children are at higher risk than individuals in early childhood and in their teens. The distributions were freely adapted from a distribution of AIDS cases for Kinshasa (Population Reports, 1986) and are displayed in Table 1.

Undoubtedly these distributions could be improved upon but our preliminary results suggest that their impact is only marginal in this simplified projection. Its impact is of even less import in the second projection model.

3. Infection can also occur from mothers to young children in the womb, during delivery or through breast milk. We assume that 30

per cent of their children are infected at birth.

There is some conflicting information on this issue. Nunn (1987:pp. 5) states that " sero-positive women have a 50 to 70 per cent chance of having an infected child, but whether the infection usually occurs in utero, during delivery, or post-partum is not known [...]." According to Population Reports (1986:pp. 205) "... how often transmission occurs in pregnancy is not certain. Researchers estimate that 20 to 50 per cent of infants born to infected mothers also will be infected..." The proportion that we use here appears to be on the conservative side and, hence, so will our estimates of the impact of the disease on the rates of growth.

4. We assume that the fertility of infected women is 80 per cent of the fertility of non-infected women. This reduction among infected women is attributable more to a change of behavior related to having acquired AIDS than to physiological losses of fecundity. Although asymptomatic infected women may not reduce their sexual activity or experience losses in fecundity, women who develop AIDS are likely to drastically curtail sexual activity thus reducing their fertility.

5. Mortality of the infected population is only dependent on AIDS. Factors such as age and sex are not considered. Half of the infected die within ten years following their infection with the survivors experiencing attrition at the same rate. No infected

individual survives past age 85. The probability that the year's infected newborns will survive past the first year of life is 90 per cent of the probability applicable to the non-infected newborns. This level of excess mortality is probably too low when one considers the apparently accelerated course of the disease among the very young (see quotation above). As for the rate of transition from HIV to AIDS and death, the assumption of a ten-year half-life is not strongly based on existing evidence which has not yet been accumulated for longer than ten years. We made the inference from evidence collected for the U.S. population. Thus, in Population Reports (1986:pp. 201) it is reported that each year about 8 to 10 per cent of asymptomatic infected persons develop some symptoms and that 2 to 10 per cent develop AIDS. In two of the longest U.S. studies, one-third of the infected males has developed either some symptoms or frank AIDS in 5 years. Survival time after diagnosis appears to be shorter in Africa than in the U.S. According to Nunn (1987:pp.7), "...preliminary work in Kinshasa suggests 1 percent symptomless HIV positive people will develop AIDS each year and 10 percent will develop AIDS related complex (ARC)...[about] one-quarter of patients with ARC go on to develop AIDS within three years..."

b) Selected results.

The results of the projection are summarized in Table 2. Various statistics associated with the projection with AIDS are

compared with those resulting from projections replicating the U.N. assumptions and with projections assuming stagnation of expectation of life at 1980-1985 levels. We also project the population with both stagnation of mortality improvements and AIDS. In a final variant we assume a higher (2 instead of 1 percent) incidence of HIV in the year 2010.

The results of these projections reveal that the AIDS epidemic has the potential to slow down the rate of growth of the population but that the losses in population growth are dwarfed by the actual growth that takes place as a consequence of the relative difference of fertility and mortality regimes. This conclusion could be altered only if the incidence rates were to reach levels higher than what can reasonably be predicted for this area of the world. Also, as is the case in all mortality crises, the impact could be much higher in smaller and more homogeneous populations such as those found in urban settings. By and large, this conclusion is in line with the results obtained in the study of the population impact of mortality crises (Watkins and Menken, 1985; Palloni, 1987).

In the next section we show that this conclusion has to be qualified somewhat when the rates of incidence are made endogenous to the mechanisms of transmission.

A PROJECTION MODEL RECOGNIZING MECHANISMS OF TRANSMISSION.

The foregoing projections made a number of simplifying assumptions which could influence the results. The most important refers to the dynamic of diffusion of the disease among the members of the population. It was assumed that the incidence of new cases (as a fraction of the total population) of HIV infection grew linearly. Although a simplified definition of incidence was a useful practical device to facilitate the projection under the first model, it is preferable to define the rate of incidence relative to the susceptible population and to distinguish incidence of asymptomatic infections from incidence of symptomatic infections. It may well be that a change of assumptions and the removal of simplifications would not alter significantly the main conclusion--that the epidemic has an ultimate effect on the total rate of increase of only modest magnitude-- but it behooves us to test whether or not this is the case.

Most epidemics proceed in such a way that new cases increase exponentially up to a certain point and that therafter they diminish rapidly as the population becomes saturated with non-susceptibles. It is very simple to change the assumption of linearly increasing incidence and replace it with an assumption that implies, for example, a logistic increase of prevalence. However, this would also produce a somewhat unsatisfactory model for several reasons. First, a logistic (or other reasonable growth

curve) would obscure the peculiar nature of the disease mainly determined by the limited forms of transmission of the agent producing it. Second, the model cannot be used to explicitly consider the timing of the process from infection to development of symptoms and from initial symptoms to eventual death. Although this is not a strategic consideration in the treatment of many other infectious diseases, it is central in the case of HIV infection given the latency times involved. Third, and most important, the model would have limited policy implications as it would reveal neither the most sensitive features of the process nor those that could be manipulated by social education or health preventative measures.

Other projection models such as the one suggested by Bulatao (1987) have explicitly considered the mechanisms of transmission but have not incorporated detailed age composition of the population and have relied on limited behavioral assumptions about one of the most important modes of transmission namely, sexual contacts. However, in this paper we incorporate several of Bulatao's insights about the dynamics of the disease.

In this part of our paper we present the essential features of a model that considers in detail the mechanisms of transmission, introduces age, and explicitly defines the various stages known to exist in the development of the disease. The complexity of the model requires the definition of many parameters whose proper

values cannot be reasonably estimated with the information currently available to us. However this, as any other projection, is not intended to predict the future but rather to explore which parameters involved can be considered the drivers of the process. By identifying them we can suggest where our measurement efforts should be directed and where our policies should be concentrated to optimize their implementation.

a) Basic Assumptions.

We assume that there are three relevant modes of transmission of HIV: at birth from an infected mother, through blood transfusions and injections, and through sexual contact. Initially, we assume that there is no drug addict population and that the transmission through injections is constrained to contact with infected needles or blood in hospitals and clinics. Although this assumption is quite acceptable for African populations it is certainly unrealistic in populations of other areas of the world. However, as will be evident from our discussion below, the assumption can be removed at the cost of introducing more clutter in the notation but without changing significantly the nature of the model.

Individuals below age X_1 and above age X_m, which signal respectively ages of entrance to and exit from a sexual market, can be infected only through birth or through injections and transfusions. Between these two ages it is assumed that

individuals can also be infected through sexual contact. Males who are in the sexual market are divided into the following groups: a) married strictly monogamous, b) married heterosexual non-promiscuous, c) married heterosexual promiscuous, d) married bisexual non-promiscuous, e) married bisexual promiscuous, f) married homosexual non-promiscuous, g) married homosexual promiscuous. Categories (b) through (g) also apply to the non married population of males. Females are divided in the same way but we do not allow for either bisexuality or homosexuality. The distinction between promiscuous and non-promiscuous is made in terms of the number of sexual partners per year and the number of sexual acts per partner per year. The promiscuous population is allowed to have not only a higher number of partners but also a higher number of sexual contacts per partner. If married, the non-promiscuous population is allowed to have a low number of sexual partners (other than the spouse) and an average number of sexual contacts most of which are with the spouse.

For each of the categories thus distinguished we create three states: non-infected at age x, asymptomatic infected at age x of duration d, and symptomatic infected at age x of duration d. The duration dimension of asymptomatic infectious refers to the time elapsed since the moment of infection. The duration dimension for symptomatic infectious is defined from the onset of full blown AIDS. Between these three states we define transitions illustrated

in Figure 1. The state of symptomatic infectious does not permit any transitions except to death due either to normal causes or to AIDS-related diseases. The state of asymptomatic infectious permits transitions to the AIDS state or to death with a force of mortality that is greater than for non-infected individuals. Finally, the state of non-infectious permits transitions to the state of asymptomatic infectious or to death with a 'normal' force of mortality. We also assume that although there can be contact between promiscuous and non-promiscuous, there are no changes of behavior, e.g., promiscuous individuals remain promiscuous throughout life. The same applies to heterosexuality (homosexuality) and to bisexuality.

The above formulation is a special case of more general models of increment-decrement tables or multistate projections. In what follows we define the corresponding rates and probabilities of transition between states.

b) Definition of transitions and of transition rates.

The types and timing of the events experienced during a year by an individual depend on the initial state occupied at the beginning of the year:

i) A non-infected individual at exact age x at time t is exposed to dyinge of normal causes (event 1) with risk $\mu(1,x)$ and of being infected (event i) with a risk $\mu(i,x)$, both applicable to age x. If he is infected, he is exposed to the risk of dying due to

normal causes with risk $\mu_1(1,x) = \mu(1,x)*(1+\alpha h(x))$ and to the risk of developing AIDS (event 2) with a risk $\mu(2,x,o)$ applicable to age x and to duration of infection d=o. If he develop AIDS he will be exposed to the risk of dying of normal causes for infectious, $\mu_1(1,x)$, and to the risks associated with AIDS (event 3), $\mu(3,x,o)$ defined for age x and for duration of AIDS o. The function h(x) is designed to make the excess mortality of infectious cases dependent on age, with higher excesses at the lower and higher segments of the age span. In our current design, h(x) is set equal to one and α is set equal to .10. Thus, the proportionate excesses of mortality are age-invariant.

ii) An infected individual at age x and with duration o of infection is exposed to the risks of dying of normal causes, $\mu_1(1,x)$, and to the risk of developing AIDS, $\mu(2,x,d)$. If he develops AIDS, he will be exposed to the risk of dying of normal causes, $\mu_1(1,x)$ and to the risk of dying of AIDS associated syndrome corresponding to age x and duration o, $\mu(3,x,o)$.

iii) An individual who attains age x and has experienced frank AIDS for d years, is exposed to the risk of dying due to normal causes, $\mu_1(1,x)$ and to the risk of dying of AIDS-associated causes, $\mu(3,x,d)$.

Four remarks are necessary here. First, the excesses of mortality experienced by individuals who are carriers but have not developed AIDS is predicated on the grounds that the infection

itself may partially damage the immune system and exacerbate the risks due to other diseases. Variability of the excess of mortality by age can be justified since the immune system of children and older people is more precarious than for individuals in the prime ages. Second, although strictly speaking there is no such thing as a cause of death due to AIDS, the definition of $\mu(3,x,d)$ is intended to capture the operation of causes of mortality that exist but have little significance under normal conditions (and as such are included in $\mu(1,x)$) and that are exacerbated by the presence of AIDS dwarfing the impact of the 'normal' causes which are NOT affected by the disease. Third, as we will show below, the risks that depend on age AND duration, $\mu(2,x,d)$ and $\mu(3,x,d)$, do so in a non-additive way: the magnitude of the risks depends on d as well as x-d, the age of onset either of the infection or of AIDS. For example, an individual who is infected at age 0 experiences a risk of developing the full- blown symptoms of AIDS at age 10 which is larger than the risk for an individual at age 30 who contracted the infection at age 20. Although both have the same duration, the onset of the infection occurred at different stages in their lives and the risks are defined to capture any differences that this may generate. The model is general enough that this feature can be easily omitted if available evidence does not support the contention of a connection between age of onset, duration and risks. Finally, the definition

of $\mu(i,x)$ includes the risks of being infected through the two sources defined before namely, injections (transfusions) and sexual contact. That is, $\mu(i,x) = \xi(x) + \rho(x)$, where the first term represents the risks of being infected through injection (transfusion) and the second term represents the risks of being infected through sexual contact. Both may be made dependent on age in ways that are explored below.

c) Definition of probabilities of transitions.

To simplify notation we will show how the projection works for males who are non-infected at age x and who pertain to one of the subgroups defined above. Straightforward extensions permit us to define the probabilities for individuals in any of the other groups and states. We will therefore omit all pertinent subscripts for sex, group and state.

The probability that a non-infected male aged x survives to the end of a year as a non-infected case is given by:

$$S_1(x+t) = \exp\left(- \int_x^{x+t} \theta(v)\, dv \right) \qquad (1)$$

where

$$\theta(v) = \mu(1,x) + \mu(i,x)$$

The probability that he will become infected during the year is:

$$P_1(x) = \int_0^1 \exp\left(- \int_x^{x+t} \theta(v)\, dv \right) * \mu(i,x+t)\, dt$$

Exchanging the roles of $\mu(1,x)$ and $\mu(i,x)$ we also obtain $P_2(t)$ the probability that he will die of normal causes. If the risks are constant within the interval, we obtain simple expressions for the probabilities:

$$P_1(x) = \frac{\mu(i,x)}{\mu(i,x)+\mu(1,x)} * (1 - \exp\{-(\mu(i,x)+ \mu(1,x))\}) \quad (3)$$

with a similar expression for $P_2(x)$. Note that the risks only depend on the age attained at the beginning of the year.

We now assume that all those who were infected became so in the middle of the year and that they are exposed to the transition to the AIDS state for only one-half of the year. With this and the assumption of constancy we obtain expressions for $P_3(x)$, the probability of developing AIDS and $P_4(x)$ the probability of dying of normal causes while experiencing the infection. For example, $P_3(x)$ is given by:

$$P_3(x) = \frac{\mu(2,x,o)}{\lambda(x,o)} * (\exp(-.5*\lambda(x,o)) - \exp(-\lambda(x,o)) \quad (4)$$

where $\lambda(x,o) = \mu_1(1,x) + \mu(2,x,o)$

The probability that neither transformation to AIDS nor death will occur and that, therefore, the individual will be an asymptomatic AIDS case at age x+1 with duration zero is:

$$S_2(x+1) = (1- (\exp(-.5*\lambda(x,o)) - \exp(\lambda(x,o)))) \quad (5)$$

Finally, we assume again that those who become symptomatic did so in the middle of the corresponding period and thereafter are exposed to $\mu_1(1,x)$ and $\mu(3,x,o)$, the risk of dying due to normal causes and the risk of dying due to AIDS related diseases at duration o, for only one-fourth of a year. For example, the expression for the probability of dying of AIDS related causes is:

$$P_5(x) = \frac{\mu(3,x,o)}{\gamma(x,o)} * (\exp(-.75*\gamma(x,o)) - \exp(-\gamma(x,o))) \quad (6)$$

where $\gamma(x,o) = \mu_1(1,x) + \mu(3,x,o)$

and the probability of surviving to the end of the year as an AIDS victim is:

$$S_3(x+1) = (1- (\exp(-.75*\gamma(x,o))-\exp(-\gamma(x,o)))) \qquad (7)$$

Similar expressions to those presented above apply to individuals who were asymptomatic infected at age x and duration d and to those who were symptomatic infected at duration d. The only change is that, where pertinent, the corresponding risks will be evaluated at duration d instead of at duration o.

d) Definition of parameters.

Although rather tedious and time consuming, application of a population projection relying on the transition probabilities defined above is a straightforward application of projections with increment-decrement tables. The difficult issue is how to define the main parameters of the model, e.g., those on which the transition rates depend. As others have noticed (Bulatao, 1987), values for many of the parameters have to be invented since there are no empirical estimates or those that exist have discouragingly wide confidence intervals. Yet, as we mentioned before, one of the tasks of the application of our models is to identify those which are key to the projection, the ones to which final results in the long and short run are most sensitive.

The complete version of our model contains a long list of

parameters some of which we feel fairly confident about and some others whose values are guessed. In what follows we define the dependency of the risks to a set of parameters. We do so for a particular case of the model, one where we assume that sexual contact is completely unconstrained by marriage and where there are no clusters created by homosexuality. Thus, the transmission of the infection via sexual contact is entirely dependent on a sexual market in which all individuals are heterosexual and where some are more promiscuous than others by having more partners and possibly engaging in more frequent sexual intercourse. Contact between promiscuous and non-promiscuous is allowed but there is no effort to establish consistency between the female and male behavior. It is important to note that in this special case of our model the possibilities of transmission are enhanced and hence the impact of the epidemic on population growth should attain an upper limit. This is an obvious result of the fact that recognizing monogamous marriages is tantamount to imposing constraints in the frequency of contacts and hence should result in a reduction of those exposed to the risk of being infected via sexual contact.

1. The probabilities of infection through sexual contact.

Males who are older than X_1 enter a sexual market with certain fixed preferences for females of various ages. These preferences are expressed as a distribution of females by age for a male aged x. Thus, a male aged x will search for females aged x-k with a

probability $\delta(k)$ where k is allowed to vary between -2 and 2. The distribution is weighted towards k=-2 and k=-1. For females we use the same distribution but with symmetric loadings, namely, the higher weights are at k=1 and k=2 to reflect the general practice of males being relatively older than females in couple formation. A contact will occur depending on whether females in the corresponding ages are or not within the sexual market. We define this as a function that grows linearly between 10 and 18 to attain and maintain until age 60 a value equal to 1. Thereafter it declines linearly to reach a value of zero at age 70. This function is identical for males and females although one could modify this to recreate the convention of a younger age of entrance and exit for females. For a male aged x the effective number of contacts with an infected female at any point in time during the year is defined as:

$$Ei(x) = \left\{ \sum_{k=-2}^{k=2} (\delta_k * Fi(x+k) * mkt(x+k)) \right\}$$

where Fi(x+k) represents the number of infected females aged (x+k), mkt(x+k) is the probability of being in the sexual market and, δ_k is the parameter expressing preference for age (x+k). Replacing Fi(x+k) by the number of non-infected females, Fni(x+k), leads to the expression for Eni(x), the effective number of contacts with non-infectious females. The ratio of Ei(x) to the

sum of Ei(x) and Eni(x) is the probability of entering in contact with an infectious female, Ci(x). Since the population of females is divided into promiscuous and non-promiscuous, we have in reality a couple of expressions representing respectively the probabilities of entering in contact with promiscuous and non-promiscuous infected females. We denote these probabilities as Ci(1,x)) and Ci(2,x). Weighting these expressions by a preference parameter representing the probability of searching among promiscuous females leads to the total probability of entering in contact with an infectious female. Thus for a promiscuous male we have:

$$CPMi(x) = \beta * Ci(1,x) + (1-\beta)* Ci(2,x)$$

and for non-promiscuous males

$$CNPMi(x) = \eta * Ci(1,x) + (1-\eta)* Ci(2,x)$$

Analogous definitions lead to the probabilities for promiscuous and non-promiscuous females aged x of entering in contact with infectious males, CPFi(x) and CNPFi(x). The parameters β and η control the degree of contact between the two populations. In case of no communication one can set β=1 and η=0. Less extreme

situations are represented by intermediate values of the parameters. For any fixed value of β, the larger the value of η, the more rapid will be the transmission of the disease and the sooner will the population approach a point of saturation. In the current version of the model we do not model the dependency between these two parameters although it is clear that when one of them increases to a unit value the other should attain its minimum of zero.

To calculate the total probability of infection we need to specify the following: a) the probability of using some protection (for example a condom), b) the efficiency of the protection, c) the probability of infection in case of no protection or of failure of protection, d) the number of contacts per person per year, and e) the number of partners per year.
Denote p(x) the probability of using protection, e the efficiency, tmf and tfm the probabilities of transmission from male to female and from female to male and, n the number of contacts and k the number of partners. The global probability of being infected during the year for a promiscuous male aged x is then:

$$Probmale(1,x)=[1-(1-CPMi(x)*(1-(1-(1-\Psi)^{n})^{k}]$$

where Ψ is the quantity (1-p(x)*e)*tfm. Similar expressions apply for the probabilities of non-promiscuous males, Probmales(2,x),

and to those for promiscuous and non-promiscuous females, Probfemales(1,x) and Probfemales(2,x).
Since these probabilities are conditional (on participation in the sexual market), the unconditional probabilities of infection are found by multiplying the above quantities by mkt(x).

In the current version of our model, individuals who develop AIDS are excluded from the pool of potential partners on the grounds that they develop markers which are easily identifiable by the rest of the population. Given the high lethality of AIDS, their inclusion would add little to the speed of diffusion of the disease. We have also simplified the model by assuming that the probabilities of transmission, tmf and tfm, are independent of duration of infection. Although there is some evidence suggesting that infectiousness is duration-dependent, we have little grounds to specify the corresponding function and have decided to utilize a duration invariant probability of transmission.

Suitable modifications of these formulae permit us to deal with the case where one introduces different groups depending on sexual preferences and constraints due to marriage. In the case of a married, strictly monogamous couple, the probability of infection of one of the spouses only depends on whether the other spouse was infected either before marriage or after marriage through injection or transfusion. In the case of a heterosexual, non- promiscuous couple, the probability of the male being

infected depends on a quantity analogous to Probmale(2,x) and the added probability of the spouse being infected appropriately modified to take into account the frequency of intercourse between them. The only two changes that are introduced when we recognize several groups are first that the pool of possible partners has to be restricted according to sexual preferences and, second that a probability of being infected by the spouse for those married has to be added. These modifications will always operate slowing down the rate of transmission of the disease. Thus the current version of the model will produce an upper limit of prevalence and incidence. Admittedly, it cannot be used to identify target groups except in the very restricted sense defined by the dimension of promiscuity.

2. The probabilities of infection through injections and transfusions.

The calculation of this probability will depend on the average number of injections per year, the proportion of unsterilized needles, the probability of transmission through a needle and, most importantly, the total proportion of infected population (symptomatic and asymptomatic). The most difficult part here is to establish a relatively accurate value for the number of injections and transfusions per year. It seems quite reasonable to assume that they should vary by age thus reflecting the different intensity of health problems over the life-time of individuals. In

fact, one could make their frequency be proportional to the force of mortality which is in itself a rough indicator of morbidity. Further, it is also necessary to separate the role of injections from the role of transfusions since the corresponding probabilities of transmission differ sharply. However, in the absence of exogenous information, we have decided to pool together injections and transfusions and make their frequency constant by age. In future versions of these models we plan to relax these admittedly restrictive assumptions. Let j denote the average number of injections or transfussions per year. The probability of being infected through them can be calculated as:

$$\text{Probinj}(x) = [\, 1-(1-\Phi)^{j} \,]$$

where Φ is the product of the proportion of unsterilized needles, the fraction of the total population who is infected and the actual probability of transmission. Here, as in the case of the probabilities of transmission by sexual contact, we exclude from the pool of infectious those who have developed AIDS.

3) The transition rate from asymptomatic infectious to AIDS.

Undoubtedly this is one of the most crucial quantities in the model, particularly if removal from the pool of carriers follows

as a result of developing AIDS. There have been several attempts to calculate the distribution of the waiting time from infection to AIDS but all are marred by severe left and right censoring problems. There seems to be, however, some agreement about the relative speed of the transformation. Several estimates suggest that between 30 and 50 per cent of those infected go on to develop frank AIDS before the end of the fifth year. It is reasonable to assume that this transformation depends on age but this has not, to the best of our knowledge, been considered in the estimation procedures.

In order to develop a plausible function we proceed in two stages. First, we assume that for each individual the risk of developing AIDS grows exponentially with duration since infection with a slope parameter κ but that individuals differ with respect to the intensity of the risk. If we assume that the parameter measuring the intensity of the risk has a gamma distribution with mean r/σ, then the average risks and survival functions are respectively given by :

$$\mu(2,d) = \left[\frac{\kappa \exp(\kappa * d) * r}{-1 + \exp(\kappa * d) + \kappa * \sigma} \right]$$

$$S(2,d) = \left[\frac{\kappa * \sigma}{-1 + \exp(\kappa * d) + \kappa * \sigma} \right] ** r$$

In the projection exercise that we carry out we have set r equal to unity. The value of the shape parameter, κ, is the

asymptotic value of the risk and controls the degree of curvature but influences little the median latency times. We have fixed its value to be .07 in all projections. To make the function dependent on the age at which infection occurs, one can assign different values to σ depending on whether the age at infection falls within the range of prime ages (say between 10 and 70). To take into account the fact that not all individuals will eventualy develop AIDS, we multiply the survival function by alternative values representing the proportion that does not develop the disease.

4.The force of mortality due to AIDS.

We adopt the same developments suggested before and assign parameter values to ensure that the survival function yields values that are roughly consistent with those observed in a variety of contexts. The values of the parameters are changed to reflect the effects of the age of onset of the full-blown symptoms. They are such that the median survival times are between 3 and 4 years when the age of onset falls within the range 10-70 and between 1 and 2 years when the age of onset falls outside that range. We assume throughout that, in the absence of other causes, all individuals developing AIDS eventually die of it.

5. Assumptions about fertility and transmission at birth.

Although it is possible to distinguish between fertility of infected and non-infected women, we will assume that they are identical. This is likely to have only minor consequences for a

couple of reasons. First, women who have developed AIDS are withdrawn from the pool of possible mothers since we assume (see above) that they are not elegible sexual partners. The result of this is to effectively reduce the net fertility rate of a cohort. Second, given relatively short latency times and high lethality of AIDS, children born infected in one period are highly unlikely to reach reproductive ages. Thus, it makes little difference if their 'potential' fertility is assigned a value equal to or smaller than that of the healthy population. The decision can be questioned only on the grounds that asymptomatic infectious women may have lower fertility. This can occur either because of physiological reasons or because infected women are selectively drawn from a pool of women who control fertility more efficiently or, alternatively, engage less frequently in sexual intercourse as a result of the infection. We know of no study showing that infectiousness has a deleterious effect on fecundability although there is evidence suggesting that asymptomatic but especially symptomatic infectious women have a higher rate of pregnacy wastage (Piot and Carael, 1988). The other possibility, namely, that infectious women are selected in terms of efficiency of birthcontrol practices cannot be considered unless one partitions the population JOINTLY by promiscuity and practice of birth control. It is unlikely, however, that this fine tuning will add significantly to the precision of the projection.

Estimates of the probability of being infected at birth by an infected mother vary widely. Further, it is unclear what mechanism of transmission among the three alternative ones (in utero, at delivery and through lactation) is most important. In what follows we have adopted a value of .70 and assumed that the infection of the newborn, if it occurs at all, does so at birth (age o).

e) The dynamics of the projection.

For every year of the projection the model produces total population, and its distribution by single years of age and by states. In addition, it yields the DURATION DISTRIBUTION BY AGE of the infected and symptomatic population. These are quantities of obvious importance for public health policy purposes. It also retrieves life tables for each of the subpopulations and calculates total, single decrement and associated multiple decrement life tables. Finally, it generates all the relevant rates: crude birth rates, crude death rates, incidence rates (of infectious and AIDS) and prevalence rates (of infectious and AIDS). The model also permits the calculation of the fraction of total expected years of life to be lived in each of the three states in a stationary population. These quantities are analogous to the expected durations before leaving a state calculated in increment-decrement life tables and solely dependent on the nature of the risks, not on the age structure of the population. They serve to gauge the intensity and age profile of the disease and

its lethality in much the same way as the life expectancy serves to summarize the intensity and age distribution of deaths in a stationary population.

If the values of the parameters were to remain constant, the outcomes can only be three: the population disappears, the disease is halted or there are cyclical fluctuations.The latter alternative is possible since the density of asymptomatic infectious in one period depends on the density of asymptomatic infectious in past periods. The formal properties of a population characterized by the prevalence of this type of disease will not be explored here but they are generalizations of those of stable populations subjected to fluctuating rates. Examination of these formal properties should prove useful to confirm some interesting conjectures. It is very likely, for example, that one important driver of the process is the variance of the ages of partners. If everything remains constant, the disease could be terminated in one generation if there were no variance of ages of partners and if injections and transfusion were NOT an independent source of transmission. The disease will linger longer when the variance of ages of partners is large and/or when the transmission by injection and transfusion is maintained at non-trivial levels. Although we do not experiment with the variance of the age distribution of partners, the conjecture is partially confirmed by the results of the projection.

f) Selected results.

We have projected forward an initial population resembling a stable population from model North, level 13, of the Coale-Demeny stable models. The initial parameters of this population are displayed in Table 3a. This population is very similar to that employed in the projection we carried out in the first part of the paper. We have assumed that fertility and 'normal' mortality remain constant throughout the projection. e.g. that there is no fertility reduction and that there are no improvements in mortality. This facilitates the assessment of the pure impact of the prevalence of the disease. Although the projection is carried out for only 50 years and is thus inappropiate to judge the long run behavior of the system, we thought that it was a sufficiently long period of time to observe the most important consequences of the epidemic and to inform the most urgent health policy decisions. In all cases we have assumed that .01 of the initial population is infected and that their age distribution is identical to that presented in Table 1. To avoid complications we have assumed that all infectious individuals at the beginning of the projection are at duration 0 and that none of them has developed AIDS. Changing the initial proportion of infectious will only move forward the horizon of the projection, making the consequences observable in 50 years less severe if we assign to it a lower value and more severe if value is higher. Changing the

duration distribution of the original infectious population will increase the incidence of AIDS and hence the levels of mortality experienced during the first 50 years. Since we are interested in the relative changes of selected outcomes, neither of these decisions is crucial. Furthermore, they can be easily altered to fit better the conditions observed in any country of interest.

To facilitate comparisons, we have carried out a 'baseline' projection with the parameters presented in Table 3b. To assess the effects of all other projections we compare results with those of the baseline. In addition, we have carried out calculations for three severe scenarios to show what are the values of the parameters that may lead to virtual extinction of a population in a relatively short period of time. Whether or not these values are realistic is an open question that could be answered only bygathering more precise information.

The first column of Table 4 displays selected indicators of the population after 50 years of projection. Thus, at the 50th year the actual rate of growth is 26.7 per thousand, the life expectancy (for females) has been reduced to 45.9 years, the joint prevalence of infection and AIDS has been multiplied by four reaching a level of 41 per thousand and the rate of incidence of infectious cases attains a level of almost 4 per thousand while that of AIDS is approximately 3 per thousand. The observed population growth during the period is 27.8 per thousand

implying a seven percent of growth foregone due to the existence of the disease (figure in the last row).

The second and third columns of the same table display the results obtained when the proportion of the actively sexual population using a condom is, respectively, decreased to .10 and increased to .90. Given the condom efficiency (.80) and the postulated mechanisms of sexual contacts, the impact of condom usage appears to be quite modest if judged by the percent of growth foregone: 9 and 6 percent for low and high usage respectively. However, the indexes of prevalence differ sharply as do the indicators of incidence. Low usage leads to incidence and prevalence that are at least three times as high as in the case of high usage and to losses of approximately 5 years of life expectancy. The contrasts in the growth of prevalence are shown in Figure 2 where the proportions of the asymptomatic and symptomatic population are plotted every five years for the projections corresponding to columns 1 through 3 (Series A through C in the graph).

As revealed by a comparison of columns 4,5 and 6, the effect of condom usage is strengthened when the latency times are increased. In fact, the projections summarized by these columns were generated with a median latency time from onset of infection to development of AIDS of about 12 to 13 years rather than the 7 to 8 years used in the baseline projection. High usage of condoms

saves close to 6 years of life expectancy. Under the regime of high usage the growth foregone is 7 percent and in the low usage regime it attains a level of about 12 percent. As before, incidence and prevalence of the infection and AIDS are at least three times higher in the low usage than in the high usage regime. As one would expect, the contrast between any one of the columns associated with low latency times and any one of the columns associated with high latency times reveals that the incidence and prevalence of the infection is higher the longer the latency times. This is dramatically demonstrated by the figures in Column 7. These were obtained assuming very short latency times (a median of about 3 to 4 years). Note that by the end of the 50th year prevalence of the disease has fallen to about half of the initial values (and continues falling rapidly thereafter). The growth foregone and losses in life expectancy are less than in the best of cases (high usage) with long latency times. Short latency and high lethality combine to limit the spread of the disease and to attenuate its impact on the vital rates. In Figure 3 we plot in five year intervals the proportions of asymptomatic and symptomatic that correspond to the projections summarized in columns 1, 4 and 7 (series A,B and C in the graph).

The first column of Table 5 displays the results that are obtained changing the force of mortality associated with AIDS so that in the worst case (when onset of AIDS occurs at ages below 10

or above 70) the median duration of life is between 2 and 3 years and in the best case (onset occurs during the prime ages) the median duration of life is between 6 and 7 years. Not surprisingly, this column shows that, after 50 years, life expectancy is somewhat higher than in the baseline projection, the growth foregone is less but the prevalence of the disease is higher.

The second column of the same table shows the results obtained by changing the communicability between promiscuous and non-promiscuous population: if the latter is less prone to enter in contact with the former, the results are virtually identical to those of the baseline model. Although larger differences occur if the horizon of the projection is increased, this dimension appears to be of relatively low importance.

The third column reveals an interesting feature of the projection which partially confirms the conjecture made before. This column displays the results that would obtain if the disease could not be transmitted via injections or transfusions: at the end of the 50th year of projection the combined incidence of AIDS and infection are virtually zero, their combined prevalence has virtually vanished, the losses in life expectancy are no longer visible and the growth foregone is negligible.

A similar conclusion is obtained if one compares columns four through seven corresponding to situations that we have considered

critical. If the scenarios underlying the result displayed in columns four through six were to take place, the population would become extinct in less than 100 years. However, if the same parameter values are maintained but the disease is NOT allowed to be transmitted through injections or transfusions, the impact although quite visible is much less dramatic. In fact, the population continues to grow at a positive rate of growth even though by the end of the 50th year it has foregone a growth equivalent to 17 percent of its potential growth. Life expectancy has decreased to about 38 years and the prevalence of infection and AIDS combined is almost 13 times as high as it was initially. However, if the projection is continued, the population eventually recovers its original profile and the disease vanishes completely. The contrasts in prevalence are apparent in Figure 4 where we plotin five year intervals the proportions of the population which is symptomatic and asymptomatic for the projections associated with columns 4, 6 and 7 of Table 5 (Series A, B, and C in the graph).

The mechanisms of sexual transmission that we have assumed coupled with the high lethality of AIDS appear to arrest the spread of the disease in a relatively short period of time. The impact on the population is more severe when transmissions through injections affect the population at all ages, when the latency times are longer and when the lethality of AIDS is diminished. In

particular, the impact of the disease could be considerable magnified if survival to reproductive ages were possible for those who are infected at early ages. Protection through condoms has an important effect regardless of latency but is not nearly as important as the removal of the risks associated with injections and transfusions.

CONCLUSIONS.

The results of our two alternative models lead to somewhat different conclusions. First, reasonable assumptions about the mechanisms of transmission of the disease reveal that a linear growth of incidence with a rate of 3 per thousand per year (as assumed in the first model) may be too conservative. During the first 50 years in a variety of scenarios, incidence and prevalence tend to grow exponentially with doubling times of less than 30 years. Second, with the exception of highly critical scenarios, the impact on population growth and growth foregone is contained in a narrow band. By and large, the impact of the disease leads to growth foregone of less than 25 percent over a 50 year period. Thus, if one is willing to neglect the critical scenarios, the conclusions reached with the first model are similar to those that are consistent with the second model. Third, the more complex model partially confirms the conjecture that the sexual mechanism of transmission coupled with median latency close to 8 years and

high lethality of AIDS lead to a slow-down of the diffusion of the epidemic within a relatively short period of time (less than 50 and certainly less than 100 years). This holds regardless of levels of usage of protection and sizes of and communicability between the various subpopulations. This conclusion should also hold in a more complex model if the impact of sexual transmission were additionally muffled by constraints imposed by relatively stable unions.

Although our projection exercises do not shed new light on the issue, we suspect that the impact of AIDS on population growth will be much less than its impact on customs and institutions that are typical in Sub-Saharan Africa. In fact, the epidemic has the potential of altering sexual behaviors and marriage patterns, of shaking the regional population distribution and of redirecting public investments away from primary health toward crisis management. The health policies advocated by the World Health Organization have placed priorities on child care and insisted on such measures as vaccinations, oral rehydration, and family planning. Inmunization programs may suffer a severe blow because of their association with one of the main means of transmission of HIV, namely, infected needles. Family planning programs may benefit insofar as they can be a means to diffuse the use of condoms as a protection against sexually transmitted HIV. However, they may also be negatively affected from being starved of funds

redirected toward more pressing health needs. The costs of fighting the epidemic and of caring for those who get the disease may deplete the health budgets of African countries and lead to a halt in mortality reductions which would have otherwise occurred.

Future modelling work should proceed in three directions. First, testing the plausibility of some of the most crucial parameters that we were able to identify: those associated with transmission through injections and transfusion, and those controlling latency times. It is clear from our projections that lacking good estimates of the probabilities of transmission through injections and transfusion and of their frequency by age may lead to wildly different results. Similarly, robust estimates of latency times are necessary to correctly identify the overall yearly rate of increase of the infectious population. Second, regional distribution ought to be incorporated in order to follow simultaneously the spatial and temporal spread of the epidemic. Different regions should be considered not only because they may be exposed to sharply different mechanisms of transmission but also, and more importantly, because the impact of the disease in each of them may be much more significant than when the phenomenon is studied in the society as a whole. Also, the contact between regions either through transient displacements or through permanent migration could fuel (or arrest) the overall diffusion of the disease. Third, incorporation of the dynamic in special

groups, those likely to be most affected by the disease, can serve to target policies and hence to employ efficiently limited resources.

Both the incorporation of regions and of special groups can be done with relative ease, by generalizing in simple ways the second model. Although these generalizations will certainly improve the practical utility of the model, it is highly unlikely that they will alter in any important way the broad conclusions that we have reached with its more simplified version.

REFERENCES

Bulatao, R. 1987. 'Initial investigation of the demographic impact of AIDS in one African country.' Draft, World Bank.

Brunet, J.B. and R.A. Ancelle, 1985. ' The International occurrence of the Acquired Immunodeficiency Syndrome.' Annals of Internal Medicine, 103670-674

Darrow, W.W., D.F. Echenberg, H.W. Jaffe, et al., 1987. 'Risk factors for human Immunodeficiency Virus (HIV) Infections in homosexual men.' American Journal of Public Health, 77(4): 479-483

Fortin, A. J., 1987. ' The politics of AIDS in Kenya. ' Third World Quarterly 9, No 3 : 906-919

May, R.M. and R. Anderson, 1987. ' Transmission dynamics of HIV infection.' Nature 326: 137-142

Nunn, P. 1987. 'Aids and Africa. ' IPPF Medical Bulletin 21, No 1: 4-6

Palloni, A. 1987. 'Assessing the levels and impact of mortaliy in crisis situations.' Working Series Paper No 87-36,Center for Demography and Ecology, University of Wisconsin.

Piot, P. and M. Carael, 1988. 'Epidemiological and sociological aspects of HIV-Infection in developing countries.' British Medical Bulletin, 44(1) (forthcoming).

Population Reports, 1986. AIDS- A public health crisis. The John Hopkins University, Series L, No 6.

Quinn, T.C., J.M. Mann, J.W. Curran, and P.Piot, 1986. ' AIDS in Africa: an epidemiological paradigm.' Science, 234: 955-963

United Nations, 1986. World Population Prospects. Estimates and projections as assessed in 1984. Department of International Economic and Social Affairs, Population Studies No 98. New York.

Watkins, S. and J.Menken, 1985. 'Famines in historical perspectives.' Population and Development Review, 11: 647-676.

TABLE 1: Age Distribution of HIV Infection.

AGE	MALES	FEMALES
0-4	.005	.005
5-9	.005	.005
10-14	.005	.010
15-19	.015	.030
20-24	.100	.225
25-29	.210	.330
30-34	.230	.220
35-39	.220	.100
40-44	.100	.050
45-49	.050	.010
50-54	.020	.005
55-59	.020	.005
60-64	.010	.002
65-69	.005	.001
70-74	.003	.001
75-79	.002	.001

TABLE 2

Projections under various assumptions concerning mortality and AIDS.

	(1) U.N. Assumptions	(2) Stagnation of mortality	(3) U.N. + 1% HIV	(4) U.N. + 2% HIV	(5) Stagnation + 1% HIV
1985					
Population	100,000	100,000	100,000	100,000	100,000
Tot Fert*	6.82	6.82	6.82	6.82	6.82
Fem. $_0e_0$*	48.9	48.9	48.9	48.9	48.9
CBR	50.1	50.1	49.9	49.8	49.9
CDR	20.9	20.9	21.1	21.3	21.1
r	2.92 %	2.92 %	2.88 %	2.85 %	2.88 %
HIV incid.	0 %	0 %	0.17 %	0.33 %	0.17 %
HIV preval.	0 %	0 %	0.4 %	0.8 %	0.4 %
AIDS DR	0	0	0.3	0.6	0.3
2010					
Population	215,216	196,479	200,043	185,572	183,019
Tot Fert+	5.58	5.58	5.58	5.58	5.58
Fem. $_0e_0$+	59.1	48.9	59.1	59.1	48.9
CBR	42.3	43.7	41.8	41.2	43.1
CDR	12.7	19.6	17.7	22.9	24.4
r	2.96 %	2.41 %	2.41 %	1.83 %	1.87 %
HIV incid.	0 %	0 %	1 %	2 %	1 %
HIV preval.	0 %	0 %	7.66 %	15.82 %	7.91 %
AIDS DR	0	0	5.6	11.6	5.8

Notes: $_0e_0$ does not include AIDS deaths.
* 1980-85
+ 2005-10

Table 3a: Characteristics of the Initial Populations.

Stable Population, Model North of Coale-Demeny Models(a):

Female Life expectancy= 50.162
Male Life Expectancy= 46.986
Rate of Natural Increase (both sexes combined) = .0297
Crude Birth Rate (both sexes combined)= .0461
Crude Death Rate (both sexes combined)= .0164
Total Fertility Rate = 6.0
Mean Age at Childbearing= 27.00

(a) To facilitate the projections, the life tables and the age distributions of the corresponding models were calculated by single years of age, from age 0 to age 120. As a result there are minor discrepancies between the initial parameters and those of the reference stable populations.

TABLE 3b: Parameters of the Baseline Projection.

a) Shape of the function for latency times (σ)= .07
b)'Level' of the function for latency times (κ)= 10 (md= 7.5years)
c) Shape of the function for AIDS mortality = .07
d)'Level' of the function for AIDS mortality =
    d.1) for 10<age<70 3.5 (md= 2-3 years)
    d.2) for age<11 and age >69 .25 (md= .8 to 1.0 year)
e) Proportionate excess of mortality infectious (α) =.10
f) Proportion using condom =.50
g) Effectiveness of condom =.80
h) tmf= .0026, probability of transmission from male to female
i) tfm= .0016, probability of transmission from female to male
j) probability of transmission with injection/transfusion= .03
k) j=4, average number of injections/transfusions.
l) probability of infected mother infecting newborn=.70
m) total proportion promiscuous=.5
n) number of partners per year promiscuous=20
o) number of partners per year non-promiscuous=3
p) number of sex acts per partner per year, promiscuous=3
q) number of sex acts per partner per year, non-promiscuous=10
r) endogeneity of promiscuous=.80
s) endogeneity of non-promiscuous=.20
t) proportion of unsterilized needles=.80

TABLE 4: Selected Parameters after 50 years of Projection. (1),(2)

	Projection						
Parameter	1	2	3	4	5	6	7
r	26.7	26.0	27.7	24.6	22.3	26.4	28.7
e	45.9	43.2	47.8	42.9	39.7	45.7	49.2
i(i)	3.9	6.9	1.9	8.7	13.9	4.8	.5
i(a)	2.9	4.9	1.6	6.0	8.2	3.3	.6
p(i)	32.8	55.4	17.8	77.7	119.0	45.8	4.1
p(a)	7.8	13.1	4.3	13.3	20.5	7.8	1.8
rr	27.8	27.2	28.1	27.3	26.5	27.9	28.6
Var	7.3	9.3	6.3	9.0	11.7	7.0	4.7

(1) The definition of symbols is as follows:
r is natural rate of increase; e is female life expectancy;
i(i) is number of new infectious cases per susceptible case;
i(a) is number of new AIDS cases per susceptible case;
p(i) is prevalence of asymptomatic infectious;
p(a) is prevalence of AIDS
rr is the observed rate of growtth from year 0 to year 50
Var is the growth foregone or the relative difference between .030 and rr.
All quantities are expressed in per thousand population except the last row which is expressed as a percent.

(2) Column 1 is for the baseline projection. Columns 2 and 3 change are for projections where the usage of condom to .10 and .90. Column 4 is as for thethe baseline but with longer latency times implying a median of about 12.5 years. Columns 5 and 6 are for projections as in Column 4 but with condom usage changed to .10 and .90. Column 7 is for a baseline projection with latency times with a median of about 3 years.

Table 5: Selected Parameters after 50 years of Projection.(1),(2)

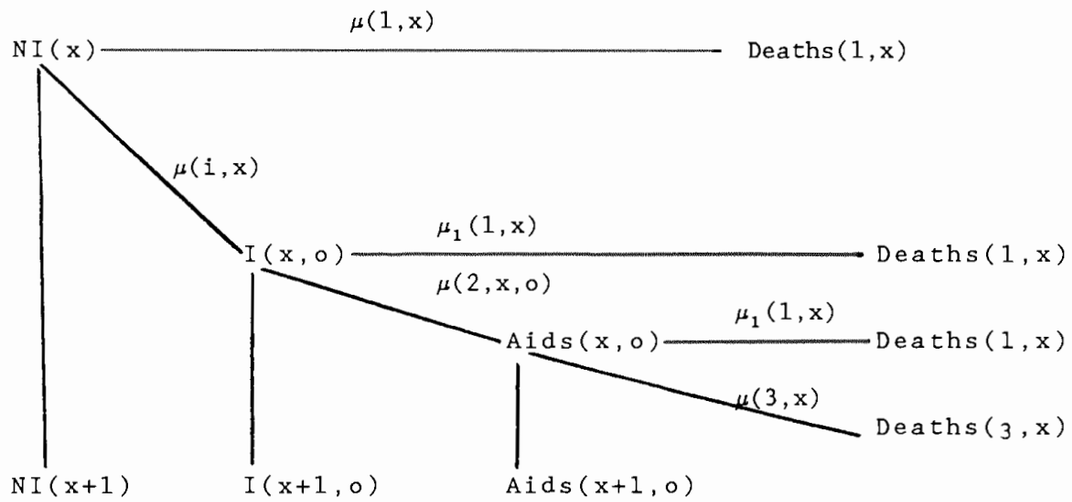
Parameter	Projection						
	1	2	3	4	5	6	7
r	26.9	26.6	29.3	-45.3	-34.6	-45.4	21.2
e	46.7	45.8	50.1	10.8	12.9	10.9	38.3
i(i)	3.8	4.0	.0	37.4	42.4	36.8	12.5
i(a)	3.0	3.0	.0	76.6	65.3	76.5	9.3
p(i)	32.5	33.7	.3	697.8	740.3	696.6	102.7
p(a)	17.0	8.0	.1	231.2	195.0	234.1	24.7
rr	28.0	27.8	29.2	-11.5	-7.9	-14.1	25.3
var	6.7	7.3	2.7	138.3	126.3	113.7	15.7

(1) See footnotes for Table 4

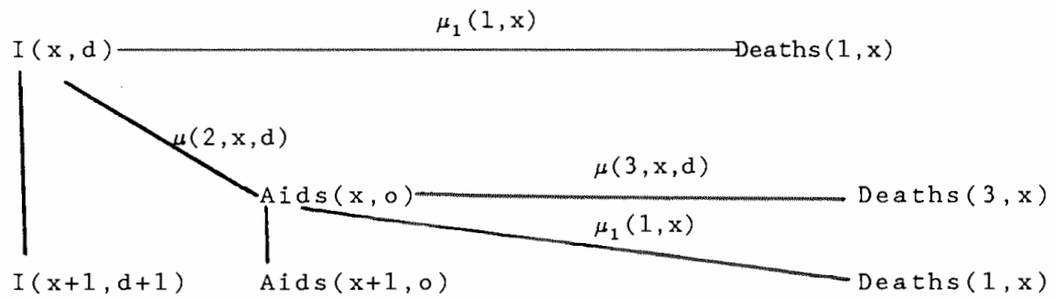
(2) Column 1 displays the result for a projection like the baseline but with a force of mortality due to AIDS implying a median survival time of about 3 to 4 years for the very young and old and of about 7 to 8 years for those in the prime ages. Column 2 displays the results of a baseline projection with high endogeneity among both promiscuous and non promiscuous (β and η are set equal to .80). Column 3 shows results of a baseline model where there is no transmission due to injections and transfusions. Column 4 displays results for a baseline model with the probability of transmission due to injections and transfusions increased to .10. Columns 5 and 6 imply .10 condom usage, .80 promiscuous and .10 as probability of transmission due to injections and transfusions. The results of column 5 depend on longer latency times than those in column 6 (median latency times of between 12 and 13 against 7 to 8). Finally, the results of column 7 depend on the same parameters as those in Column 6 but with the probability of transmission through injections set equal to .03

FIGURE 1: TRANSITIONS BETWEEN STATES.

a) Transitions for non-infectious.



b) Transitions for infectious of duration d>o.



c) Transitions for AIDS cases.

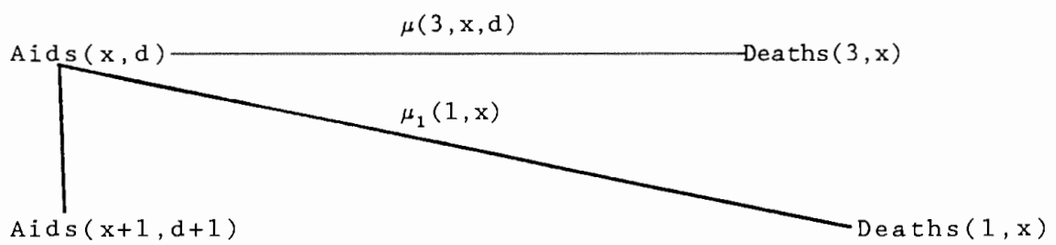
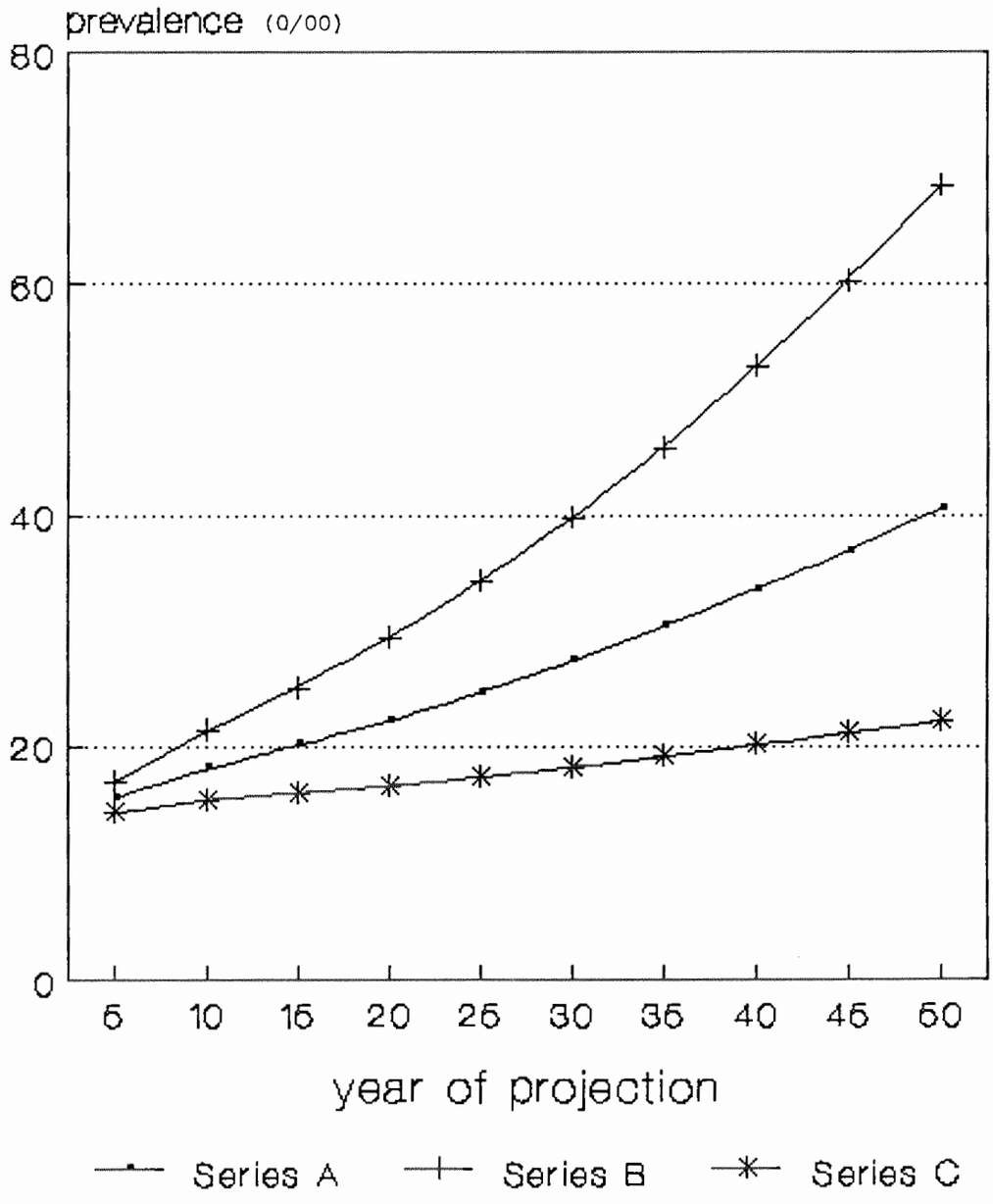
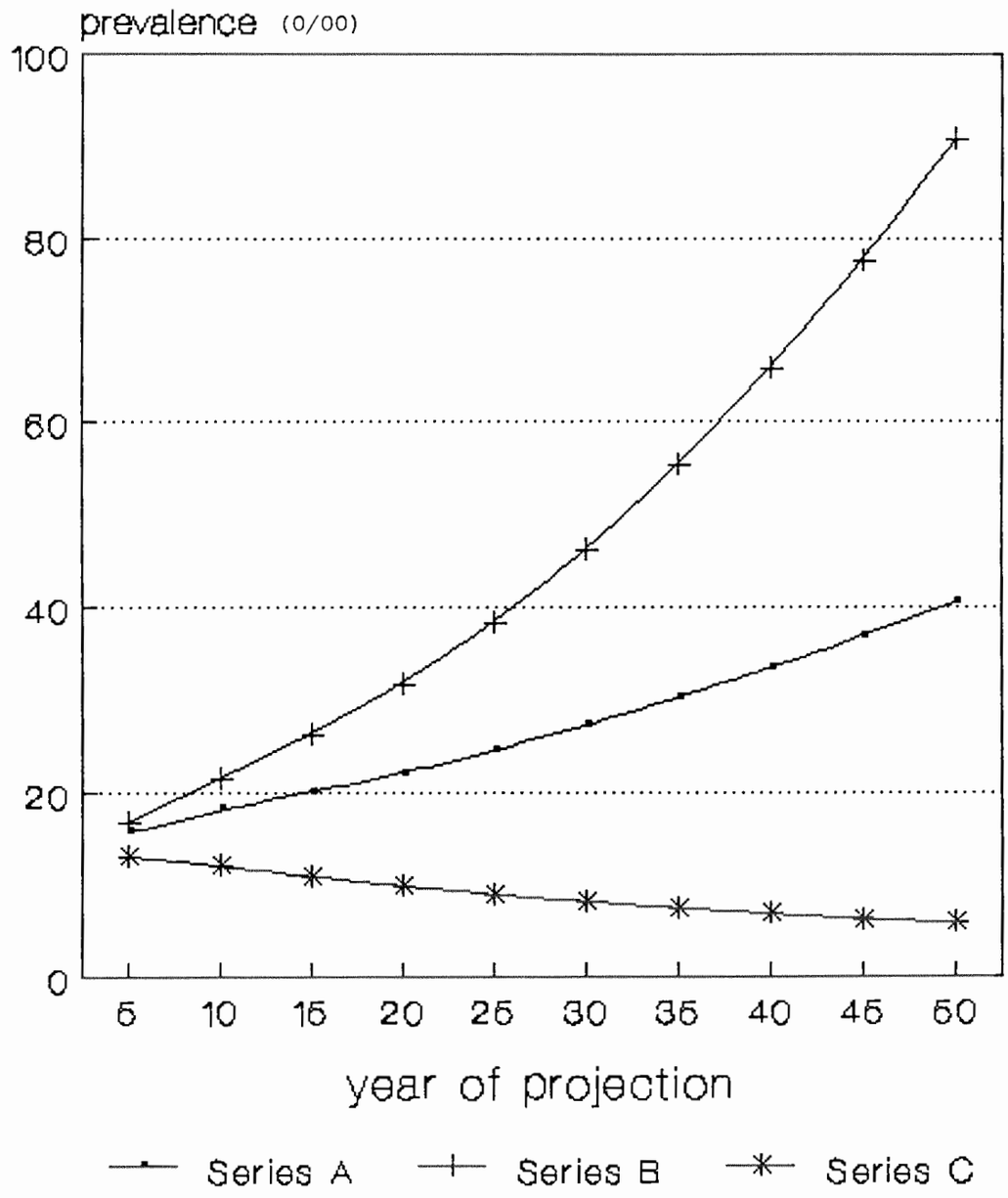


Figure 2
Prevalence of infection and aids



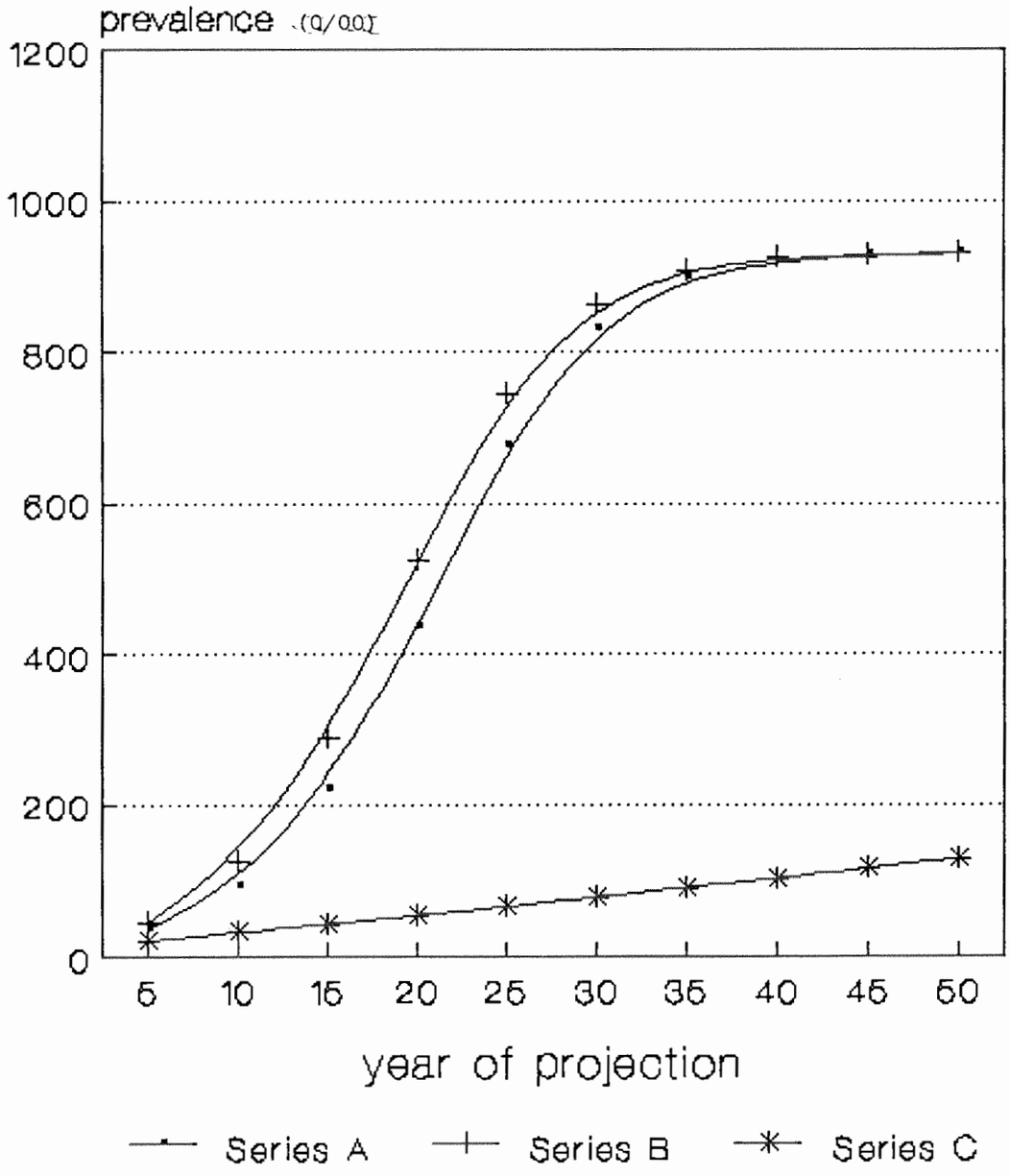
See columns 1,2,3 in Table 4

Figure 3
Prevalence of infection and aids



See columns 1, 4, 7 in Table 4

Figure 4
Prevalence of infection and aids



See columns 4,6 and 7 in Table 5