

# Dynamical models of HIV-AIDS effect on population growth

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## Abstract

We review some known dynamical models of epidemics, given by coupled systems of differential equations, and propose a simple SI-type model of HIV-transmission that accounts for population growth. It allows exact analysis of solution and makes specific predictions on the spread of HIV and its effect on populations. Then we outline an extended version of the model based on several behavioral/gender groups. We develop numerical code for solving thi system. The extended model corroborates some qualitative predictions of simple 2D system. We compare our predictions with some recent demographic data on the spread of HIV in several African countries.

The spread of epidemics could be modeled by coupled systems of differential equations, representing various populations and their interactions (behavioral patterns). Such modeling aims to analyze and predict the spread of epidemics, and also develop control/prevention strategies.

## 1 SI and SIR models with constant population.

The standard models of epidemics involve interacting populations:  $S$  - susceptible,  $E$  - exposed,  $I$  - infectious,  $R$  - removed (or recovered), etc. (see [3]). When  $I$  - individual infects  $S$  - individual a ceratin (latent) period could pass before he becomes infectious (e.g. 7 days for measles). The basic parameters in such models are the recovery/removal rate  $\lambda$  ( $1/\lambda$  - mean illness period) and transmission rate  $\beta$ . In some cases (short lived epidemics, like flu) the total population is considered constant,  $S + I + \dots = N$ , in other cases (AIDS) the population (growth) dynamics becomes important.

The basic  $SI$  - model has the form

$$\begin{cases} \dot{S} = -\beta \frac{SI}{N} + \lambda I \\ \dot{I} = \beta \frac{SI}{N} - \lambda I \end{cases} \quad (1)$$

where  $\beta$  is the transmission rate (number of infections per each  $I$ , per unit time) and  $\lambda$  - recovery. Model (1) allows no immunity, so after recovery  $I$  group move

immediately to the  $S$  compartment. To account for immunity one takes  $SIR$  model

$$\begin{cases} \dot{S} = -\beta \frac{SI}{N} + \mu R \\ \dot{I} = \beta \frac{SI}{N} - \lambda I \\ \dot{R} = \lambda I - \mu R \end{cases} \quad (2)$$

where  $\mu$  is the loss of immunity rate (e.g.  $\lambda = \frac{1}{7days}$ ,  $\mu = \frac{1}{150days}$  for flu). In case of finite exposure period, one needs a  $SEIR$  model

$$\begin{cases} \dot{S} = -\beta \frac{SI}{N} + \mu R \\ \dot{E} = \beta \frac{SI}{N} - \varepsilon E \\ \dot{I} = \varepsilon E - \lambda I \\ \dot{R} = \lambda I - \mu R \end{cases} \quad (3)$$

with latent period  $1/\varepsilon$ .

Clearly, all 3 models (1, 2, 3) conserve the total population  $N = S + I + \dots$ , and allow rescaling, so  $N$  could be made 1. Moreover, one could reduce each system to a lower order one, for instance (1) turning into a single logistic-type ODE:  $\dot{S} = (1 - S)(-\beta S + \lambda)$ . They share another common feature – two equilibria:  $E_1 = (N, 0, \dots)$  – the entire susceptible population, and  $E_2$  having persistent fraction  $I_0 \neq 0$  of infected (endemic infection). Both equilibria depend on a single dimensionless parameter

$$R_0 = \frac{\beta}{\lambda} \quad (4)$$

(“transmission” over “recovery”), called the *basic reproduction number*. If  $R_0 < 1$ , equilibrium  $E_1$  becomes stable, so no matter the initial state (e.g. large infected population), all solutions converge to  $E_1$ , hence we eventually eradicate epidemics. The implication for control strategy are clear now: one would like to bring  $R_0 = \frac{\beta}{\lambda}$  below 1 either by decreased  $\beta$  (prevention), or by cure (increased recovery rate  $\lambda$ ).

**Remark 1** *Let us note that the above models are appropriate for short lived and nonlethal infections, like flu or measles. Also for constant  $N$  one often writes transmission term as product  $bSI$  (e.g. [1], [2], [3]), equivalent to  $\beta \frac{SI}{N}$*

## 2 SIR-type models with variable population.

We claim that population change is essential for understanding AIDS on its time scales (incubation): years or dozen years, comparable to human life span and significant demographic changes. To this end we need to augment the appropriate equations with the growth (birth-death) terms. In the simplest case we take  $SI$  system with linear growth and consider two models:

$$(i) \begin{cases} \dot{S} = bN - dS - \beta \frac{SI}{N} + \lambda I \\ \dot{I} = -dI + \beta \frac{SI}{N} - \lambda I \end{cases} ; \quad (ii) \begin{cases} \dot{S} = aS - \beta \frac{SI}{N} \\ \dot{I} = \beta \frac{SI}{N} - \lambda I \end{cases} \quad (5)$$

Here  $b$ ,  $d$  - birth/death rates,  $a = b - d$  - growth rate,  $\lambda$  - recovery rate, and  $\beta$  - as above transmission rate. Model (i) assumes nonlethal infection so the removed population moves back to  $S$ , also all newborn are assumed susceptible. It does not represent AIDS, of course but rather a long term treatable disease (e.g. TB).

Model (ii) corresponds to a lethal infection, where parameter  $\lambda$  combines possible growth of infected population (e.g. newborn AIDS children) and removal due to illness,  $\lambda = d_I - b_I$ .

Note the total population  $N = S + I$  is not conserved in either case, it obeys an equation  $\dot{N} = aN$  in case (i), and a  $\dot{N} = aS - \lambda I$  in case (ii). Besides both systems are scale invariant,  $(S, I) \rightarrow (rS, rI)$ . Hence they have degenerate (one-parameter) equilibria, and the conventional stability analysis (section 1) fails.

To account for scaling property of (5) we introduce fraction<sup>1</sup>  $m(t) = \frac{I(t)}{S(t)}$ , that obeys a differential equation

$$(i) \frac{\dot{m}}{m} = [\beta - (b + \lambda)] - (b + \lambda)m; \quad (ii) \frac{\dot{m}}{m} = \beta - (a + \lambda); \quad (6)$$

The former one (i) is logistic, its roots (and stability) depending on control parameter  $\gamma = \beta - (b + \lambda)$ , the latter (ii) gives simple linear growth model with  $\gamma = \beta - (a + \lambda)$ .

The evolution of systems (i)-(ii) is completely determined by the sign of control parameter

$$\gamma = \beta - (b + \lambda) \text{ (i), or } \beta - (a + \lambda) \text{ (ii)} \quad (7)$$

Equivalently, we can introduce a modified version of the basic reproduction rate (4)

$$R_0 = \frac{\beta}{b + \lambda} \text{ or } \frac{\beta}{a + \lambda} \quad (8)$$

so that  $\gamma \leq 0$  corresponds to  $R_0 \leq 1$ . As above,  $R_0 > 1$  ( $\gamma > 0$ ) gives increased fraction of the infected population

$$m(t) \rightarrow m^* = \frac{\gamma}{b + \lambda}, \text{ as } t \rightarrow \infty \quad (9)$$

in case (i), and exponentially increasing  $m$  for AIDS (case ii), while  $R_0 < 1$  ( $\gamma < 0$ ) leads to eradication of epidemics.

Next we show that both systems (5) allow exact analytical solutions in terms of function  $m(t)$ , and thus are amenable to prediction and control.

## 2.1 Nonlethal case (i)

Here we get

$$\begin{aligned} S(t) &= \frac{N(t)}{1+m(t)} \approx \frac{N}{1+m^*} \\ I(t) &= \frac{m(t)N(t)}{1+m(t)} \approx \frac{m^*N}{1+m^*} \end{aligned}, \text{ as } t \rightarrow \infty,$$

<sup>1</sup>Alternatively, one could also use fraction  $I/N$ .

where  $N = N_0 e^{at}$  ( $N_0$  - initial population at  $t = 0$ ), and  $m^*$  - the limiting value of  $m$  (9).

So  $\gamma > 0$  ( $R_0 > 1$ ), i.e.  $m^* > 0$ , implies endemic level of infection,  $\frac{m^*}{1+m^*}$  - fraction of the total population  $N$ . Yet all three groups  $S$ ,  $I$  and  $N$ . maintain the natural growth rate  $a = b - d$ .

The implication for controlling epidemics (i) are similar to the previous case (section 1): either prevention (decreased  $\beta$ ), or cure (increased  $\lambda$ ) would bring  $R_0$  below 1.

## 2.2 AIDS case (ii)

Here exact analytic solution (Appendix), yield

**Case**  $\gamma > 0$  (uncontrolled growth  $\frac{m(t)}{m_0} \rightarrow \infty$ ). At large time solutions behave as exponential functions,  $\frac{S(t)}{S_0} \approx e^{(a-\beta)t}$ ,  $\frac{I(t)}{I_0} \approx e^{-\lambda t}$ , where  $a - \beta < -\lambda$ . So the infected population with larger exponent will dominate. Furthermore, assuming  $\lambda = d_I - b_I > 0$  (death rate due to illness exceeds birth rate among  $I$ ), both populations will eventually collapse to 0, as illustrated in Figures 1-2. The collapse (fig. 2) could be delayed by decreased transmission  $\beta$ .

Solutions in figures 1-2 use the standard growth (birth/ death) parameters

$$\begin{aligned} b &= \frac{\phi}{2L}, d = \frac{1}{L}; \text{ for } S \text{ - population} \\ b_I &= \frac{\phi}{2L}, d = \frac{1}{L_I}; \text{ for } I \text{ - population} \end{aligned} \quad (10)$$

where  $\phi$  denotes fertility,  $L$  - life expectancy of  $S$ ,  $L_I$  - illness duration. Here we adopt the following typical demographic values

$$\begin{aligned} \phi &= 4.5; L = 65 \text{ years}; L_I = 10 \text{ years}; \\ m_0 &= .01 \text{ (initial fraction of } I) \end{aligned} \quad (11)$$

**Case**  $\gamma = 0$  ( $R_0 = 1$ ). Here fraction  $m(t)$  remains constant, and both groups have the same asymptotic growth rate

$$\frac{S}{S_0} \approx e^{a't}; \quad \frac{I}{I_0} \approx e^{a't};$$

But the exponent  $a' = a - \frac{\beta m_0}{1+m_0}$  depends on the initial infected fraction  $m_0 = m(0)$ . The net effect of such stationary AIDS is to lower the natural growth rate  $a$ , maintaining a fixed infected fraction.

**Case**  $\gamma < 0$ . Now the growth pattern of  $m$  is reversed,  $m(t) \rightarrow 0$ . Thus we eradicate AIDS, restore the natural growth rate for  $S$ ,  $\frac{S}{S_0} \approx e^{at}$ , and drive  $\frac{I}{I_0} \approx e^{(a+\gamma)t} \rightarrow 0$ .

**Control strategy.** To eradicate epidemics we need once again to bring  $\gamma = \beta - (a + \lambda)$  below zero ( $R_0 < 1$ ). Clearly, prevention (decreased  $\beta$ ) would lower  $\gamma$  and  $R_0$ , as above (section 1, case (i)). But the effect of cure is quite different now, compared to previous cases. We recall the removal rate  $\lambda = d_I - b_I$ . Hence

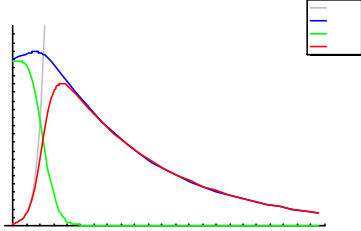


Figure 1: Population collapse for AIDS model with growth parameters (11) and  $\beta = 1.1$ , hence  $\gamma = 1.015$ ,  $R_0 = 13$ .

the “cure” (for incurable AIDS) would lower the death rate  $d_I$  without changing  $b_I$ . But that could only decrease  $\lambda$ , hence increase  $\gamma$  (7).

Here our simple 2D model of AIDS behaves markedly different from other epidemic models, it pinpoints unequivocally to prevention, as the only way of controlling epidemics.

### 3 AIDS model with behavioral groups

Next we shall extend our simple 2D model by including several behavioral groups and their interaction patterns. For the sake of demonstration we concentrate on sexually transmitted AIDS, and break  $SI$  populations into 3 groups: (male) homosexual, (male) heterosexual and females, designated by letters  $H, M, F$  for the  $S$ -group, and  $h, m, f$  for the  $I$ -group.

The growth parameters will include birth and death rates, determined by fertility and life expectancy (10), equally partitioned between two genders, and a (small) fraction  $\alpha$  of newborn homosexual males. The interaction (transmission) pattern is described in the following table

	h	m	f	
H	$\beta_{Hh}$	0	0	(12)
M	$\beta_{Mh}$	0	$\beta_{Mf}$	
F	0	$\beta_{Fm}$	0	

Here coefficients  $\beta_{ij}$  represent transmission rates among various groups, from  $I$  individuals (low case subscript) to  $S$ -individuals (upper case). Thus homosexuals could transmit AIDS to heterosexuals, the latter could exchange it with females and vice versa. The resulting 6D system of coupled differential

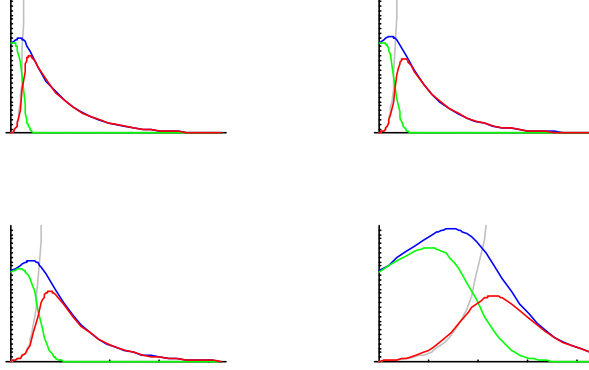


Figure 2: The effect of decreased  $\beta$  on AIDS dynamics for growth parameters (11). Plots (a), (b), (c), (d) correspond to  $\beta = 1.1, .8, .5, .2$ , and  $\gamma = 1.01538, 0.715385, 0.415385, 0.115385$ , respectively.

equations takes on the form

$$\begin{aligned}
 \dot{H} &= \frac{\alpha\phi}{L}F & -\frac{1}{L}H & -\beta_{Hh}\frac{H}{H+h}h \\
 \dot{M} &= \frac{(1-\alpha)\phi}{L}F & -\frac{1}{L}M & -\beta_{Mh}\frac{M}{M+m}h & -\beta_{Hf}\frac{M}{M+m}f \\
 \dot{F} &= \frac{\phi}{L}F & -\frac{1}{L}F & -\beta_{Fm}\frac{F}{F+f}m \\
 \dot{h} &= \frac{\phi}{L}f & -\frac{1}{L_I}h & +\beta_{Hh}\frac{H}{H+h}h \\
 \dot{m} &= \frac{(1-\alpha)\phi}{L}f & -\frac{1}{L_I}m & +\beta_{Mh}\frac{M}{M+m}h & +\beta_{Hf}\frac{M}{M+m}f \\
 \dot{f} &= \frac{\phi}{L}f & -\frac{1}{L_I}f & +\beta_{Fm}\frac{F}{F+f}m
 \end{aligned} \tag{13}$$

birth                      death                      transmission

System (13) seems fairly complicated to allow exact analytic solutions, its mathematical analysis posing a challenging task.

Here we shall bring some numeric results computed with Mathematica. We adopt the basic growth parameters of (11), beside we assume fraction  $\alpha = .01$  of newborn homosexual males, and take the following (hypothetical) transmission rates

$$\begin{array}{c|c|c|c}
 \beta_{Hh} & \beta_{Mh} & \beta_{Mf} & \beta_{Fm} \\
 \hline
 .1 & .01 & .6 & .4
 \end{array} \tag{14}$$

The following 3 plot show numeric simulation of the AIDS dynamics (13) for different initial infected fractions, and transmission rates. All runs are initialized with a small fraction  $h_0$  of the infected homosexuals, but eventually it spreads to all groups.

All three plots show qualitatively similar dynamic patterns to the 2D case (Fig. 1-2), the  $S$  - groups reaching peak first, followed by the  $I$  peaks and

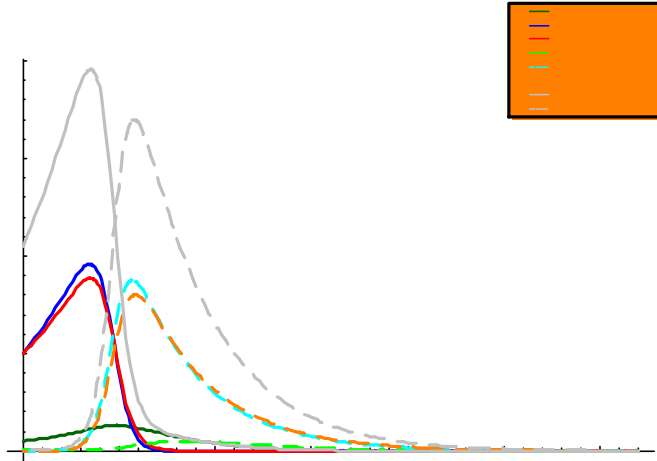


Figure 3: Initial 1% of infected homosexuals. Solid curves represent  $S$  - populations, dashed curves -  $I$  - populations.

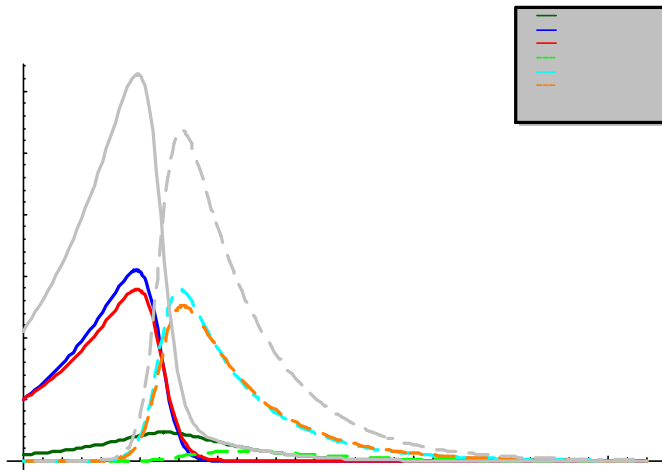


Figure 4: Initial 0.1% of infected homosexuals.

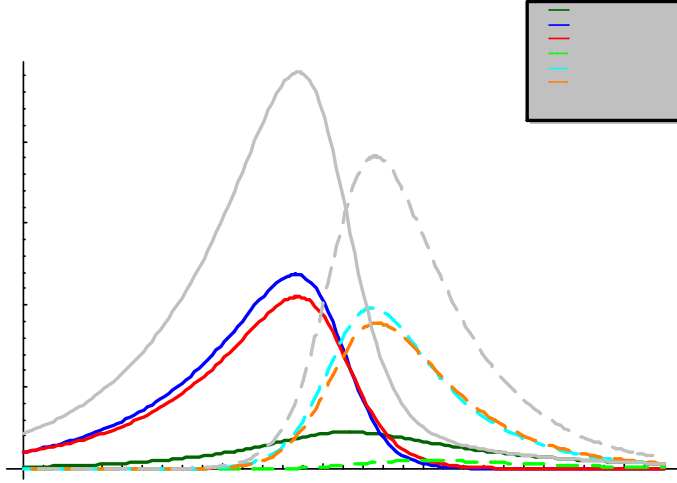


Figure 5: Same initial fraction  $h_0 = 1\%$  as plot 3, but transmission rates (14) reduced by factor 2.

the general decline of all populations. It takes about 12 years since the start of epidemics for the  $M, F$  -peak in plot 3, about 18 years in 4 and 40 years in 5, while homosexual group  $H$  has its peak delayed to 18, 23 and 48 years respectively. The  $S$  - peaks are followed by a steep decline: 10,15 and 30 years for males, roughly twice that long for females and yet slower decay of homosexual  $S$  - group. While the  $S$  - groups rapidly decline passed their peak, the  $I$  ones keep growing and soon dominate the balance. Yet after a while they also reach their peaks (27, 25, 60 years for female AIDS), and the entire population goes into irreversible collapse, for about 110, 120 and 140 years from the start of epidemics.

Comparison of fig. 3 and 5 shows the effect of prevention (decreased  $\beta_{ij}$ ), it delays peaks for all  $S$  and  $I$  groups, but does not change significantly the overall patterns and duration. More detailed analysis and numeric are needed to determine whether collapse could be averted, and how to control epidemics in the most efficient way.

Some recent observations of AIDS prevalence and its development in sub-Saharan Africa corroborate qualitative predictions of our simple models. But more work and substantial data analysis are needed to estimate parameters and validate them.

## 4 Summary

Epidemics in general and AIDS in particular, could be modeled by coupled systems of differential equations for  $S$  (susceptible) and  $I$  (infected) populations. Unlike other (short lived) epidemics, AIDS requires to accounts for the population growth, due to its lethal nature and time scales comparable to the human life-span. Here we propose a simple  $SI$  -type model whose basic parameters include removal rate for the I-group,  $\lambda = d_I - b_I$  (“death” - “birth”), and transmission rate  $\beta$ . Its analysis reveal an important control parameter  $\gamma = \beta - (a + \lambda)$ , or equivalent reproduction rate  $R_0 = \frac{\beta}{a+\lambda}$ . Or 2D model allows exact analytic solution, amenable to analysis, prediction and control. Thus  $\gamma > 0$  ( $R_0 > 1$ ) leads to the unchecked spread of epidemics and eventual collapse of the entire population, while  $\gamma < 0$  ( $R_0 < 1$ ) leads to its eradication.

In all cases control strategy requires  $R_0 < 1$ , and in most cases “cure” and “prevention” contribute equally to decreased  $R_0$  (or  $\gamma$ ). Yet unlike other epidemics, our 2D (incurable nature) AIDS allows only efficient control mechanism to make  $\gamma$  negative, namely through prevention (decreased  $\beta$ ). Partial cure without prevention would lower  $d_I$ , but would have opposite effect on  $\gamma$ .

We extend further the basic 2D model to accommodate various behavioral groups (homosexual, male, female) and transmission patters (for sexually transmitted AIDS). The resulting 6D system could be solved numerically. It exhibits qualitatively similar behavior, and resembles some recent data on AIDS trends in several African counties

The proposed models need further improved and validated by (i) including additional groups, population age bins, spatial distribution and migration patterns; (ii) by properly estimating the basic growth-transmission rates and patterns; (iii) by studying time or state dependent growth-transmission rates (change in behavioral patterns, or preventive measures).

One interesting aspect of sexually transmitted AIDS, suggested by our models, has to do with possible feedback of AIDS prevention strategies on the basic fertility and growth rates, hence its stabilizing effect on population growth. We plan to continue our study and address some of those issues.

## 5 Appendix: analytic solution of 2D AIDS model

By definition of  $m(t) = \frac{I}{S}$ , we get  $\frac{\dot{m}}{m} = \frac{\dot{I}}{I} - \frac{\dot{S}}{S}$ , hence for AIDS model (5) (ii)

$$\frac{\dot{S}}{S} = a - \beta \frac{m}{m+1}; \quad \frac{\dot{I}}{I} = \beta \frac{m}{m+1} - \lambda$$

Thus we get a differential equation (6) for  $m$ , that could be solved in quadratures and integrated to compute  $S$  and  $I$

$$\log \frac{S}{S_0} = at - \beta \int_0^t \frac{m}{m+1} dt; \quad \log \frac{I}{I_0} = -\lambda t + \beta \int_0^t \frac{1}{m+1} dt$$

The off-shoot are explicit formulae for  $S$  and  $I$

$$\frac{S}{S_0} = \begin{cases} e^{at} \left( \frac{1+m_0}{1+m_0 e^{\gamma t}} \right)^{\beta/\gamma} \approx e^{(a-\beta)t} & \gamma \neq 0 \\ \exp \left[ \left( a - \frac{\beta m_0}{1+m_0} \right) t \right]; & \gamma = 0 \end{cases};$$

$$\frac{I}{I_0} = \begin{cases} e^{(\beta-\lambda)t} \left( \frac{1+m_0}{1+m_0 e^{\gamma t}} \right)^{\beta/\gamma} \approx e^{-\lambda t} & \gamma \neq 0 \\ \exp \left[ \left( a - \frac{\beta m_0}{1+m_0} \right) t \right]; & \gamma = 0 \end{cases}$$

## References

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