Prevention and Medication of HIV/AIDS - The Case of Botswana

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Abstract

Governments in developing countries are spending more and more money on antiretroviral medication programs in hope that the programs curb the spread of HIV/AIDS and reduce the suffering of those infected. This makes the question of determining the optimal mix of prevention and medication ever more pressing. We construct and parameterize a model of the spread of HIV/AIDS in Botswana that allows us to compute the balance of costs and benefits associated with various policy mixes. Today Botswana is the only country in the world that is freely providing antiretroviral medication to all those who need it. We conclude that medication-only programs can not avoid a population decline and in some cases it decrease social welfare even relative to doing nothing at all. Prevention-only programs always yield the highest welfare levels. School based programs and mass media type education programs are complementary. Adding a medication program to a combination of the two reduces the efficiency of the prevention interventions. Uganda has significantly lowered its HIV/AIDS prevalence without the use of medication. Our findings suggest that a policy-mix more focused on Uganda-type education campaigns and less on medication would be better for Botswana.

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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 footsteps. In this paper, we construct a model of the dynamics of the HIV epidemic and parameterize 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 model and the social welfare function that we use. Section 3 discusses the parameterization of the model and the final section provides the results.

2 The Model

The model that we use here belongs to the class of age-structured epidemic models [Hop77, CC89] and is a modification of [San04], which is already calibrated to Botswana data and it predicted aggregate population growth in Botswana from 1993 to 2001 almost exactly. In [San04] a one-sex model is described 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.

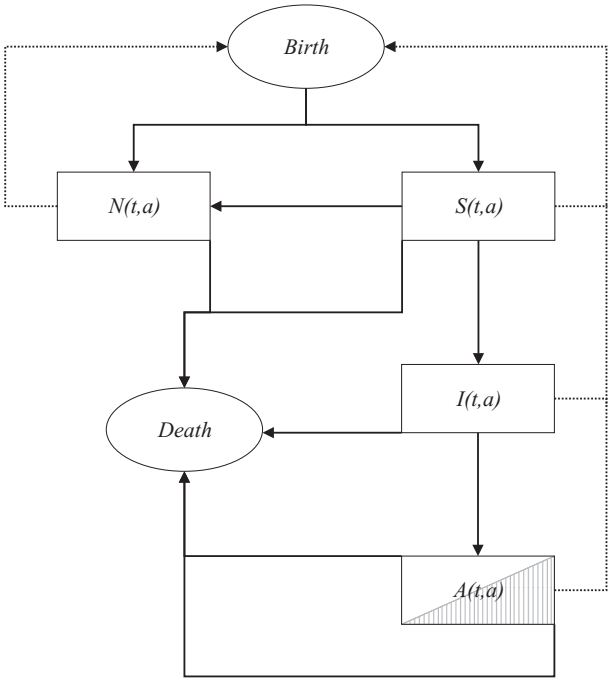


Figure 1: Scheme of the different population groups and the flow between these groups. The fraction of the medicated persons in the A-group is indicated by the dashed area.

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 have 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).

The dynamics of the model can be described by the equations below for $t \in [0, T]$, $a \in [0, \omega]$.

$$
\begin{aligned}
N_t(t,a) + N_a(t,a) &= -\mu_d(a)N(t,a) + \Psi(v(t))\kappa(a)S(t,a) && (1)\\
S_t(t,a) + S_a(t,a) &= -\mu_d(a)S(t,a) - \mu_{SI}(t,a)S(t,a) - \Psi(v(t))\kappa(a)S(t,a) && (2)\\
I_t(t,a) + I_a(t,a) &= -\mu_d(a)I(t,a) - \mu_{IA}(a)I(t,a) + \mu_{SI}(t,a)S(t,a) && (3)\\
A_t(t,a) + A_a(t,a) &= -\mu_d(a)A(t,a) - \mu_{dA}(a)(1-\delta(w(t)))A(t,a) - && (4)\\
&\quad - \mu_{dM}\delta(w(t))A(t,a) + \mu_{IA}I(t,a)
\end{aligned}
$$

with initial and boundary conditions

$$
\begin{aligned}
N(0,a) &= N_0(a), \qquad N(t,0) = \alpha(B(t) + C_I(t) + C_M(t)) && (5)\\
S(0,a) &= S_0(a), \qquad S(t,0) = (1-\alpha)(B(t) + C_I(t) + C_M(t)) && (6)\\
I(0,a) &= I_0(a), \qquad I(t,0) = 0 && (7)\\
A(0,a) &= A_0(a), \qquad A(t,0) = 0, && (8)
\end{aligned}
$$

where

$$
\begin{aligned}
B(t) &= \int_0^\omega \Theta(u(t,a))\varphi(t,a)(N(t,a) + S(t,a))da && (9)\\
C_I(t) &= \int_0^\omega \Theta(u(t,a))\varphi_I(t,a)I(t,a)da && (10)\\
C_M(t) &= \int_0^\omega \Theta(u(t,a))\varphi_M(t,a)\delta(w(t))A(t,a)da. && (11)
\end{aligned}
$$

Here $\mu_{SI}(t,a)$ denotes the transition rate from susceptible to infected, i.e., the incidence rate, and $\mu_{IA}(t,a)$ 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), $\mu_{dA}(a)$ is the death rate due to AIDS without medication and $\mu_{dM}(a)$ is the death rate due to AIDS while under treatment.

Furthermore, $\delta(w(t))$ 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 $\varphi(a)$, $\varphi_I(a)$, and $\varphi_M(a)$ 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.

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

The control $v(t)$ represents the par capita money spent for long term transition 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)

$$P(t,a) = \int_0^\omega \rho(a,a') \frac{I(a') + \eta_A(1-\delta(w))A(a') + \eta_M\delta(w)A(a')}{S(a') + \eta_N N(a') + I(a') + \eta_A(1-\delta(w))A(a') + \eta_M\delta(w)A(a')} da', \tag{12}$$

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 between 0 and 1 which denote the different sexual activity in the different groups, and the different infectivity of the groups. Now the incidence rate can be written 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 $u(t, a)$:

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

In order to measure the efficiency of an intervention scenario we use the total economic output as follows:

$$J = \int_0^T e^{-rt} \int_0^\omega \{(g_N N + g_S S + g_I I + g_M\delta(w)A + g_A(1-\delta(w))A) - 2((\varepsilon_1 u + \varepsilon_2\kappa v)(N+S+I+A) + \varepsilon_3\left[c^* - w\right]\delta(w)A)\}\, da\, dt \tag{14}$$

where g_N, g_S, g_I, g_A, and g_M are the "values" at time t of one individual of age a in the specific group (or more specific their contribution to the GDP), $c^*(t)$ is the market price of the medication and ε_1, ε_2, and ε_3 are multipliers which allow to disregard the cost of prevention and medication.

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 [CCCHL89] and [TCC93] 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 [FTV04]

[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.

which, however, is not calibrated with real data. The results obtained for Botswana in Section 4 of the present paper are qualitatively consistent with those in [FTV04].

We mention also that the performance index (14) has a purely economic meaning and does not take into account ethical considerations. Nevertheless, disregarding the cost of prevention (by taking $\varepsilon_1 = \varepsilon_2 = \varepsilon_3 = 0$) and assuming equal productivity turns (14) into a purely demographic index. In fact, the economic output (14) plays only a secondary role in the subsequent analysis.

3 Parameters

For the fertility rate $\varphi(t, a)$ we are using data available on www.census.gov. Figure 2 shows the fertility rates from 1990, so there shouldn't be any effects due to HIV. All other parameters are from 1993. Hence the fertility rate is reduced by 5%. Furthermore, the rate has to be divided by two because this is an one-sex model. The fertility rate for the I-group is $\varphi_I = 0.8\varphi$ and the fertility for the medicated people is $\varphi_M = 0.8\varphi$. Furthermore we assume an exponential decrease of the fertility rate down to an average of 2 children per women in 2093. (This does not include the effects of prevention programs, such as free condoms, which reduce the fertility even more.) Furthermore we assume that all newborns of non-medicated AIDS-suffering mothers are HIV-positive. The fraction of HIV-negative newborns in the I group is 0.5 and in the M group 0.8. The fraction of non-susceptible births α is assumed to 0.1, i.e. 10% of the newborns are non-susceptibles.

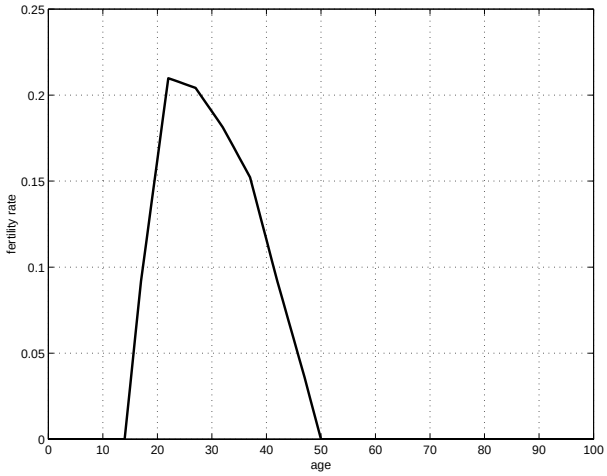


Figure 2: The age-specific fertility rate of Botswana from 1990

The data for the mortality rate $\mu_d(a)$ is derived from the Coale-Demeny model. Figure 3 shows the age-dependent rate. We assume that the mortality rates due to AIDS

(without treatment), $\mu_{dA}(a)$, an due to AIDS under treatment, $\mu_{dM}(a)$, are independent of the age. Furthermore we assume that 50% of the people having AIDS die per year. 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, the death rate is $\mu_{dM} = 0.105$.

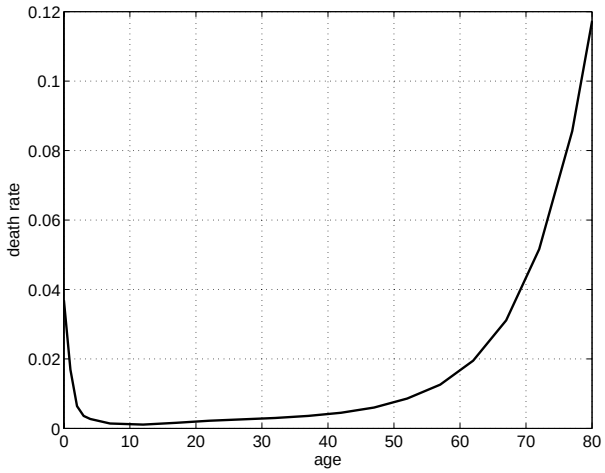


Figure 3: Natural death rate without HIV in Botswana (females)

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 sexual activity of η_N, η_A, η_M.

For the infection rate $\gamma(a)$ the same estimates are used as in [San04]. They are shown in Figure 4. The inter-age contact rates $\rho(a, a')$ is the fraction of (indirect) contacts of an a years old susceptible with an a' year 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 fulfill

$$\int_0^\omega \rho(a, a')da' = 1.$$

We used the following form for $a \geq 21$

$$\rho(a, a') = \begin{cases} 0.094 & \text{for } |a - a'| \leq 2 \\ 0.023(6 - |a - a'|) & \text{for } 2 < |a - a'| \leq 6 \\ 0.0037 & \text{for } |a - a'| > 6 \end{cases}$$

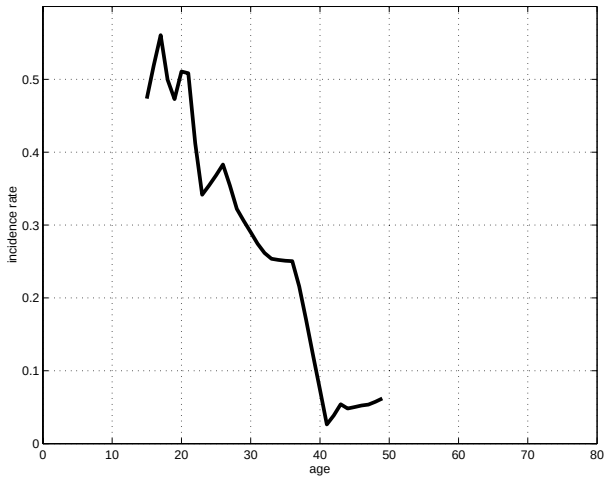


Figure 4: The estimates of the age-specific level of risk of infection (cf. [San04]).

and for $15 \leq a \leq 20$ the same function is used, but it is cut off at $a' = 15$ 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 infectiousness. In our simulation experiments we set this parameters to $\eta_N = 0.5$, $\eta_A = 0.5$, and $\eta_M = 0.1$. Here 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.

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, $\mu_{IA}(a) = \frac{2}{17}$.

In order to measure the economic effects of HIV/AIDS we use the per capita contributions to the GDP g_N, g_S, g_I, g_A, g_M. Despite that our model counts only the women, these constants are aggregated for all the adult population. For the groups N, S, and I we assume a per capita GDP of \$3020 . The contribution of persons suffering from AIDS is reduced to a third and the medicated persons are as productive as the healthy groups. Since we consider only the female population, we have to double this values to get them for the whole population. For a scenario where only the total population is evaluated, all contributions are set to 2 and all εs are set to 0. Furthermore we are using a 3% discount rate in our calculations ($r = 0.03$).

The influence of prevention programs on the number of risky contacts is represented by $\Phi(u)$. We assume an exponential decrease of risky contacts if the program is increased.

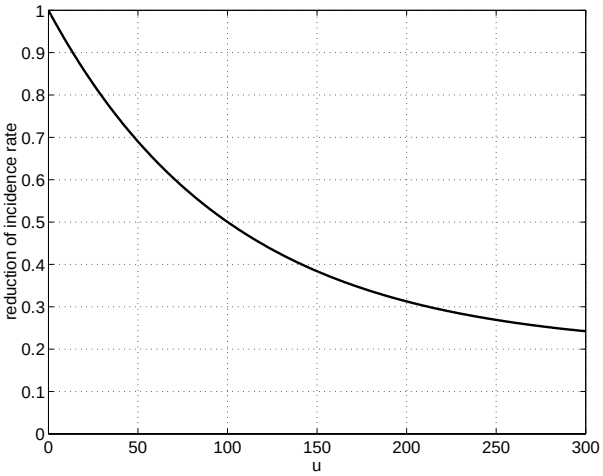


Figure 5: Reduction of the incidence rate due to money spent for instantaneous prevention programs

Furthermore we assume that the program can reduce the risky contacts only up to 80%. Hence, we have the form (see Figure 5)

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

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. Also in this case we use an exponential function to model the effect of diminishing returns (see Figure 6):

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

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

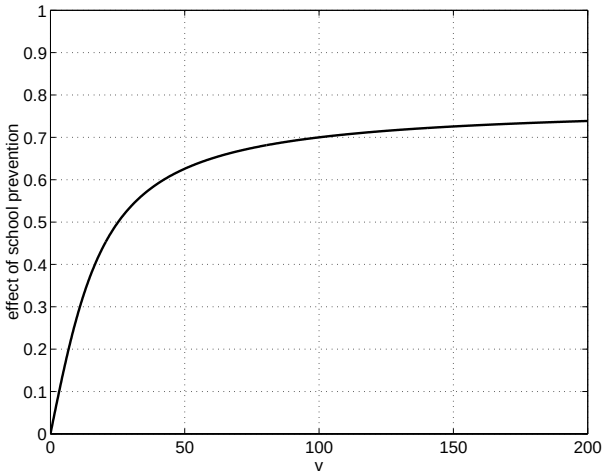


Figure 6: Effect of school-based prevention programs

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

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

$$\delta(w) = 0.7e^{-0.005w}.$$

These means that 70% of the female population would get medication if it is for free (see Figure 7). The market price of medication $c^*$is assumed to be constant at \$500.

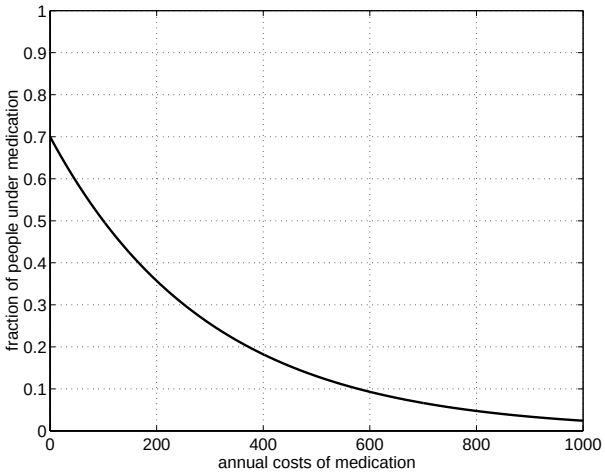


Figure 7: Fraction of people who can afford medication for different medication costs

Summary of parameters

parameter	description/value
$\phi(t,a)$	fertility rate of N and S group; fertility rate from 1990, exponential decrease to 2 children per woman in 2090.
$\phi_I(t,a)$	$[= 0.8\phi(t,a)]$ fertility rate of I group
$\phi_M(t,a)$	$[= 0.8\phi(t,a)]$ 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
$\mu_{IA}(a)$	$[= \frac{2}{17}]$ 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 N group
η_A	$[= 0.5]$ reduced sexual activity of A group
η_M	$[= 0.1]$ reduced sexual activity (=reduced infectivity) of M group
g_N, g_S, g_I, g_A, g_M	[=6040 (for N, S, I, M); =2013 (for A)] contributions of the different groups to the GDP
r	[=0.03] discount rate
$\Phi(u)$	influence of prevention programs on the number of risky contacts (see Figure 5)
$\Theta(u)$	$[= \frac{\Phi(u)+1}{2}$influence of prevention programs on the fertility rate
$\Psi(v)$	influence of school-based prevention programs (see Figure 6)
$\kappa(a)$	effective ages for the school-based prevention program
$\delta(w)$	fraction of medicated women depending on the price of medication w
c^*	$[= 500]$ market price of medication

4 Results

We made several simulations with different scenarios concerning the level of control which is used. If we look at the completely uncontrolled situation where no money is spent for (school-based) prevention and the price of medication is at the market price, we get results based on calculations starting from 1993. The total economic output according to equation (14) using a 3% discount rate is $J = 128.860$ billion\$. The total population will increase till 2010 and than it will decrease because of the high HIV prevalence which reaches a level of up to 28%. By 2050 the total population will be only two third of the 1993 situation. The number of deaths due to HIV will increase up to 30000 per year and than will decrease due to the decreasing population size.

Effects of single control applications

For each of the three controls we define 3 different scenarios:

1. apply no control (for the medication price this means that it is at market level)
2. 35% efficiency of the controls:

   medication price (w): The price is at \$206 per year, which allows 35% of the population to afford the medication if needed.

   school-based prevention (v): We spend \$13.54 per individual during the age of 12 to 17 for prevention. Hence $\Psi(v) = 0.35$, which leads to a total of about 15% non-susceptibles.

   instantaneous prevention (u): We spend \$58 per woman per year which results in a 35% reduction of the incidence rate.

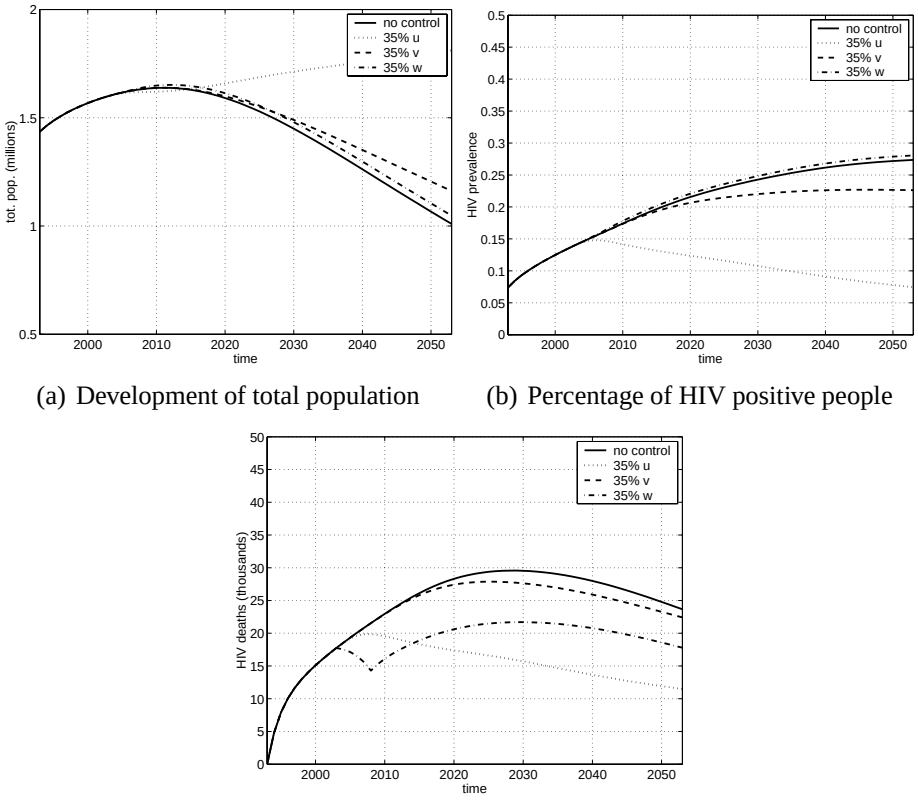
3. 70% efficiency of the controls:

   medication price (w): In this scenario we have free medication, which allows 70% of the population to afford the medication if needed.

   school-based prevention (v): We spend \$100 per individual during the age of 12 to 17 for prevention. Hence $\Psi(v) = 0.7$, which leads to a total of about 33% non-susceptibles.

   instantaneous prevention (u): We spend \$212 per woman per year which results in a 70% reduction of the incidence rate.

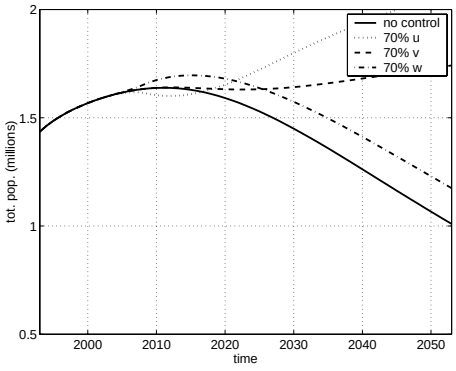
If a control is applied, it is done in the following way: The first 10 years until 2003 no control is applied, than during a 6-year period the control is increased linearly till it reaches the given level.



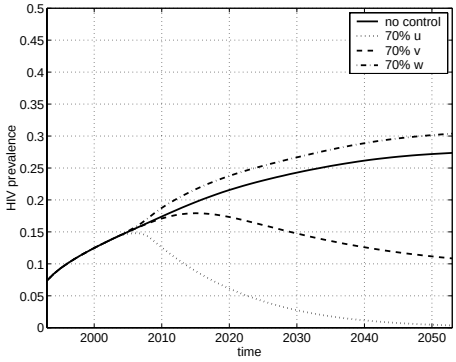
(a) Development of total population (b) Percentage of HIV positive people

(c) Total number of HIV induced deaths

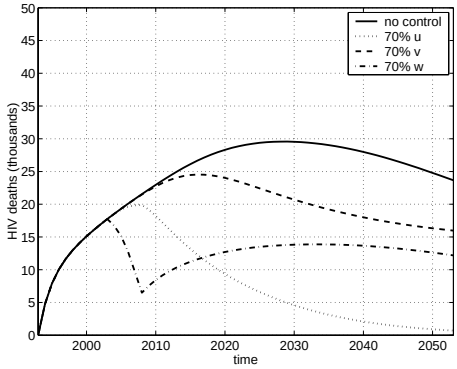
Figure 8: The development of the total population size, the HIV prevalence, and the number of HIV induced deaths from 1993 to 2053 using either no control or a 35% scenario for each intervention (instantaneous prevention, school-based prevention, subsidising medication) separately.



(a) Development of total population



(b) Percentage of HIV positive people



(c) Total number of HIV induced deaths

Figure 9: The development of the total population size, the HIV prevalence, and the number of HIV induced deaths from 1993 to 2053 using either no control or a 70% scenario for each intervention (instantaneous prevention, school-based prevention, subsidising medication) separately.

Figure 8 shows the effects, if we apply each control separately at a medium level and Figure 9 shows them for high levels of control. Instantaneous prevention is very effective on the long run, since the HIV prevalence will be reduced and the total population size will increase. The school-based prevention gains similar effects as the instantaneous prevention. From the figures we may conclude that school-based prevention is less effective, but we spend less money for that type of intervention. Furthermore we see that medication has a positive effect on the total population size at the beginning, but even if 70 per cent of the population have access to medication in the long run population size decreases. The reason is that medication prolongs peoples life, but in fact it increases also the HIV prevalence.The total economic outputs for the different scenarios are:

	0	35%	70%
u	124.860	133.832	132.823
v	124.860	126.683	133.500
w	124.860	126.056	129.532
w ($\eta_m = 1.1$)	123.835	124.189	126.017

This table shows that both types of prevention make sense also from an economic point of view. Instantaneous prevention is economically very efficient, but it is possible to "overshoot". A very high level of instantaneous prevention reduces the fertility and therefore the population shrinks and the total economic output is reduced. School-based prevention is quite effective, too, but there is not the problem of reducing the fertility. Whereas subsidising the medication costs is less effective. This, however, leads to a population at an endemic state with much lower total size and with about 30% prevalence. If we assume a higher infectiousness of the medicated population, the efficiency of subsidising the medication is reduced and for the 35% scenario in the long run the total population size falls below the level without subsidising (cf. Figure 10).

Combining controls

Now we consider the case when we apply a mixture of controls. For that purpose we investigate the marginal effects of a control in the presence of another one. Figure 11 shows the total population at different time points depending on the level of the instantaneous prevention u ($v = w = 0$). We see that at about \$50 per capita spending, the total population size can be stabilized. For values below, the population decreases, and above it increases. At about \$150 this control reaches its maximum efficiency with respect to the population size.

If we look at the efficiency of the prevention at time $T = 60$, that is in year 2053, with a simultaneous subsidisation of the medication price (see Figure 12), we recognize that a change of the medication price has only a minor influence on the positive effects of the

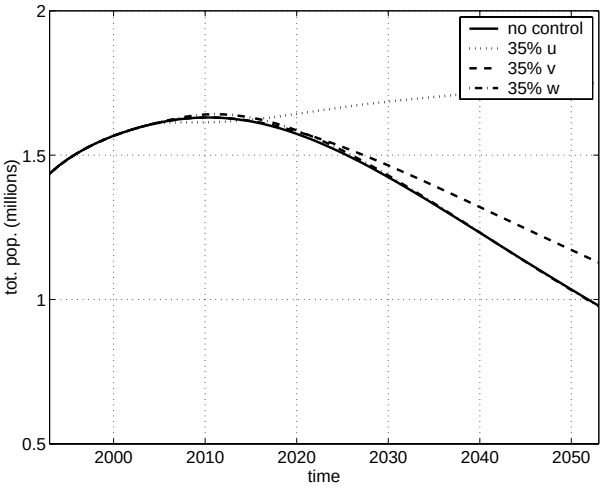


Figure 10: The development of the total population size from 1993 to 2053 using either no control or a 35% scenario for each intervention (instantaneous prevention, school-based prevention, subsidising medication) separately and assuming a higher infectiousness of the medicated people.

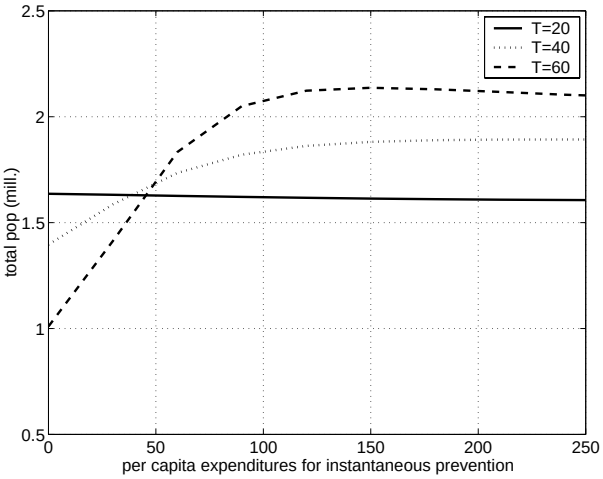
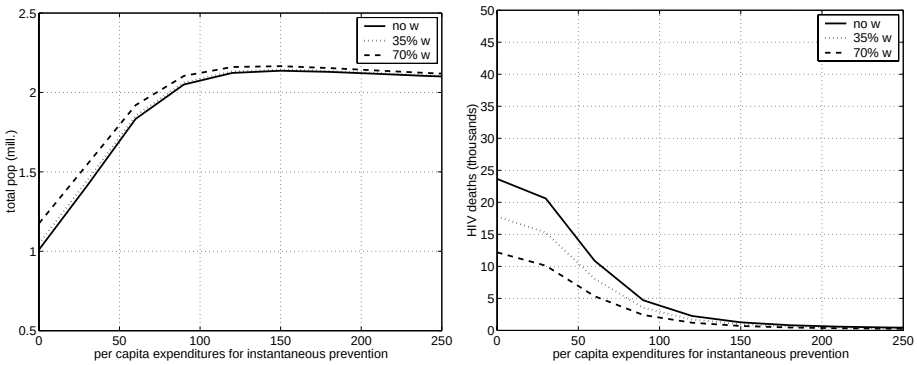


Figure 11: The total population after 20, 40, and 60 years (in 2013, 2033, and 2053) for different levels of instantaneous prevention.



(a) Total population in 2053

(b) Total number of HIV induced deaths in 2053

Figure 12: Comparing the effects of different levels of instantaneous prevention in the presence of different subsidies of the medication costs in 2053.

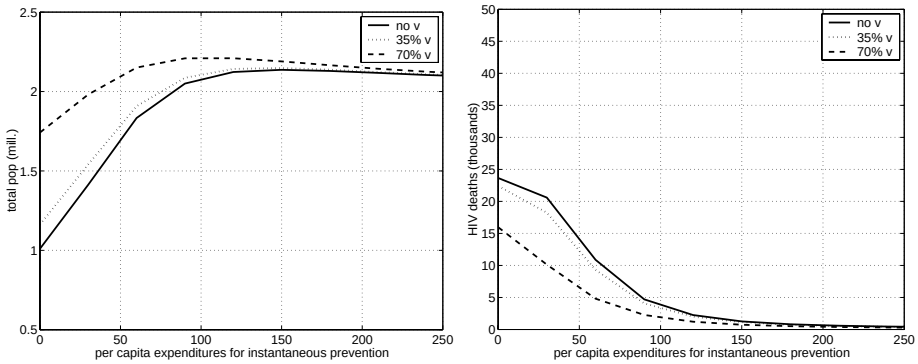
prevention program regarding the total population size. For low prevention programs a low medication price can reduce the number of deaths substantially.

On the other hand a school-based prevention program increases the efficiency of the instantaneous prevention (see Figure 13). If we spend about $100 per woman of age 12-17 for school-based prevention (70% scenario), we can reduce the instantaneous prevention by $50 per woman per year to reach the same level of population size.

Looking at the economic output (see Figure 14) we see that especially for low prevention levels school-based prevention is more efficient than a low medication price.

The effects of school-based prevention programs are straight forward. With about $75 per person spending we can stabilize the population size (see Figure 15). In contrast to the instantaneous prevention the medication price has no impacts on the efficiency of the school-based prevention (cf. Figure 16). Hence, high levels of medication allows to reduce the number of AIDS deaths at the end of the time horizon. Figure 17 brings up another interesting aspect: With an increasing level of school-based prevention, the level of instantaneous prevention would be reduced, because a medium level results in a higher population size and a higher economic output.

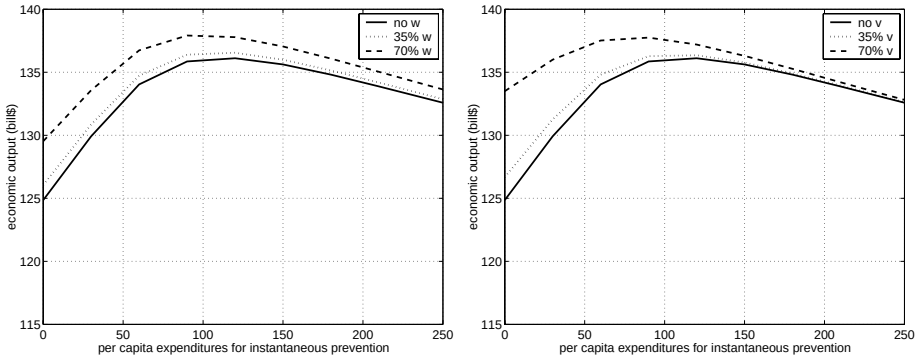
If we are looking at the effects of the medication price w (see Figure 18), we see that even with free medication it is not possible to stop the population decrease, if we do not use any other type of control. Furthermore the medication price has not a very strong influence on the population size in the sense that with low medication prices the population decreases. In the case of school-based prevention (see Figure 19) low prices for medication worsen the HIV prevalence but reduce the number of deaths. Figure 20 shows that if we apply a high level of instantaneous prevention, medication prices have no significant influence on the number of deaths in 2053 and the economic output.



(a) Total population in 2053

(b) Total number of HIV induced deaths in 2053

Figure 13: Comparing the effects of different levels of instantaneous prevention in the presence of a school-based prevention program in 2053.



(a) Economic output for different levels of instantaneous prevention for different medication levels

(b) Economic output for different levels of instantaneous prevention for different school-based preventions

Figure 14: The economic output for different levels of instantaneous prevention.

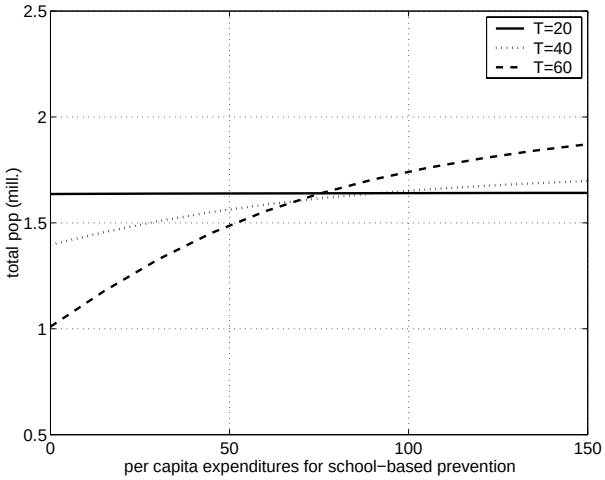
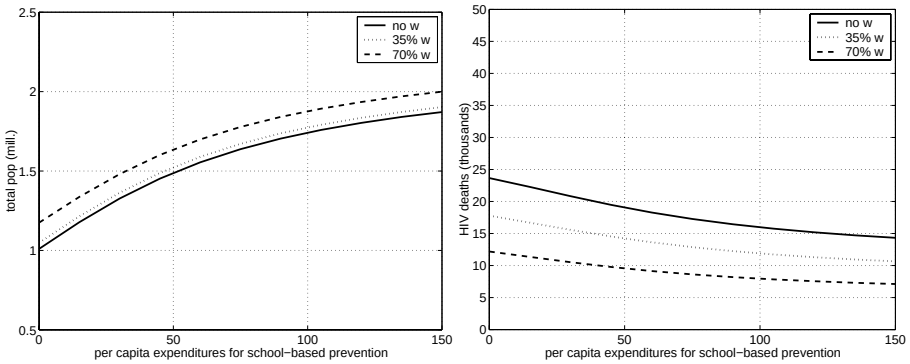


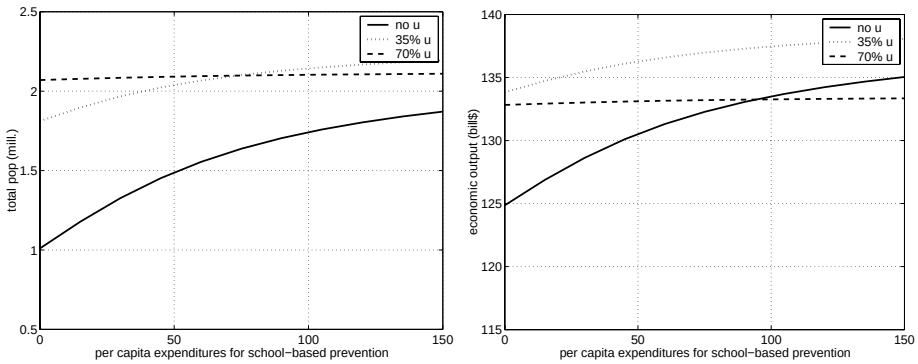
Figure 15: The total population after 20, 40, and 60 years (in 2013, 2033, and 2053) for different levels of school-based prevention.



(a) Total population in 2053

(b) Total number of HIV induced deaths in 2053

Figure 16: Comparing the effects of different levels of school-based prevention on the number of HIV deaths in the presence of different subsidies of the medication costs in 2053.



(a) Total population in 2053 (b) Economic output

Figure 17: Comparing the effects of different levels of school-based prevention in the presence of an instantaneous prevention program

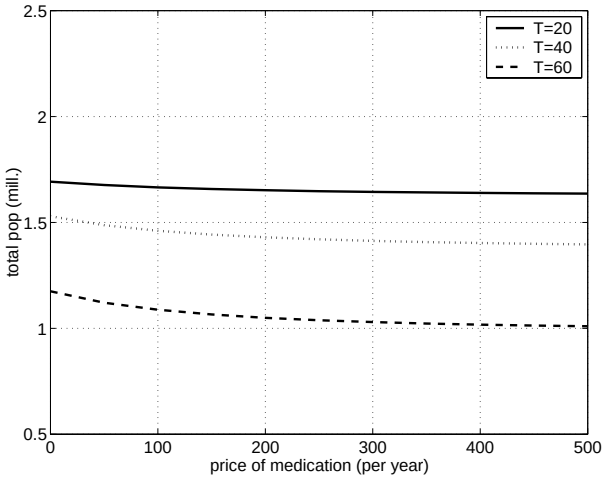
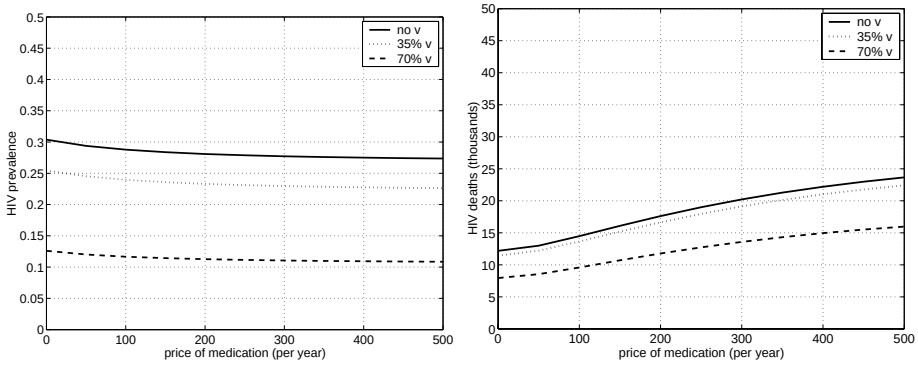


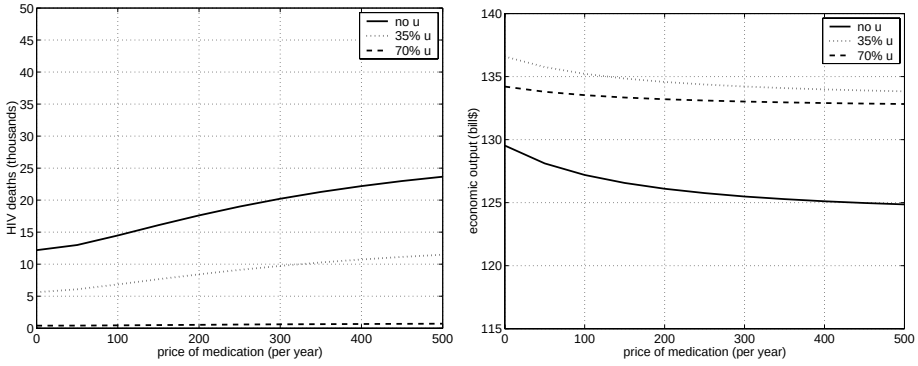
Figure 18: The total population after 20, 40, and 60 years (in 2013, 2033, and 2053) for different levels of medication costs.



(a) Percentage of HIV positive people in 2053

(b) Total number of HIV induced deaths in 2053

Figure 19: Comparing the effects of different medication prices in the presence of a school-based prevention in 2053.



(a) Total number of HIV induced deaths in 2053

(b) Economic output

Figure 20: Comparing the effects of different medication prices in the presence of an instantaneous prevention program.

5 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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