Prevention and Medication of HIV/AIDS -

The Case of Botswana

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Abstract

In this paper we developed a mathematical model which allows estimating and projecting the effects of prevention and treatment programs on the total population size, HIV-induced deaths, and life expectancies. Considering only the female population we project the changes of the demographic developments and the situation of HIV/AIDS for Botswanan up to 2060. Our mathematical model is used to project the female population development considering their age-structure. Treatment programs are included through selecting a price for medication (or giving it for free). Prevention programs consist of two parts: school-based programs which try to change risky behavior and instantaneous prevention (e.g. free condoms) which has only a short-time effect on the infection risk. The main conclusions drawn from our results are that prevention-only programs always yield the fastest decrease in HIV/AIDS prevalence. Adding a medication program reduces the efficiency of the prevention interventions regarding prevalence, but it reduces the number of HIV-induced deaths and increases life expectancies.

Keywords: HIV/AIDS, mathematical model, age distribution, Botswana, prevention and treatment.

1 Introduction

On World AIDS day 2003, the World Health Organization announced the details of its 3 by 5 plan, whereby 3 million people in developing countries would receive antiretroviral medications by 2005. This is a massive increase in number of people in those countries currently receiving such treatment. The scale of the WHO plan raises the question of the appropriate mix of anti-HIV policies. We know that Uganda and Thailand have been able to reduce their HIV prevalence rates significantly without the use of much medication. What we do not know is the likely contribution to social welfare that can be expected from a much more expanded use of antiretrovirals. In *Epidemics*, Hippocrates, who is regarded as the father of modern medicine, wrote: "As for diseases, make a habit of two things to help or at least to do no harm." This is a good dictum for contemporary health care policy-makers, as well. In this paper, we seek policy mixes that help or least do no harm.

Today Botswana is alone in Sub-Saharan Africa in implementing a program of providing anti-HIV medication free to those who need it. South Africa will shortly follow in its foot-steps. In this paper, we construct a model of the dynamics of the HIV epidemic and param-eterize it to make it roughly consistent with the Botswana experience. We include the costs and benefits of three types of interventions: (1) school-based education programs, which permanently increase the proportion of the population practicing safe sex; (2) mass media and mass education programs that influence the riskiness of sexual behavior during the time the programs are implemented, and (3) programs of antiretroviral use.

In Section 2, we present the structure of the model. Section 3 discusses the parameterization of the model and the final sections provide the results and conclusions.

2 The Model

The model that we use here belongs to the class of age-structured epidemic models based on Hoppensteadt (1977) and Castillo-Chavez (1989) and is a modification of Sanderson (2004), which is already calibrated to Botswana data and it predicted aggregate population growth in Botswana from 1993 to 2001 almost exactly. Sanderson describes a one-sex model which includes only the female population. There are no HIV prevalence data in Botswana for males and including them with parameters that were impossible to measure would have brought it too far from reality for our tastes. In our modified model $t \in [0, T]$ is the time and $a \in [0, \omega]$ is the age of an individual, where T is the time horizon, and ω is the maximal length of life. Furthermore it is assumed that age $a = 15$ corresponds to the beginning of sexual activity. For simplicity we assume that there are no infected newborns. In fact few HIV-positive newborns survive to the age of sexual activity.

The group $N(t, a)$ denotes the non-susceptible individuals, who practice only safe sex, live in monogamous relationships, or who are naturally immune (which is the case for about 10 per cent of the population). $S(t, a)$ represents the susceptible group. The transition from S to N presumably happens about the beginning of sexual activity, which is indicated by the function $\kappa(a)$ which has a support around this age. The non-susceptible group has only a

minor influence on the other groups in that sense that if they have sexual contact (in this case only indirect, which means via a male) with an individual from the susceptible (or infected) group they do practice safe sex and therefore the susceptible has also a safe contact instead of the usual unsafe contacts. The susceptibles can get infected and move than into the infected group $I(t,a)$. This group represents the HIV positive females who are asymptomatic. Most of them do not even know that they carry the virus. Once they get symptoms they move into the $A(t,a)$ group of persons who have developed AIDS. A fraction of this group can afford the medication (which depends on the price of the medication) and has therefore a reduced death rate (cf. Figure 1).

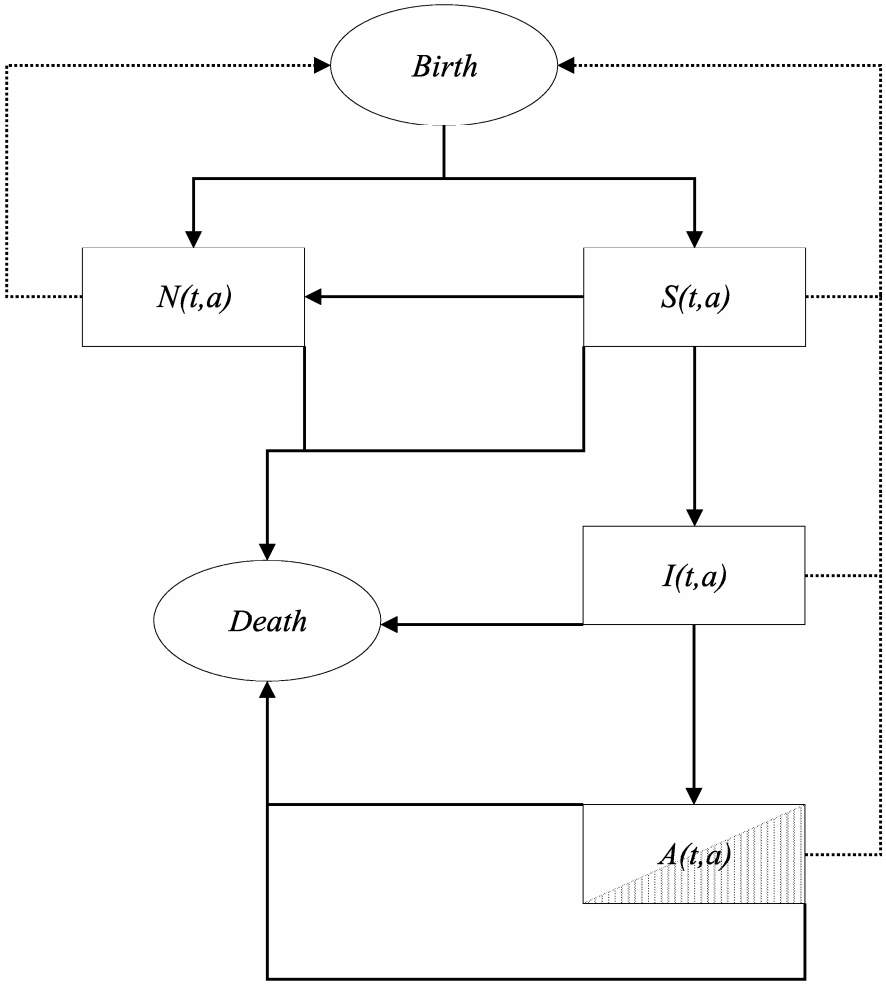

Figure 1: Scheme of the different population groups and the flow between these groups. Starting from Birth individuals move either to the susceptible group $S(t,a)$ or the non-susceptible group $N(t,a)$ (due to immunity). If susceptibles are infected with HIV they move into the group $I(t,a)$ and when they develop AIDS, they move on to the group $A(t,a)$. This group splits up in two parts: Those who can afford medication and therefore have a reduced death rate (dashed area) and those who get no treatment.

The dynamics of the model can be described by the equations below for $t \in [0,T]$, $a \in [0,\omega]$ (For a better readability the arguments (t,a) are omitted for the functions $N(t,a)$, $S(t,a)$, $I(t,a)$, $A(t,a)$).

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a}\right)N = -\mu_d(a)N + \Psi(v(t))\kappa(a)S \tag{1}$$

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a}\right)S = -\mu_d(a)S - \mu_{SI}(t,a,u(t,a))S - \Psi(v(t))\kappa(a)S \tag{2}$$

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a}\right)I = -\mu_d(a)I - \mu_{IA}I + \mu_{SI}(t,a,u(t,a))S \tag{3}$$

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a}\right)A = -\mu_d(a)A - \mu_{dA}(1-\delta(w(t)))A - \mu_{dM}\delta(w(t))A + \mu_{IA}I \tag{4}$$

with initial and boundary conditions

$$N(0,a) = N_0(a), \qquad N(t,0) = \alpha\big(B(t) + C_I(t) + C_M(t)\big) \tag{5}$$

$$S(0,a) = S_0(a), \qquad S(t,0) = (1-\alpha)\big(B(t) + C_I(t) + C_M(t)\big) \tag{6}$$

$$I(0,a) = I_0(a), \qquad I(t,0) = 0 \tag{7}$$

$$A(0,a) = A_0(a), \qquad A(t,0) = 0 \tag{8}$$

where

$$B(t) = \int_0^{\omega} \Theta(u(t,a))\varphi(t,a)(N+S)\mathrm{d}a \tag{9}$$

$$C_I(t) = \int_0^{\omega} \Theta(u(t,a))\varphi_I(t,a)I\mathrm{d}a \tag{10}$$

$$C_M(t) = \int_0^{\omega} \Theta(u(t,a))\varphi_M(t,a)\delta A\mathrm{d}a \tag{11}$$

Here $\mu_{SI}(t,a)$ denotes the transition rate from susceptible to infected, i.e., the incidence rate. It depends on the expenditures for instantaneous prevention (e.g. free condoms) $u(t,a)$. μ_{IA} denotes the rate at which infected people develop AIDS. We distinguish between three different death rates: $\mu_d(a)$ is the natural death rate (all deaths excluding AIDS), μ_{dA} is the death rate due to AIDS without medication and μ_{dM} is the death rate due to AIDS while under treatment.

Furthermore, $\delta = \delta(w(t))$ is the fraction of people having AIDS and being under treatment, which depends on the medication costs $w(t)$. The control w may be supposed bounded: $w(t) \in [0, c^*]$, where c^* is the non-subsidized price. However, the upper bound can be disregarded.

The fertility rates for the non-infected, infected, and medicated groups are $\varphi(t,a)$, $\varphi_I(t,a)$, and $\varphi_M(t,a)$ respectively. The symptomatic non-medicated individuals are assumed non-

fertile.[1] This fertility rates are multiplied by a function $\Theta(u(t,a))$ which represents the effect of prevention programs on the total population. If we assume a prevention program like giving free condoms, this has major effects on the fertility rate. Since some of the newborns are naturally immune, they start directly in the N-group, the others are susceptible. With α we denote the fraction of naturally immune newborns.

The control $v(t)$ represents the per capita money spent for long term transitions from S to N. Presumably the multiplier $\kappa(a)$ is the characteristic function of the interval $[12,17]$, but it can be a smooth function with support about the above interval.

The incidence rate $\mu_{SI}(t,a)$ depends on the prevalence. In our case we use a normalized prevalence of the form (the time parameter t is omitted for readability)

$$P(t,a) = \int_0^{\omega} \rho(a,a') \frac{I(a') + \eta_A(1-\delta)A(a') + \eta_m \delta A(a')}{S(a') + \eta_N N(a') + I(a') + \eta_A(1-\delta)A(a') + \eta_m \delta A(a')} \mathrm{d}a' \qquad (12)$$

where $\rho(a,a')$ represents the relative number of potential risky indirect[2] contacts between an a-year-old and an a'-year-old woman. η_N, η_A, η_M are factors which denote the sexual activity and infectiousness of the different groups. Now the incidence rate can be writ-

[1] The fertility rates of the HIV positives $\varphi_I(t,a)$ and $\varphi_M(t,a)$ include only the healthy newborns.

[2] In our case we assume that the usual way of infection is via heterosexual contacts. Since this is a one-sex model, we do not have direct contacts but there is always a man linking two women and transferring the virus from one woman to another.

ten as a product of the age-specific level of risk $\gamma(a)$, and a control factor $\Phi(u(t,a))$ which represents the fraction of safer sex contacts due to the prevention effort $u(t,a)$:

$$\mu_{SI}(t,a) = \gamma(a)\Phi(u(t,a))P(t,a) \tag{13}$$

Remark. The above model is implicitly based on several simplifying assumptions such as single sex, empirical (not strictly balanced) mixing, homogeneity within an age group, etc. In particular, in Castillo-Chavez et al. (1989) and Thieme and Castillo-Chavez (1993) it is argued that the duration since infection could be an important factor in the dynamics, since both the infectivity and the rate of developing full-blown AIDS strongly depend on this duration. The duration since infection is explicitly taken into account in a model developed in Feichtinger, Tsachev, Veliov (2004) which, however, is not calibrated with real data. The results obtained for Botswana in Section 5 of the present paper are qualitatively consistent with those in Feichtinger, Tsachev, Veliov (2004).

3 Parameters

Since there are not all data available for 2005 we start with a simulation of the HIV epidemic between 1993 and 2005. So we are able to check if the model is consistent with the real data, and we are able to generate the missing parameters for 2005. In the following part we will discuss each parameter necessary to complete the model.

For the fertility rate $\varphi(t,a)$ we are using data from the year 1993 available on *www.census.gov* and assume an exponential decrease to 2.85 children per woman in the year 2005. For the simulations between 2005 and 2060 we use data from 2005 and assume an exponential decrease to 2 children per woman in 2060. Since we do not consider HIV-positive newborns in our model, we assume that the fertility rates for healthy newborns for the I -group and the medicated people are 80% of the fertility rates of the healthy groups. Hence, it is necessary to adapt the original fertility rate by increasing it by 15%, so that this reduction is compensated. The fraction of non-susceptible births α is assumed to be 0.1, i.e. 10% of the newborns are non-susceptibles.

The data for the mortality rate $\mu_d(a)$ is derived from the Coale-Demeny model. For the initialization phase (1993-2005) we use fixed estimates based for 1993, and for the further simulations we use estimates for 2005. We assume that the mortality rates due to AIDS (without treatment), $\mu_{dA}(a)$, and due to AIDS under treatment, $\mu_{dM}(a)$, are independent of the age. Furthermore we assume that each year 50% of the people having AIDS die. This gives a death rate of $\mu_{dA} = 0.693$, while only 10% of the people under treatment die due to AIDS per year. Hence, their death rate is $\mu_{dM} = 0.105$. This is a very optimistic estimation. Due to Over (2004) antiretroviral therapy may add between 2.5 and 10 years to the lives of the people.

There are three parameters which determine the incidence rate:

1. the infection rate $\gamma(a)$,

2. the inter-age-contacts $\rho(a,a')$, and

3. the factors for the relative sexual activity η_N, η_A, η_M of the different groups.

For the infection rate $\gamma(a)$ we use the same estimates as in Sanderson (2004). The inter-age contact rates $\rho(a,a')$ is the fraction of (indirect) contacts of an a-years-old susceptible with an a'- year-old individual. The underlying idea is that a man has most times sexual contact with women who are of about the same age. Hence, a woman is more likely infected by a man who himself got the infection from a woman of similar age. Therefore the value of $\rho(a,a')$ is high if the difference $|a-a'|$ is small, and the value is low if the age difference is large. $\rho(a,a')$ is normalized to fulfil

$$\int_0^{\omega} \rho(a,a')\mathrm{d}a' = 1.$$

We used the following form for $a \geq 18$

$$\rho(a,a') = \begin{cases} 0.1163 & |a-a'| \leq 2 \\ 0.029\left(6-|a-a'|\right) & 2 < |a-a'| \leq 6 \\ 0.0009 & |a-a'| > 6 \end{cases}$$

and for $12 \leq a \leq 17$ the same function is used, but it is cut off at $a' = 12$ and normalized according to the above restriction. The constants η_N, η_A, η_M describe the reduced participation on the "sex market" of the groups N, A, and M as well as their different infec-

tiousness. In Over (2004) it is stated that antiretroviral therapy may reduce the infectiousness by a factor two to eight. In our simulation experiments we set this parameters to $\eta_N = 0.5$, $\eta_A = 0.5$, and $\eta_M = 0.1$, which means that we may overestimate the reduction of the infectiousness of the medicated group. Furthermore, we assume that non-medicated people suffering from AIDS reduce their number of sexual contacts because they are feeling sick. Medicated people are less infectious and we assume that they may change their sexual behavior. If this assumption is not true and medicated people do not behave in a responsible way and medication is not so effective regarding the infectiousness, we may also use values around 1 or even higher for η_M. We calculated also scenarios with a high infectivity of $\eta_M = 3$ of the medicated group.

Once a person is infected, the outbreak of AIDS depends mainly on the time since infection with HIV. The above model is not capable of tracking that time period. Therefore we calculate the rate in such a way that the mean duration between infection and the outbreak of AIDS is 8.5 years. Hence, $\mu_{IA}(a) = 2/17$ (cf. Over 2004).

The influence of prevention programs on the number of risky contacts is represented by $\Phi(u)$, where $u(t,a)$ is the per capita monetary expense for instantaneous prevention (e.g. free condoms). We assume an exponential decrease of risky contacts if the program is increased. Furthermore we assume that the program can reduce the risky contacts only up to 80%. Hence, we assume

$$\Phi(u) = (1 - 0.2)e^{-0.0098u} + 0.2$$

Such prevention programs like free condoms have not only an influence on the number of risky contacts, but also on the fertility rate, because it reduces the risk of unintended pregnancies. We use the same function as above, but the effect is reduced by one half.

$$\Theta(u) = \frac{\Phi(u) + 1}{2}$$

On the other hand school-based prevention programs are effective before or around the age of starting sexual activities. These type of programs determine the distribution of non-susceptible and susceptible women. We assume that 10% of the population are automatically in the non-susceptible group and 20% of the population are always in the susceptible group when reaching the age of sexual activity. In this case we use an arcus tangens function to model the effect of diminishing returns:

$$\Psi(v) = \frac{14}{9\pi} \arctan\left(\frac{\tan(0.45\pi)v}{100} \right)$$

The multiplier $\kappa(a)$ in (1) indicates the age range, when such a prevention program can be effective:

$$\kappa(a) = \begin{cases} 0.2 & 12 \le a \le 17 \\ 0 & \text{otherwise} \end{cases}$$

In order to measure the effect of price subsidies for medications, we use the function $\delta(w)$, which gives the fraction of the women who can afford the medication at a price of w

$$\delta(w) = 0.7e^{-0.0034w}$$

These means that 70% of the female population would get medication if it is for free. The usual market price of medication c^* is assumed to be \$500, which means that 13% could afford medication.

4 Initialization and Validation

For 2005 there are no detailed data available on the initial population structure, especially for HIV-positive and AIDS-suffering women. Hence, we start our simulations in 1993. For that year more data is available. We generate the initial data for the projections from 2005 to 2060. Before we can start this data simulation it is necessary to analyze the HIV-situation in Botswana between 1993 and 2005. In 1997 the government of Botswana started a school-based prevention program by providing special training for teachers. In 2002 a treatment program was launched to provide free antiretroviral therapy for special population groups. So we set the necessary parameters such that we consider a 10%-level of the school-based prevention between 1997 and 2005 and a 20%-level treatment program between 2002 and 2005.[1]

For the model validation we compare the measured natural growth rate provided by the U.S. Bureau of Census for the years 1994 to 2005 with the results of our model. Furthermore we compare the HIV prevalence in 2002 with data from surveys among pregnant women (see Figure 2). For the years 2001 to 2005 there is a nearly perfect fit of the natural

[1] cf. http://www.avert.org/aidsbotswana.htm (06/06/2005)

growth rate of the population. In the years before the model underestimates the growth rate, but this fact may be caused by some inaccuracy in the initial data. For the HIV prevalence the comparison is more problematic, since the data available is from relatively small samples of pregnant women. We compare the data with two factors: the total prevalence rate based on the total population, and the prevalence rate based only on the healthy or asymptomatic population, neglecting persons who are suffering from AIDS (independent if they are under treatment or not). It is not surprising that the prevalence rate of the 15-19 year-old women is far below the available data set. A significant part of the population of that age may be sexually inactive. So investigating only pregnant women will result in a clear overestimation of the real prevalence rate. For the other age-groups the model is within an acceptable range.

Based on this validation we may conclude that our model is accurate enough for doing future projections. Since we do not consider any migration in the model — Botswana has a high immigration rate of about 0.6% of the total population per year — it is not possible to predict the change of the total population size. Since migration depends on many different factors (economic situation, wars, epidemics, …) it is hardly possible to estimate migration in 10 or 20 years. Therefore our investigations are restricted to the natural change of the population due to birth and death. But in order to use a correct initial scenario, we adapt the outcome of the initialization phase 1993-2005 of the model in such a way that we meet the real population figures available for 2005.

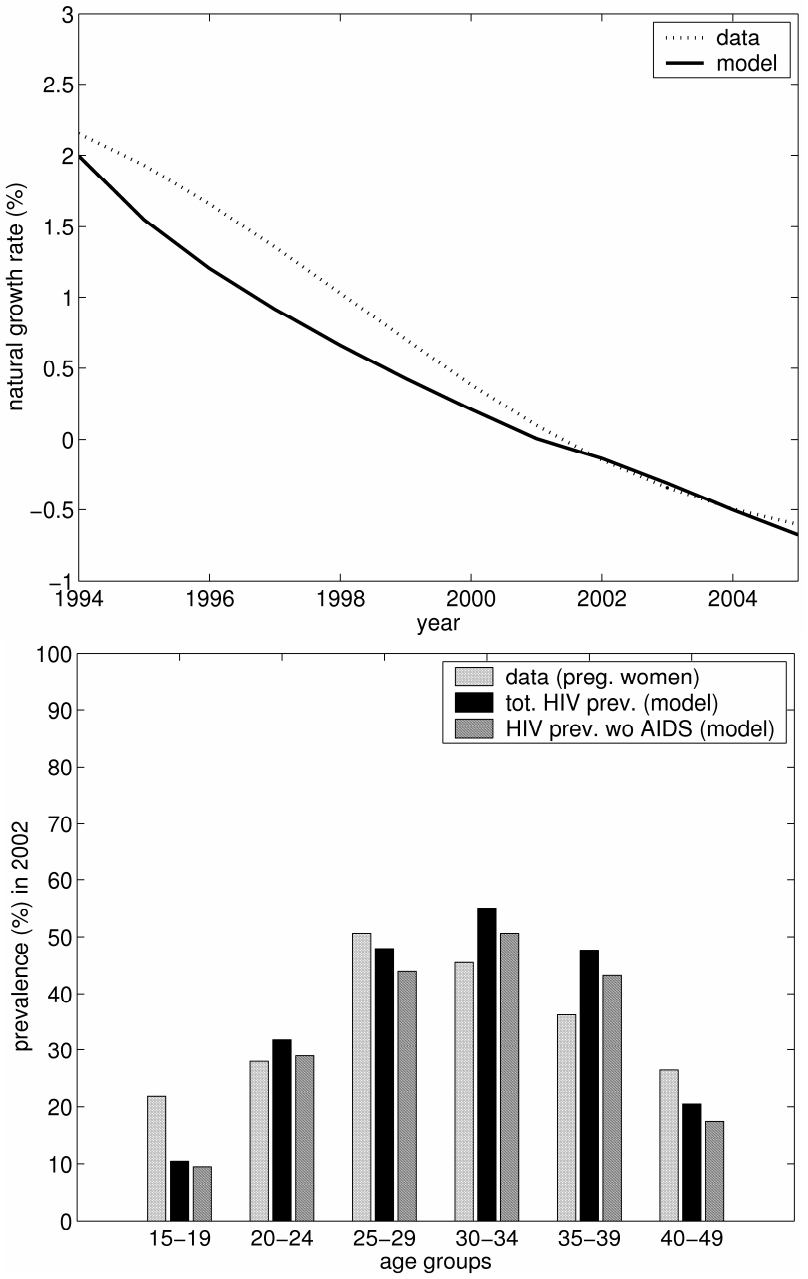

Figure 2: Comparison of data for the natural growth rate of the population and prevalence rates for pregnant women available from the U.S. Census Bureau (www.censos.gov) with the results of the model.

5 Results

Based on the results of the initialization phase we made several simulations with different intervention scenarios. If we freeze the prevention and treatment expenditures at the levels of 2005, the total population will decline from 1.6 millions in 2005 to about 0.65 millions in 2060 (without considering any migration) The total HIV prevalence will increase from 19% in 2005 to about 27% in 2020 and then will slightly decline to 22% in 2060. Between 2005 and 2030 2.7 million people will die due to AIDS and in the following 30 years another 2 millions will die. Life expectancy of the 15-years-old will decline from 40 years in 2005 to 36 in 2020 and then will slightly increase to 38 in 2060. (The reason for taking the life expectancy of the 15-years-old is that we do not consider HIV-positive newborns and we are interested in the effect of sexually transmitted HIV. Therefore we consider the population at the beginning of sexual activity for measuring the life expectancy.) So without any further interventions the HIV prevalence will stabilize on a very high level and the population will shrink dramatically.

We investigated the following different scenarios:

p0/t0: Keep interventions on the level of 2005.

pF/t0: Here we apply maximal prevention (instantaneous and school-based prevention). It is assumed that the maximal instantaneous prevention reduces the infection risk by 70% and the maximal school-based prevention reaches about 70% of the population between 12 and 17 years.

pH/t0: Similar to the previous scenario, but we assume only half of the maximal possible prevention effort (35%).

p0/tF: In this scenario medication is extremely cheap such that 70% of the population can afford the medication if necessary.

p0/tH: In this case only 35% of the population can afford medication.

pH/tH, pF/tF, pH/tF, pF/tH: all possible combinations of the above scenarios.

Furthermore we distinguish between the cases if the medicated group has a low infectivity (iL) or a high infectivity (iH). For the scenarios, where the level of interventions is different from the current situation in 2005, we start from the current level and linearly increase the effort till we reach the given level in 2010.

Table 1 shows the projected values for total population size (Ptot), HIV/AIDS prevalence (Prev), life expectancy at age 15 (LE15), annual HIV-induced deaths (annHIV), and accumulated HIV-induced deaths (accHIV) for the years 2030 and 2060 assuming the different intervention scenarios. These results can be interpreted as follows:

1. Prevention-only scenarios allow to reduce the prevalence significantly and they can stop the population decline. With a full prevention program HIV/AIDS may extinct till 2060 and the life expectancy reaches its natural value. If only a medium level of prevention is applied, the situation improves considerably compared with p0/t0, but the annual number of deaths is still relatively high in year 2060.

2. Treatment-only scenarios (p0/tH and p0/tF) lead to considerably higher prevalence compared with p0/t0. In the low infectivity scenario (iL) the treatment slightly decreases the number of deaths and increases the life expectancy. However, in the high infectivity scenario (iH) both the full treatment and the half treatment programs give worse results in 2030 and in 2060, with respect to all indicators: population size, prevalence, number of deaths, life expectancy. Thus the benefit from the treatment-only programs is questionable; such programs might be even counterproductive.

3. If treatment is added to prevention (compare pF/t0 with pF/tF, for example), there would be a slight positive effect on the population size (in the low infectivity scenario), but the effect on the other indicators is not positive in the years 2030 and 2060.

If we take a more detailed look on the developments through time (cf. Figures 3 and 4) we see that the treatment has a positive effect on the population size and the life expectancy in the first 5-10 years. However, it leads to a high prevalence, which results in a decline of the population on the long run and to an endemic state of the disease. In contrast, the pure prevention program is superior on the long run and leads to extinction of the disease, while at the beginning the population size and the life expectancy is below the level for pure treatment. As seen from Figure 3, medication does not have positive effect on the life expectancy even when combined with full prevention, excepting the first 10 years.

Table 1: This table shows the projected numbers for 2030 and 2060 for different prevention-treatment scenarios with low and high infectivity of the medicated group. The best values are written in bold characters.

Scen.	Year	Ptot[1]	HIVPrev[1]	LE15[1]	annHIV[1]	accHIV[1]
p0/t0/iL	2030	1.20	25.6%	36.8	123,601	3.497
	2060	0.65	22.0%	38.0	58,354	6.113
pH/t0/iL	2030	1.48	9.6%	52.6	59,416	2.514
	2060	1.58	2.3%	65.6	14,903	3.435
pF/t0/iL	2030	1.61	**2.8%**	**63.1**	**20,534**	1.759
	2060	1.69	**0.1%**	**70.0**	**560**	1.928
p0/tH/iL	2030	1.22	26.1%	37.1	124,419	3.453
	2060	0.67	22.6%	38.2	60,007	6.112
p0/tF/iL	2030	1.31	28.3%	38.3	126,958	3.238
	2060	0.76	25.3%	39.4	67,148	6.071
pH/tH/iL	2030	1.49	9.9%	52.6	60,217	2.490
	2060	1.59	2.4%	65.6	15,120	3.425
pF/tH/iL	2030	1.61	2.9%	62.9	21,263	1.749
	2060	1.70	**0.1%**	**70.0**	583	**1.925**
pH/tF/iL	2030	1.54	11.3%	52.3	63,928	2.372
	2060	1.64	2.7%	65.6	16,201	3.374
pF/tF/iL	2030	**1.63**	3.6%	62.0	25,070	**1.697**
	2060	**1.71**	**0.1%**	**70.0**	722	1.999
p0/t0/iH	2030	1.16	26.8%	36.0	125,265	3.590
	2060	0.60	22.3%	37.2	54,765	6.148
pH/t0/iH	2030	1.44	10.9%	51.1	64,971	2,616
	2060	1.47	3.5%	63.4	20,844	3.714
pF/t0/iH	2030	**1.59**	**3.1%**	**62.7**	**22,043**	**1.803**
	2060	**1.68**	**0.1%**	**70.0**	**674**	**1.989**
p0/tH/iH	2030	1.15	28.1%	35.6	127,846	3.605
	2060	0.59	23.1%	36.9	54,086	6.177
p0/tF/iH	2030	1.19	32.6%	35.5	135,880	3.517
	2060	0.60	26.5%	36.9	56,620	6.235
pH/tH/iH	2030	1.43	12.3%	49.9	70,572	2.660
	2060	1.39	5.0%	60.9	27,285	3.941
pF/tH/iH	2030	**1.59**	3.4%	62.1	24,153	1.820
	2060	1.67	**0.1%**	69.9	839	2.031
pH/tF/iH	2030	1.41	17.6%	46.8	87,467	2.702
	2060	1.19	11.1%	53.8	45,05	4.515
pF/tF/iH	2030	1.58	5.2%	59.8	33,418	1.842
	2060	1.64	0.3%	69.6	2080	2.181

Ptot: total Population (millions)
HIVPrev: total HIV prevalence (including all HIV positive individuals)
LE15: total life expectancy of individuals at age 15
annHIV: annual HIV-induced deaths for the year 2030/2060
accHIV: total number of HIV-induced deaths from 2005 to 2030/2060 (millions)

Due to the uncertainty in some of the information needed for the model, we have performed sensitivity analysis with respect to some of the parameters. In particular scenarios with lower initial prevalence and with lower efficiency of the medication are investigated. The above observations remain true for these scenarios, although in both cases the effect of the treatment on the number of deaths and on the life expectancy tends to be slightly better (or less negative).

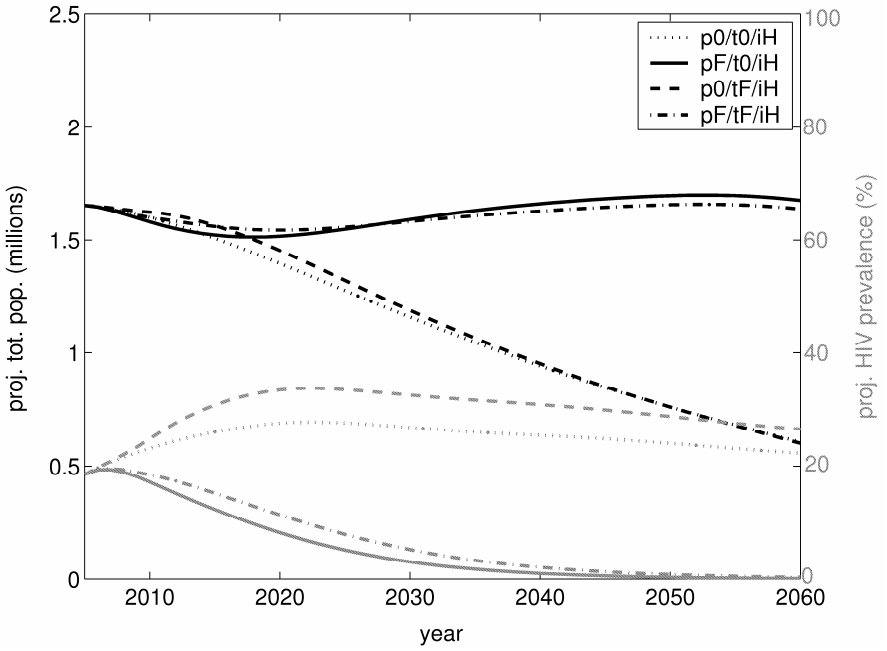

Figure 3: Projection of the total population size and HIV prevalence for Botswana for four different scenarios.

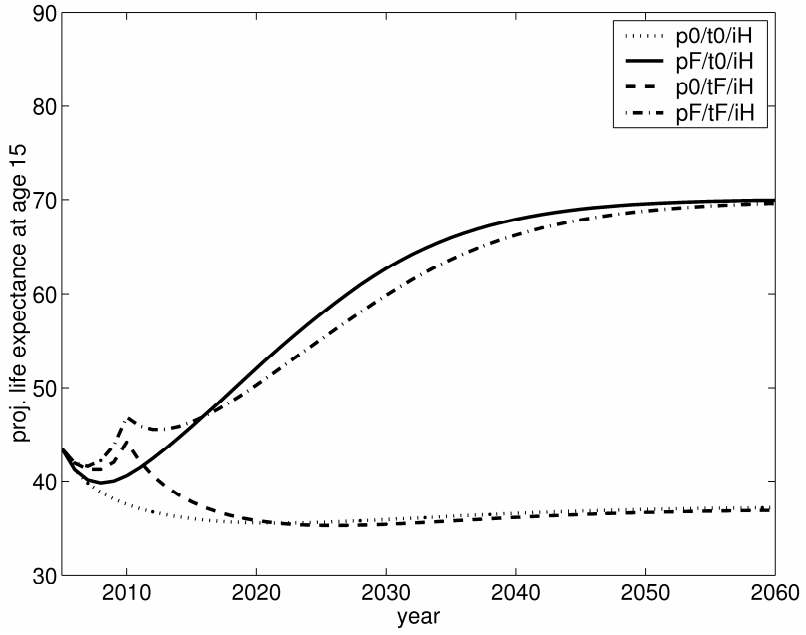

Figure 4: Projection of life expectancy at age 15 in Botswana for four different scenarios.

6 Conclusions

We show in this paper that the designs of programs aimed at ameliorating the effects of the HIV epidemic are important. High-tech programs of medication distribution may be a costly way to improve welfare. Indeed we show that, in the long-run, it could be worse than doing any type of prevention. Prevention programs interact with one another, changing the marginal benefits of additional expenditures. In developing countries with high rates of HIV prevalence, inefficiencies resulting from poorly designed health care interventions can have a high social cost.

The social cost of poorly designed anti-HIV programs is not just academic. Large sums of money can easily be spent on programs that reduce the welfare of people who are already suffering terribly. Models, such as the one presented here, can help sensitize policy-makers to the trade-offs that they face and help guide them to choices that, at least, do no harm.

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A Summary of parameters

$\varphi(t,a)$	fertility rate of N and S group; fertility rate from 2005, exponential decrease to 2 children per woman in 2006
$\varphi_I(t,a)$	[$= 0.8\varphi(t,a)$] fertility rate of I group
$\varphi_M(t,a)$	[$= 0.8\varphi(t,a)$] fertility rate of M group
α	[$= 0.1$] fraction of non-susceptible newborns
$\mu_d(a)$	non-HIV death rate; derived from a Coale-Demeny model
$\mu_{dA}(a)$	[$= 0.693$] mortality rate due to AIDS (non-medicated)
$\mu_{dM}(a)$	[$= 0.105$] mortality rate due to AIDS (medicated)
$\gamma(a)$	age-specific level of risk
μ_{IA}	[$= 2/17$] rate of developing AIDS
$\rho(a,a')$	inter-age contacts; depends mainly on the age difference $\lvert a-a' \rvert$
η_N	[$= 0.5$] reduced sexual activity of the N group
η_A	[$= 0.5$] reduced sexual activity of the A group
η_M	[$= 0.1$ or 3] reduced or increased sexual activity and infectivity of the M group
$\Phi(u)$	influence of prevention programs on the number of risky contacts
$\Theta(u)$	[$= 0.5(\Phi(u)+1)$] influence of prevention programs on the fertility rate
$\Psi(v)$	influence of school-based prevention program
$\kappa(a)$	effective ages for the school-based prevention program
$\delta(w)$	fraction of medicated women depending on the price of medication w