RESOURCE ALLOCATION STRATEGIES FOR COMBATING HIV EPIDEMICS

A Thesis in Mathematics
by
Asya Pritsker

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The thesis of Asya Pritsker was reviewed and approved* by the following:

Timothy Reluga  
Assistant Professor of Mathematics and Biology  
Thesis Advisor

Joel C. Miller  
Assistant Professor of Mathematics and Biology

Svetlana Katok  
Professor of Mathematics  
Head of Graduate Studies

*Signatures are on file in the Graduate School.
Abstract

Sub-Saharan Africa has some of the highest rates of HIV-1 infection in the world; some communities suffer infection rates upward of 30 percent. Apart from affecting the health of individual people, the impact on community structure is significant. The disease affects the ability of adults to provide for their communities, and high adult mortality has led to many orphaned children. In many cases, it is impossible for a community to afford medication for all of the ill people. As the prevalence of HIV increases, the ability of the community to provide for itself is even further depleted. It has also been observed that a high ratio of children to adults causes decreased social stability and interrupts cultural continuity.

We propose a targeted approach to battling the epidemic consisting of focusing available resources on treating only a particular demographic. We use a mathematical model to compare how different resource allocation strategies might affect health outcomes as well as the economic state of the community. We evaluate strategy outcomes based on mortality and prevalence, as well as by measuring whether a sustainable ratio of producers to consumers is maintained. The model is discrete time, continuous population, and compartment-based.

Based on our construction, we demonstrate that exhausting significant resources on treating consumers makes very little impact on retarding the epidemic, while focusing on treating producers makes a very measurable impact.
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Chapter 1

Introduction

1.1 HIV and AIDS

Human Immunodeficiency Virus, or HIV, emerged approximately thirty years ago, and has rapidly become a major health and economic problem all over the world.

HIV leads to acquired immunodeficiency syndrome, or AIDS. The effect of AIDS on an individual’s health is catastrophic. HIV damages the immune system, and the patient suffers weight loss, neurological symptoms, fungal overgrowth, and many opportunistic infections. The progression of the disease is usually measured through the CD4+ count; a lower count indicates a more damaged immune system. There is no cure for HIV and no vaccine. Prevention measures include using barrier protection methods during sexual intercourse, not sharing needles, limiting number of contact partners, and getting tested at regular intervals after possible exposure. The time from when an individual is exposed to the virus until he or she develops visible symptoms can range from 2 to 15 years [1]. There is evidence that the incubation period is much shorter in impoverished regions of Africa, with a median close to four years, than in the United States and Europe, where the median is between 8 and 11 years [2].

HIV is transmitted through bodily fluids, both by direct transmission of the virus and by transmission of infected cells present in bodily fluid. The greatest incidence is due to unprotected sex, but practices such as sharing needles, either in clinics or for recreational intravenous drug use, also contribute to the spread of the virus. Transmission also occurs from mother to child; this is called vertical transmission. If a mother is HIV positive and there is no intervention, the probability that she will pass the virus to her child either during pregnancy or breast feeding is between 15 an 45 percent [1]. Testing for HIV is done through a couple of different assays such as ELISA. A home HIV test has become available in the United States in the last six months. Broad availability of such a test has the potential to decrease spread of disease, but the effect of testing without counseling is not clear. Individual knowledge of HIV status is only a small part of changes in behavior that would lead to decreased transmission.
The only effective treatment currently is a complicated course of multiple antiretroviral drugs, often abbreviated ARV or ART. The most effective and common regimen currently in use consists of three different medications, and is known as highly active antiretroviral therapy, or HAART. It is very effective at reducing symptoms as well as viral load. An important additional benefit of this is that individuals on this combination therapy are much less likely to transmit the disease either through interpersonal contact or vertically. Unfortunately, it is often difficult for patients to adhere to the regimen because of its complexity and side effects. Therapy or counseling in conjunction with treatment becomes extremely important to realizing the full potential of the drugs.

1.2 The Epidemic in Sub-Saharan Africa

Today, the greatest impact of HIV can be seen in Sub-Saharan Africa (SSA), where infection rates reach 37 percent of the adult population in some communities. This region is home to 68 percent of all infected individuals, while the population is 12 percent of the global total. While incidence is dropping globally, 70 percent of new infections occurred here in 2010 [3]. Even more staggering is that an estimated 85% of deaths due to HIV occur in this region [4]. In a limited way, this paper will explore some of the reasons for why HIV has spread so much more rapidly in this part of the world than in the United States or European countries.

![Figure 1.1: Estimated adult age 15-49 prevalence rates by country in 2011. Source is UNAIDS country reports.](image)

<table>
<thead>
<tr>
<th>Country</th>
<th>Adult HIV Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Africa</td>
<td>17.30%</td>
</tr>
<tr>
<td>Namibia</td>
<td>13.40%</td>
</tr>
<tr>
<td>Zimbabwe</td>
<td>14.90%</td>
</tr>
<tr>
<td>Tanzania</td>
<td>5.80%</td>
</tr>
<tr>
<td>Mozambique</td>
<td>11.30%</td>
</tr>
<tr>
<td>Kenya</td>
<td>6.20%</td>
</tr>
<tr>
<td>Congo</td>
<td>13.80%</td>
</tr>
<tr>
<td>Zambia</td>
<td>12.50%</td>
</tr>
<tr>
<td>Malawi</td>
<td>10.00%</td>
</tr>
<tr>
<td>Botswana</td>
<td>23.40%</td>
</tr>
</tbody>
</table>

It is important to note that even within each country, there is great regional variation in prevalence rates. The wealthier parts of South Africa have infection rates of a fraction of a percent, while some rural impoverished communities suffer prevalence among adults of over 30 percent [5]. Risk factors which might vary by region include sexual contact rates, intravenous needle sharing, the cultural importance of blood-based rituals, and general medical understanding of disease spread mechanisms. When modeling a particular community, it is crucial to understand
these differences to be able to intervene effectively. Furthermore, contrary to stereotype, the primary mode of transmission of HIV in this region is probably not sexual. HIV is not transmitted very efficiently through sexual means and the spread of HIV has not be very closely correlated with the spread of other STIs since 1998 [5], at least in some regions of SSA.

Throughout the 80s and early 90s, the scope and severity of the HIV epidemic became more clear, but the epidemic continued to grow as governments and international organizations mounted a response. The impediments have been many, as there is a great stigma associated with the disease in many cultures and, as described above, treating already infected individuals is both expensive and difficult. Today, the World Health Organization (WHO), UNAIDS, and UNICEF are the leaders in setting global policy for battling the epidemic; they work with local governments to help them organize and implement prevention and treatment programs. It is extremely important that local governments strive for sustainability and self-sufficiency in this issue.

Two notable large-scale interventions in SSA are those organized by the national governments of South Africa and Malawi. Official acknowledgment of HIV as a national problem is a crucial step toward overcoming prejudice against individuals with HIV and stopping the epidemic.

In South Africa, an organized government response was mounted approximately 15 years ago [5]. The non-medical response includes laws protecting individuals from discriminatory hiring practices relating to infection status and requiring safe working conditions which minimize the rise of HIV transmission. The medical response has been based around the formation of accredited public health facilities and the implementation of treatment guidelines. To receive treatment, an individual must at least be either in WHO Clinical stage 4 of disease (obviously symptomatic) or have a CD4+ count of less than 200. The treatment facility personnel also have the discretion to make the decision to enroll a patient based on his or her demonstrated reliability and willingness to commit to the treatment regimen. The purpose of the is to focus on using resources efficiently and making sure that patients get the maximal benefit of the treatment. Incomplete treatment can lead to relapsing and the evolution of drug resistance in the virus. Medical criteria for treating children are lower than those for treating adults, but there is the additional requirement of the presence of an adult able to oversee treatment and transport the child to the clinic regularly. It is obvious that this could be a limiting factor. The third portion of the initiative which we want to highlight is the active effort to educated the population, especially teenagers and young adults, about the mechanisms and risks of transmission.

In Malawi, the first organized response was mounted in 2003 [4]. Between 2004 and 2006, the number of public HIV treatment facilities was scaled up from 9 to 60. These hospitals provide ART free of charge. Realistically, Malawi hopes to have 50% of eligible infected individuals (around a quarter million) receiving treatment by 2010. The country is actively trying to scale up treatment of children. The public clinics provide counseling as well as treatment for eligible individuals. In Malawi, these are those who have tested positive for HIV and are either at least in WHO clinical stage 3 or have a CD4+ count lower than 200 parts per µL. DNA-PCR tests are used and relied upon in these clinics.
Thanks in part to the government acknowledgment of and commitment to fighting the HIV epidemic, some of the best data we have is from studies conducted in the KwaZulu-Natal region in South Africa and from the regional hospital in Chiradzulu in Malawi.

Until recently, testing for HIV has been very rare in SSA. The tests are expensive, and there is often a severe stigma against the infected, so an individual would not go to be tested even if he or she thought she had been exposed. Awareness of seropositive status is less than 20% in many parts of SSA [3]. In the last couple of years, inexpensive and quick tests using saliva have become available. Currently, the most effective scale-up of testing involves trained health workers going door to door and including HIV testing among other medical services. The uptake is increasing, but it will probably be some time before this can be reflected in the epidemic [6]. Unfortunately, it is also the case that the HIV epidemic has put further strain on health care systems in developing countries. Most preventative strategies are not effective without a large amount of trained manpower for things like counseling about sexual behaviors and adherence to treatment [7].

The AIDS epidemic has fundamentally changed the demography of these countries. It is estimated that the life expectancy of an individual at birth has been shortened by 20 years. In addition, the make up of the population is now heavily skewed toward young people.

<table>
<thead>
<tr>
<th>Country</th>
<th>Percent of population under age 15 (2011)</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Africa</td>
<td>28.4</td>
</tr>
<tr>
<td>Namibia</td>
<td>33.4</td>
</tr>
<tr>
<td>Zimbabwe</td>
<td>40.6</td>
</tr>
<tr>
<td>Tanzania</td>
<td>45.0</td>
</tr>
<tr>
<td>Mozambique</td>
<td>45.7</td>
</tr>
<tr>
<td>Kenya</td>
<td>42.5</td>
</tr>
<tr>
<td>Congo</td>
<td>45.3</td>
</tr>
<tr>
<td>Zambia</td>
<td>46.3</td>
</tr>
<tr>
<td>Malawi</td>
<td>44.9</td>
</tr>
<tr>
<td>Botswana</td>
<td>33.5</td>
</tr>
</tbody>
</table>

Figure 1.2: Table containing the percent of population of country under age 15. Source is CIA World Factbook.

This phenomenon has been referred to as the orphan problem; because a very large proportion of children have lost one or both parents and are living with extended relatives [8]. High adult mortality has led to great social instability in many regions of SSA, not only because of the disproportionate numbers of children, but because households which have lost at least one adult member are many times more likely to dissolve than those who have not [9]. Households which dissolve, or have too many children being cared for by not enough adults cannot function as a cohesive social unit. They suffer increased poverty as a result of losing a producer, which in turn leads to fewer opportunities for children to go to school.

The economic impact of AIDS on communities, especially those that rely on agriculture to
survive, is devastating. In most of these, health insurance and disability insurance are unheard of, and government unemployment assistance is limited. Many adults work in labor jobs; they become gradually less productive as the infection reaches later stages until they are finally laid off [10]. AIDS is particularly economically devastating because it hits the most productive portion of the population, ages 20-45, the hardest.

1.2.1 The Impact of ART on Health and Economy

The impact of antiretrovirals in controlling the AIDS epidemic is undeniable. Besides improving individual health outcomes by lowering viral load and improving CD4+ counts [11], ART has enormous positive effects on community stability and productivity. Individuals undergoing ART live longer, and work more days per month [12–14], which allows them to contribute to their communities almost as much as uninfected individuals. A very recent paper has shown that just being in a community with high ARV treatment rates decreases an individual’s chance of contracting the disease [15].

There is also evidence that there are improved education outcomes for children in households where the HIV-infected adults are receiving treatment, especially in the over 12 age group [12]. Beyond the overall positive impact of education on community development, this is a critical time to educate children about how disease spreads and to implement prevention initiatives.

1.3 Resource Allocation Strategies: Our model

In this paper, we want to focus on designing the most efficient intervention strategies that a community could implement in deciding how to use any available ART. Knowing that it is impossible to treat everyone, which subpopulation is the most important to target with ART? We propose what we believe to be a novel approach: to focus entirely on treating adults. It is absolutely crucial to decrease the number of new infections occurring, but it is also important to consider the overall outcome for a community. Rather than looking only at raw prevalence and incidence rates as a measure of the success of an intervention, we must strive to make sure that communities are developing and sustaining themselves. The key to this is to ensure a productive and able-bodied adult population.

Treating adults has numerous secondary consequences, some of which we have outlined above. In summary, children living in households with able-bodied adults are at a great advantage in terms of health and education, making them less likely to become infected. Furthermore, not only are adults primarily the individuals who spread disease (thus making treating adults more important just to prevent spread through contact), there is evidence that undergoing ART in a structured program significantly reduces risky sexual behavior among infected individuals [?, transmissionhetero]

While current initiatives are clearly working toward stemming the epidemic- incidence rates have falling 20% in the last ten years [16], funding for HIV interventions is flatlining. The reasons
for this vary, and include economic changes in the West, but the need for understanding the most
efficient use of resources—both human and medical—is becoming all the more important [17].
The ultimate goal is for a community to be able to afford to treat all of the infected members.

We approach this question by building a mathematical model and testing the effects of different
resource allocation strategies within it. Chapter 2 describes in detail the construction of our
model, and Appendix A gives details about the parametrization. Briefly, we use a compartment-
based approach with three stages of infection (susceptible, asymptomatic infected, symptomatic
infected) and two age stages [18]. Our two age stages are child and adult, which we also identify
with consumer and producer. From the epidemiological standpoint, this distinction makes sense
because the modes and rates of transmission vary most fundamentally between these two groups.
We consider those under age 15 to be children—this agrees with all of the studies we found.
The assumption that all people over the age of 15 are productive adults is certainly arguable,
as the elderly almost certainly become dependents at some point. But overall, adults over the
age of 60 make up a very small portion of the population, and it seems as if the infection rates
and sexual behaviors of this small subpopulation fit most closely with those of younger adult
[19]. The model could be altered with an additional stage of adult consumers if there were more
information about this group in a particular community.

The distinction between symptomatic and asymptomatic individuals is also important. Rates
of transmission in these two groups will differ, because symptomatic individuals are more infec-
tious through contact. We group together WHO stages I and II as asymptomatic individuals or
those with very mild symptoms that do not necessarily indicated HIV infection, and WHO stages
III and IV as severely impaired symptomatic individuals and those very low CD4+ counts. This
is well aligned with our economic model (described in the next paragraph) and with treatment
regimens in many countries, such as Malawi and South Africa.

We also include a very simple linear economic model; it has parameters for consumption and
production. We assume that productivity is negatively impacted by progressing disease, and
that ART returns a significant portion of the productivity lost to an individual suffering from
AIDS [13, 14]. Our economic model is not meant to measure the actual economic output of a
community—it is meant to give an overall measure of working potential and the ratios of healthy
producers to the rest of the community. The question of the actual economic impact of HIV is
very complicated and would depend on the specific setting, as well as the industry upon which
the community depends. As just one example of this complexity, the productivity of men and
women is affected differently, and there is evidence that when one member of a family falls ill,
another may be able to compensate for this. Another complication (though a positive one) can
arise because some private companies actually provide HIV treatment to their employees [10].
The question is being studied by economists [13, 20], but it outside of the scope of our model.
While we rely on this data to tell us the magnitude of ARV on an individual’s productivity, we
otherwise pick parameters for consumption and production that illustrate the magnitude of the
impact.

Our model is meant to represent a limited type of community: rural, subsistence (typically
farming) villages. These are areas where resources are particularly limited, and access to medication is the most difficult to provide. Travelling to a clinic can take many hours, and would take an individual away from their work and from the community. If he or she were already very ill, this might not even be possible. Most importantly, we believe our simplified economic model is sufficient in this scenario, because public health decisions would be made on the level of the community, probably by village elders. Our model does not deal with the impact of non-medical interventions, as these are difficult to quantify.

Chapter 2 describes the construction of the model and gives all of the equations. Chapter 3 describes the results of our simulation, and Chapter 4 explores the robustness of the model.
Chapter 2

Model Construction and Assumptions

We will explain the construction of the model in this chapter, but please see Appendix A for more detailed information about choice of parameters and the values we use for them in the simulations.

2.1 General Approach

Our goal is to study the efficacy of different resource allocation strategies in battling the HIV epidemic in Sub-Saharan Africa. We are interested not only in minimizing mortality rates, but in maintaining the adult community as a priority.

We use an SI model with six compartments which distinguishes between healthy, infected, and treated and untreated transmission. The compartments are susceptible producers, $S_p$, susceptible consumers, $S_c$, infected producers in the early (mostly asymptomatic) stages of disease $I_p$, infected asymptomatic consumers $I_c$, producers suffering from late stages of disease, $I_{pa}$, and consumers who are suffering from full-blown AIDS, $I_{ca}$. Each compartment has rates of contact and degrees of infectiousness, as well as different death rates and other demographic parameters, but individuals in each compartment are indistinguishable from one another. We do not incorporate possible changes in behavior with HIV status into the model because there is no consistency across communities with respect to this. We also do not incorporate testing, because it is not readily available or used in many communities in rural Africa.

We are considering small villages of approximately 100 people. We assume linear birth rates; there is no density dependence in the population growth.
2.2 Economic set up and assumptions

We do not incorporate information about family structure. For most purposes, this is the level on which economic resources are shared, but it is not the level on which treatment decisions would be made. Public health decisions, such as resource allocation for ARVs, would be made by village elders.

We make the simplifying assumption that people in the community fall into two categories—producers and consumers. Consumers are children under the age of fourteen, and they are assumed to be entirely dependent on producers. Producers are people over the age of fourteen; they provide for the community in some way. We categorize the elderly as producers since they provide very important non-economic benefits to the community, such as child care. For instance, it has been shown that the presence of grandparents has a significant positive impact on the life expectancy of children.

Thus, we measure the overall economic health of the community as reflected by the balance of able-bodied producers and incapacitated producers with the entire population. This is not the actual money generated by the community. The distinction between producers and consumers also allows us to capture the differences in disease transmission between the two groups.

To be able to measure the impact of the disease on the community, we need to capture how the increase in infected producers lowers the overall amount of resources the community is able to provide for itself.

HIV impacts the ability of adults work and to care for children, so infected producers who are not undergoing treatment are less productive than healthy or infected asymptomatic adults. We assume that treatment makes infected individuals fully able-bodied, as well as less likely to spread infection through contact.

We assign a consumption rate for consumers and producers, \( \eta_c \) and \( \eta_p \) units per time interval, respectively. Then we suppose that susceptible producers produce \( \lambda \) units per time period, asymptomatic producers have a productivity of \( \lambda_h \), and symptomatic producers have the most severely reduced productivity \( \lambda_a \). Producers who are receiving ARV treatment will have an improved productivity that is \( \lambda_{tr} \), which reflects how close an individual returns to the productivity they has as an asymptomatic individual.

2.3 Population dynamics in the absence of HIV

The model of the population dynamics in the absence of disease is linear, and can be described by a 2x2 projection matrix \( M \). The time projection interval is a month.

We only consider two compartments, \( S_c \) and \( S_p \), which stand for susceptible consumer and susceptible producer, which we take to correspond to child and adult. As mentioned in Chapter 1, we consider older adults to still be producers. This demographic makes up a very small percentage of the population, and healthy older people provide services like child care. Their presence contributes to the social stability of a community, and their sexual contact patterns and
rates are similar to those of younger adults [19].

The death rates for consumers is labeled $\gamma_1$, and the death rate for producers is labeled $\gamma_2$. Children grow up at a rate $\beta$, and are born at a rate $\alpha$ per adult (producer). The assumption that these rates are linear may not be accurate, since this assumes a uniform distribution of all ages in each cohort. We use this average, but this could be made more accurate by using (for instance) five year cohorts instead of only two large groups.

$$M = \begin{pmatrix}
(1 - \gamma_1)(1 - \beta) & \alpha \\
\beta & 1 - \gamma_2
\end{pmatrix}$$  

(2.1)

Then the population vector at time $t + 1$ is $N(t + 1) = MN(t)$, where $N(t) = [S_c(t), S_p(t)]$

We can measure the economic health of the community by saying the resources available at time $t + 1$ are

$$R(t + 1) = R(t) + \lambda S_p(t) - S_p(t)\eta_c - S_c(t)\eta_p$$  

(2.2)

As we increase the number of compartments in the model, $M_{ij}$ will always describe the rate at which compartment $N_j(t)$ contributes to $N_i(t + 1)$.

### 2.4 Spread of HIV in the absence of treatment

Now we introduce the effects and dynamics of HIV infection into the model. We do not include the possibility of treatment at this stage.

We call the class of infected producers who are not yet exhibiting obvious symptoms of infection $I_p$ and call the group of adults suffering from full-blown AIDS $I_{pa}$. Analogously, we separate infected consumers into $I_{ca}$ and $I_c$. $I_{ca}$ and $I_{pa}$ are our two classes of symptomatic individuals—those who reach WHO disease stage III or IV (measured by symptoms) OR whose CD4+ count falls below 200 parts per $\mu$L. We made this particular choice of compartments for the following reasons: firstly- individuals in the late stages of HIV will have significantly depleted productivity, secondly- individuals in the later stages of infection have a much higher viral load than those recently infected and thus are more likely to spread the disease with contact, and thirdly- in most countries, ART is not available to individuals in the earlier stages of disease.

In many countries, an individual is only eligible for treatment when their CD4+ count is below 200 [4,5], and around 200 parts is a number seen in many studies as the median at which patients initiate treatment. In the last few years, these guidelines have changed, as treating patients earlier leads to better morbidity and mortality outcomes. Furthermore, the bulk of sexual transmission (70 percent) is from the untreated group of individuals who have CD4+ counts of $<200$ parts per $\mu$L [21]. We note here that viral load is usually a better indicator of transmissibility and health status than CD4+ count, and baseline CD4+ counts vary among people significantly, but obtaining this count is the most common information available. Even
large scale treatment programs, such as the ones in Malawi, do not have the technology for obtaining viral load.

Testing is not explicitly a part of our model. As we have mentioned before, it has not been widely utilized until very recently; discussion of the effects and uptake of testing can be found in Chapter 1 of this thesis. Even with testing, asymptomatic individuals will probably not receive treatment, though they will hopefully receive counseling as well as general health care (such as vitamins). The model can be interpreted in a couple of different ways. We can suppose that treating only symptomatic individuals means that they are being treated on the presumption of HIV infection due to the secondary infections characteristic of the disease, or we can assume that testing is part of receiving treatment. This would increase the inherent monetary and human cost of administering medication. We also show the model the case where we treating anyone who is asymptomatic or symptomatic with equal probability. This is the case more likely to represent what might happen if the entire population were tested, or if people were targeted for testing due to previously known behavior.

2.4.1 Equations

To make keeping track of parameters much easier, we will number the compartments as in the figure below; these numbers correspond to the index of the compartment in the population vector. The model is no longer linear, as sexual transmission is a non-linear effect which depends on both the density of the at risk population and the density of the infectious population and is thus not included in the flow chart. Please see Appendix A for detailed information about the parametrization.

Susceptible consumers grow up to become susceptible producers at the rate $\beta$ as before, and infected consumers grow up to be infected producers at the same rate. The transition from asymptomatic to symptomatic is described by $\tau_1$ for children and $\tau_2$ for adults. These rates capture the latency period of HIV. Birth rates are shown on the labeled dashed lines. The rate $\alpha$ is the number of healthy children born per healthy adult each month, as before. For ease of notation, $\alpha_{ij}$ is the rate at which individuals from compartment $i$ give birth to individuals in compartment $j$. For example, $\alpha_{61}$ is the rate at which producers suffering from symptomatic AIDS, and not undergoing treatment, give birth to healthy children. The rate $\gamma_i$ is the death rate from compartment $i$. For example, $\gamma_6$ is the death rate for producers suffering from AIDS. The parameters $\zeta_1$ and $\zeta_2$ are linear rates of disease transmission to consumers and producers, respectively. These rates include various factors that would not be density dependent on the rates of infection within the community, such as needle sticks during doctors visits, or sexual contact with individuals outside of the community.

Finally, we describe two kinds of nonlinear transmission: via sexual and nonsexual contact. We assume that nonsexual contact is the same for both consumers and producers, and we call this rate $\iota_{ns}$. Sexual contact occurs only between adults, and the rate is denoted by $\iota_s$. We
Figure 2.1: Flow chart showing the six compartments and all linear rates of transfer between them. The compartments are defined as follows: $S_c$ are susceptible consumers, $S_p$ are susceptible producers, $I_c$ are infected asymptomatic consumers, $I_p$ are infected asymptomatic producers, $I_{ca}$ are symptomatic consumers, and $I_{pa}$ are symptomatic producers.

Assume that interpersonal contact behavior does not change with infection status. Again, it is very important to measure these rates in a specific community. There is no agreement between studies trying to estimate rates of sexual contact, and it is well documented that these rates depend highly on the religion and customs of a particular community. The time-dependent term $\epsilon_1(t)$ describes the overall transition rate (with both linear and non-linear terms) from $S_c$ to $I_c$, and $\epsilon_2(t)$ is the transition rate from $S_p$ to $I_p$.

The population at time $t$ is now the vector $N(t) = [S_c(t), S_p(t), I_c(t), I_p(t), I_{ca}(t), I_{pa}(t)]$. Define $P(t)$ to be the total population size at time $t$.

The new projection matrix becomes

$$M(t) = \begin{pmatrix}
(1-\gamma_1)(1-\beta)(1-\epsilon_1(t)) & \frac{\alpha}{\beta} & 0 & 0 & 0 & 0 \\
\epsilon_1(t) & (1-\gamma_2)(1-\epsilon_2(t)) & 0 & 0 & 0 & 0 \\
0 & \epsilon_2(t) & (1-\gamma_3)(1-\beta)(1-\tau_1) & 0 & 0 & 0 \\
0 & 0 & \beta & (1-\gamma_4)(1-\tau_2) & 0 & 0 \\
0 & 0 & 0 & \tau_1 & (1-\beta)(1-\gamma_5) & 0 \\
0 & 0 & 0 & 0 & \tau_2 & (1-\gamma_6)
\end{pmatrix}$$

(2.3)
The terms $M_{31}(t) = \epsilon_1(t)$ and $M_{42}(t) = \epsilon_2(t)$ now include non linear elements from disease transmission through interpersonal contact within the community.

\[
\epsilon_1(t) = \zeta_1 + \frac{I_c(t) + I_p(t) + I_{ca}(t) + I_{pa}(t)}{P(t)} \cdot \zeta_{ns} \\
\epsilon_2(t) = \zeta_2 + \epsilon_1(t) + \frac{I_p(t)}{S_p(t) + I_p(t) + I_{pa}(t)} \cdot \zeta_s + \frac{I_{pa}(t)}{S_p(t) + I_p(t) + I_{pa}(t)} \cdot \zeta_a
\]

The population dynamics will look like $N(t+1) = M(t)N(t)$.

The resources available to the community at time $t+1$ will then be

\[
R(t+1) = R(t) + \lambda S_p(t) + \lambda_h I_p(t) + \lambda_a I_{pa}(t) \\
- (S_c(t) + I_c(t) + I_{ca}(t))\eta_c - (S_p(t) + I_p(t) + I_{pa}(t))\eta_p
\]  

2.5 Disease, Community provision of treatment

Now that we have built a model framework in which to study disease dynamics in the absence of available treatment, we can introduce the idea of antiretroviral therapy into the model. Our goal will be to study resource allocation strategies, which will dictate how to choose individuals to treat. Since it may often be impossible to provide medication to all of the infected individuals, it is important to understand the most efficient way to administer available doses. Our goal will be to measure the effect of the allocation strategy on the ration of producers to consumers.

We will first consider the scenario in which ARVs are being provided by an agent outside of the community, such as a local NGO, or local government HIV intervention program. We discuss some examples of these in Chapter 1.

Antiretroviral therapy affects our model in multiple ways. An individual being treated will not suffer from the symptoms of AIDS as much as someone who is not; he or she will have a productivity $\lambda_t$ that is between that of a healthy producer and one who is suffering from full-blown AIDS. Thus, treating producers will benefit the economic health of the community. The epidemiological effect is that individuals have less of the virus circulating in their blood stream, and are thus much less likely to spread the infection through interpersonal contact. We will think of ARV treatment as a protective factor, reducing the transmissibility of infection from the treated individual by a certain rate. $p$ will be the reduction in rate of sexual transmissibility in an individual in compartment $I_{pa}$ who receives treatment.

2.5.1 How the resource allocation portion of the model works

We will consider two different types of resource allocation strategies. The first will be to treat a fixed fraction of the population suffering from AIDS. This allows us to describe our results analytically a little better. The second will be to assume that at every time interval, a fixed number of doses are provided to the community from an outside source, and the allocation
strategy will be to determine which fraction of those doses are used to treat consumers as opposed to producers.

2.5.1.1 First type of strategy

Let $\mu_c$ be the fraction of $I_{ca}$ that are treated with antiretroviral therapy at every time step, and let $\mu_p$ be the fraction of $I_{pa}$ that are treated. A resource allocation strategy has these two rates fixed.

The linear portion of the model is affected only in the rates of healthy and sick children born to $I_{pa}$ individuals, and death rates of $I_{ca}$ and $I_{pa}$. We called $p$ the protective factor due to ARV against sexual transmission, so we will call $p_c$ the protective factor of ARV against vertical transmission. $l_1$ and $l_2$ are the reductions in death rate (increase in life expectancy) for children and adults suffering from AIDS who receive ARV, respectively.

The new population projection matrix becomes

$$M(t) =
\begin{pmatrix}
(1-\gamma_1)(1-\beta)(1-\epsilon_1(t)) & \frac{\alpha}{\beta} & (1-\gamma_2)(1-\beta)(1-\epsilon_2(t)) & 0 & \alpha_{41} & 0 & \alpha_{61} \\
\epsilon_1(t) & 0 & (1-\gamma_3)(1-\beta)(1-\tau) & 0 & 0 & \alpha_{43} & 0 \\
0 & \epsilon_2(t) & (1-\gamma_1)(1-\tau) & \beta & 0 & 0 & \alpha_{63} \\
0 & 0 & \tau & 0 & \tau & 0 & 0 \\
0 & 0 & 0 & 0 & \tau & 0 & 0 \\
\end{pmatrix}
$$

The terms $M_{31}(t) = \epsilon_1(t)$ and $M_{42}(t) = \epsilon_2(t)$ now include non linear elements from disease transmission through interpersonal contact within the community.

$$
\epsilon_1(t) = \zeta_1 + \frac{I_c(t) + I_p(t) + (1 - \mu_c + \mu_c p)I_{ca}(t) + (1 - \mu_p + \mu_p p)I_{pa}(t)}{P(t)} \cdot \nu_{ns} \\
\epsilon_2(t) = \zeta_2 + \epsilon_1(t) + \frac{I_p(t)}{S_p(t) + I_p(t) + I_{pa}(t)} \cdot \nu_s + \frac{(1 - \mu_p + \mu_p p)I_{pa}(t)}{S_p(t) + I_p(t) + I_{pa}(t)} \cdot \nu_a
$$

The resources available to the community at time $t + 1$ will then be

$$
R(t + 1) = R(t) + \lambda S_p(t) + \lambda h I_p(t) + (\mu_p \lambda tr + (1 - \mu_p) \lambda a) I_{pa}(t) \\
- (S_c(t) + I_c(t) + I_{ca}(t)) \eta_c - (S_p(t) + I_p(t) + I_{pa}(t)) \eta_p
$$

2.5.1.2 Second type of strategy

Let $D$ be the number of doses provide to the community at each time interval. Then let $\delta_1$ be the fraction of those doses administered to $I_{ca}$ and $\delta_2$ be the fraction of those doses administered to $I_{pa}$. Setting $\delta_1$ and $\delta_2$ will be our resource allocation strategy.

Let $\mu_c$ and $\mu_p$ be as before, the fraction of the respective subpopulation suffering from AIDS that receive treatment. The difference is that now, these fractions must be recalculated at every
time step as the population size varies.

The simulation we use to study these strategies runs iteratively, so the equations for each time step will be identical to those for the previous strategy, where we fix $\mu_c$ and $\mu_p$. The only change we make is to set, before we calculate the population distribution at $t + 1$,

\begin{align*}
\mu_c &= \frac{\delta_1 D}{N_5(t)} \\
\mu_p &= \frac{\delta_2 D}{N_6(t)}
\end{align*}

2.6 Disease, incremental investment in treatment

Finally, we want to answer the question whether a community might be able to provide treatment for itself. Here we will introduce a price per dose of medication, and the number of dosages purchased will depend on the resources available after basic consumption needs from the previous time step.

This introduces a feedback into the model, since as infection rates increase, the community is less able to purchase medicines and thus causing infection rates to increase further.

Implementing the simulation to study this case will be almost the same as for the case where we have disease, free treatment, and implement the second allocation strategy. The one change that we make is that $D$, the number of doses, is no longer fixed in time. Define $\psi$ to be the cost of a single dose of medication for one time unit. Assume that all excess resources that are available at time $t$. Then $D(t) = \frac{R(t)}{\psi}$
3. Simulation Results and Discussion

Please see Appendix A for many details on parametrization.

3.1 No Disease

First, we will look at the model in the absence of disease to have a baseline comparison for the population dynamics. Notice that this is not a model of what the population would really look like today in the absence of HIV, due to the many confounding factors. Economic development would have probably lowered birth rates, as well as death rates. There have been many projections using much better models than ours for this purpose, so we will not attempt to predict any scenarios like this. This simulation uses the parameters for birth and death used in the rest of the model, strictly for comparison purposes. That is $\alpha = 0.047$, $\gamma_1 = 0.0367$, $\gamma_2 = 0.0105$.

We see a 1.57 percent overall annual population growth. This is not unreasonable for this area of the world. It is important to note that, in the long term, this simulation yields a stable population distribution with approximately 28.25 percent children. This is a little high for a developed country, but is definitely a reasonable population distribution. This population growth rate is more sustainable than what we see in the area currently; in some regions the population growth rate overall is closer to 2.5 percent.

3.2 HIV spread in the absence of treatment

For another baseline comparison, we will look at a projection of the epidemic in the absence of ARV. To make sure the effects we are seeing are from the introduction of disease and not from the population naturally tending toward the stable distribution, we use initial conditions close to the stable distribution we find without disease, except we make some of our producers infected. The exact initial population distribution used in the simulations is 37 susceptible consumers, 83 susceptible producers, and 10 infected asymptomatic producers.
For the analysis in this paper, to be very conservative with the simulation, we set $\zeta_1 = 0$ and $\zeta_2 = 0.005$. These are linear rates of disease transmission, so they play an enormous role in the disease dynamics. It would be impossible to disregard these rates, but the actual values are highly dependent on the circumstances of a particular community. Linear rates of transmission to both children and adults might come from immigration from other communities, shared needles, and various traditional practices, such as widow cleansing and virgin rape. Sexual contact with people outside of the community would be considered a linear rate, since the outside community is effectively infinite. If this model were to be applied to analyze resource allocation strategies for a particular community, it would be extremely important to have information specific to that group of people.

We run the simulation for thirty years, or 360 months. One of the most striking things we see by playing with the parameters is that the spread of the disease is overwhelmingly determined by the linear rates, more so than we expected when building the model. HIV is not transmitted very easily through sexual contact, so it is not very surprising that communities with extremely high infection rates must have other common routes of transmission. We see a sharp decrease in able-bodied producers in the community- the numbers of consumers and able bodied producers become nearly equivalent. The overall fraction of the population that are consumers rises as the epidemic spreads, from 28 percent when infection is introduced, to 36 percent when infection levels rise to 31 percent. While this change may not seem very dramatic, it is important to realize that there is a double effect on the community- the addition of consumers that need to be cared for, with the simultaneous loss of producers and the even greater loss of healthy producers. The economic model is very effective at capturing this balance, and we will use the resources available from here onward to summarize this balance.
3.3 Disease, community provision of treatment

Now we will run the simulation with the inclusion of disease, and the possibility of providing antiretroviral treatment to those suffering from AIDS.

3.3.1 General Allocation Strategy

There are a number of realistic scenarios to explore with how treatment might be made available to the population. Below we will explore the different impacts of resource allocation strategies when treatment is only given to those in the advanced stage of disease as well as when ART
is provided to anyone infected. The latter scenario would necessitate some kind of large scale testing program; this may become available in the near future.

Another distinction we make is in describing resource allocation strategies which treat a percent of a subpopulation. This helps us understand which portion of the population it is most important to target, and the degree to which we can allow error. Do we really have to treat all of the producers? Or is it enough to treat 90 percent of them? As it turns out, the model is not very sensitive to such changes.

3.3.1.1 Treating only symptomatic individuals

In the most natural version of our model, only those individuals who are suffering from the symptoms of HIV would have the opportunity to get treatment. As mentioned previously, the treatment guidelines in many countries (including, until recently, the WHO guidelines), are to consider individuals eligible for treatment when their CD4+ count falls below 250. In many studies we surveyed, the average CD4+ count at which treatment was initiated was around 200. We use our simulation to compare the following four allocation strategies:

1. Treating no one. This will look exactly like the case where HIV spreads without treatment intervention, as above.

2. Treating all of the consumers, and none of the producers. We think of this as $\mu_c = 1$ and $\mu_p = 0$.

3. Treating all of the producers and none of the consumers. We think of this as $\mu_c = 0$ and $\mu_p = 1$.

4. Treating the entire population of symptomatic individuals. We think of this as $\mu_c = 1$ and $\mu_p = 1$.

The epidemiological impact of various allocation strategies is shown in Figure 3.3. Treating producers preferentially leads to lower prevalence rates, greater population growth, and a higher proportion of producers in the population. The impact of treating only consumers is practically indistinguishable from the impact of treating no one, and the impact of treating only producers is indistinguishable from the impact of treating the entire population. From this, we conclude that treating as many producers as possible is the most efficient use of ARV.

We also see that only treating symptomatic individuals does not have an enormous effect in controlling the spread of the disease. The effect is measurable: over the course of thirty years, the prevalence might only rise to 25%, as opposed to 30%, but this is not as dramatic an improvement as one might hope from such an huge investment. The reasons for this seem to lie in the fact that these individuals are not the main cause of the infection spreading. Even though they are highly infectious, they have very high mortality rates, and they form a relatively small part of the population.
On the other hand, the effect on overall mortality and population growth is very significant. The strategy of treating all symptomatic producers yields almost a 20% increase in the population after 30 years over the scenario where HIV spreads unchecked. If we treat only half of the symptomatic producers, the population falls evenly between the curve of no treatment, and treating all symptomatic individuals.

The payoff of this investment becomes even more clear when we look at how the economic well-being of the community is affected, below.

Figure 3.4 shows the impact of varying resource allocation strategies on the resources generated by the community at every time step. As the disease spreads, the number of able-bodied producers in the population falls, and the proportion of the population that is made up of consumers rises, leading to depleted savings.

While the numbers in the graphs seem somewhat large and positive, one must consider that it actually means the community would be saving something like $200 per month in the absence of HIV. In a community of over 100 people, yielding only a couple of dollars in savings per family. This number depends completely on our choice of parameters but the magnitude of the effect of the allocation strategies comes from parameters that we have found in the research literature.

In summary, the positive effect of treating producers is marked. The more producers we treat, the greater the resources available to the community. Treating consumers makes no measurable difference to the economic health of the community. Treating all producers doubles the savings of the community per month over the long run.

### 3.3.1.2 Treating all infected

After using our model to analyze the impact of various resource allocation strategies that only treat symptomatic individuals, we see that the impact of this is not as great as we would like. The reasons for this include the high death rates of symptomatic individuals, as well as the fact that HIV is not actually a very efficient sexually transmitted disease. Knowing more about the modes of transmission in a particular community would be very important to assess the magnitude of impact of different strategies.

A natural follow-up would be to ask about the impact of treating both symptomatic and asymptomatic individuals. If our strategy consists of treating a fraction of all infected, this might correspond to the scenario where a fraction of the population is willing to submit to testing, so that these individuals are identified.

We tested the impact of three different allocation strategies: treating all infected consumers, treating half of the infected consumers and half of the infected producers, and treating all of the infected producers. The resulting population distributions were very similar to those in the scenario where we treated only symptomatic individuals, and the overall rates of HIV prevalence were indistinguishable. There is approximately a 10% increase in overall population size in the long term, due to the increased survivorship when individuals are treated earlier.

This result would be different depending on how infectious individuals in the earlier stages of disease were found to be. These numbers are particularly uncertain and difficult to find.
in the literature- it is very possible we have underestimated them. Furthermore, our model
does not include the fact that individuals who get treatment early enough may never become
symptomatic. In other words, treating individuals in compartments \( I_c \) and \( I_p \) will affect the
latency period \( \tau_1 \) and \( \tau_2 \). We have no information about these parameters- but this inclusion
makes an enormous difference when we enter some toy values. We cannot emphasize enough
how important including this information is. In the best case scenario, where ARV treatment
stops the progression of disease altogether, treating all infected individuals can completely restore
population growth.

While the epidemiological impact of treating all infected individuals was not as big as we
hoped, the economic (and thus social) benefit is very significant.

### 3.3.2 Fixed Dosage Available

In the following two cases we suppose that 10 doses of ART are provided at the beginning of
each month. The allocation strategy is given by \( \delta_c \), which tells us the fraction of those doses
which will go to consumers, and \( \delta_p \), which tells us the fraction that will go to producers. There
are a few options pertaining to what to do with excess doses, but we will assume that they are
not used. Given the numbers we have seen in the epidemiological model, this will be enough to
treat everyone at first and then become insufficient.

First we only consider treating those individuals who are suffering from the advanced stages
of disease- \( I_{pa}, I_{ca} \). The population dynamics here do not differ greatly from our previous
approaches, so we will not include them. On the other hand, the economic equations are much
more sensitive to changes in the allocation strategy.

The graph on the left shows the scenario when we treat only symptomatic individuals, and
the graph on the right compares strategies that involve treating any infected individual with
the same probability as any other. Looking at the first scenario, we see an interesting pattern.
For the first couple of years, the strategies of giving half of the doses to consumers and half to
producers and giving all doses to the producers look identical. This is because there are not so many infected

### 3.4 Disease, incremental investment in treatment

This final scenario is probably the most interesting and important. The effect we wish to capture
here is the feedback in the case when a community might be buying ARV treatment incrementally
(every month). The number of doses that the community is able to purchase depends on the
resources available; as the prevalence of HIV increases, the community will be less able to purchase
treatment.

We will continue to show graphs of resources remaining each month, now with the additional
impact of medical expenditures. While there is still a surplus (the community can afford to
follow the given allocation strategy), this is an accurate reflection of the model. When the price
of treatment rises high enough that the community cannot afford to follow the given strategy using surplus generated that month, this may not represent the community actions any longer. It seems unlikely that surplus money would remain untouched in a subsistence community to significantly affect the long term outcome of treatment strategies.

The first step in the simulation is to calculate the excess resources available in the current time step based on the number of healthy and sick and treated and untreated producers from the previous time step and then subtract the baseline consumption based on the current numbers of consumers and producers. The remaining resources are considered available for purchasing medication. Given a price per dose of medication, we then calculate how many individuals the community could potentially treat. Then the community follows the resource allocation strategy and purchases the doses necessary. If the strategy dictates that all doses are given the symptomatic consumers, only enough doses to do this are purchased. This way, not all of the community’s resources are expended.

Figure 3.8 shows the outcome of three different allocation strategies that describe how a community might expend its resources on treatment. The second two strategies: splitting purchased medication evenly between symptomatic producers and symptomatic consumers, and giving all of the purchased doses to symptomatic producers, look identical. We compare those two strategies to one which consists of giving all purchased doses to symptomatic consumers. From the epidemiological point of view, the two strategies which preferentially treat producers are more successful- prevalence rates are lower, and population growth rate is higher. From an economics standpoint, we see that these strategies are also beneficial. What we see is that when more sick producers are treated, more medication is purchased (and thus more money is spent), but the population is simultaneously more productive. In the long term, the community has more resources remaining in excess every month if symptomatic adults are targeted. Thus, spending more money on treatment, when it is preferentially used to treat symptomatic adults, is still the most economically beneficial strategy.

The reason we cannot distinguish between the two top strategies which consist of giving all doses to producers or half of the doses to producers is that, with the parameter values we have chosen, the community is able to treat all of the symptomatic producers in both cases. One can see immediately that the cost of treatment is a determining factor.

Figure 3.9 shows the comparison of the same three allocation strategies as before, but now any infected individual is equally likely to receive treatment. The first is to give all doses to infected consumers, the second is to give half of the doses purchased to infected consumers and half to infected producers, and the third is to give all purchased doses to infected producers. In comparison to only treating symptomatic individuals, these strategies will lead to a greater expenditure.

Figure 3.9a shows the resources remaining each month after each allocation strategy is followed. The first strategy allows all of the infected consumers to be treated. As one can see in figure 3.9b, there are very few infected consumers, so this does not significantly deplete the savings of the community. The resulting curve is nearly identical to that which results when the
community does not pay for treatment and all infected consumers are treated, as in figure 3.4.

The second strategy calculates the number of doses which could be purchased using the resources available. Half of those doses are used to treat any infected consumers and half are used to treat any infected producers. If there are not enough sick consumers to necessitate half of the doses, the excess is not purchased. This is why the second and third strategies diverge. For approximately the first five years of the epidemic, half of the doses that can be purchased is a sufficient number to treat all of the infected producers. After five years, the number of infected producers increases at a higher rate than the number of infected consumers. The result is that the last strategy, purchasing as many doses as possible to target treatment of any infected producers, leads to higher expenditures for the community.

To see whether the greater economic investment in treating producers preferentially makes sense, we must look at figure 3.9b. What we see is the same as before- the more producers receive treatment, the more successful the strategy is as increasing population growth and slowing the spread of disease. The improvement in survivorship is very significant and primarily stems from the increased numbers of susceptible producers. While the community savings are depleted somewhat, this strategy treats many more individuals.

Figure 3.10 shows an example of the outcome when the price of treatment rises such that all resources are exhausted trying to follow the allocation strategy.

The differences can be seen most dramatically in the case where the strategy is to treat all of the infected producers. At the higher price per dose, the community exhausts all of its resources trying to do this. This strategy is still the most successful of the three at preventing disease, but the gap is survivorship is more modest. If this is the case, the community might have to weigh its immediate economic needs against the amount invested in treatment. The cost of treatment becomes too high to offset the gains in productivity.

### 3.4.1 Purchasing all possible doses each month

In the cases above, we considered what would happen if the community only purchases the number of doses necessary to follow the allocation strategy. This allows the community to retain some savings, if the medicine prices are low enough. To illustrate, consider the case where the strategy dictates that all doses that can be purchased are given to infected consumers. For the prices we considered, the community is able to purchase more doses than there are infected consumers, and we show what would happen if they chose not to purchase the excess doses and distribute them to the other infected people.

In this section, we will consider a different type of decision making. Now, the community will calculate how many doses it is able to purchase using the excess resources produced that month (what is left after basic consumption), and then purchase all of the doses necessary according to the allocation strategy. If this number is less than the number that could have been purchased, then the community will also purchase as many excess doses as it can to treat any remaining infected people.
We only show two strategies here, because the number of infected consumers remains small, and the community is usually able to treat all of them. Any strategy that involves giving a fraction of the doses purchased to consumers and a fraction to the producers, with the excess going to producers will look nearly identical to the case where we preferentially treat consumers and give all excess to producers.
Figure 3.3: Population dynamics in the presence of different allocation strategies, only symptomatic individuals treated. Treating producers preferentially leads to lower prevalence rates, greater population growth, and a higher proportion of producers in the population.
Figure 3.4: Resources produced by community each month during HIV epidemic. Comparing various allocation strategies, treating only symptomatic individuals. The most successful strategies are treating all symptomatic producers or treating all symptomatic individuals—these two cannot be distinguished. After that, the strategies shown in decreasing order are treating 80%, 60%, 40% and 20% of producers and no consumers. The least successful strategies which cannot be distinguished are treating no one or treating only symptomatic consumers.
Figure 3.5: Population dynamics in with different allocation strategies, treating any infected person. The most successful is treating all infected producers, the second is treating half of the infected consumers and half of the infected producers, and the least successful is treating all of the infected consumers.
Figure 3.6: Resources produced by community each month during HIV epidemic. Comparing various allocation strategies, treating any infected individual with equal likelihood. The strategies are the same as before, in 3.4.
Figure 3.7: Resources in community when fixed number of doses is available at every time interval.

(a) Resources when fixed number of doses (10) is provided, vary allocation strategies

(b) Resources when fixed number of doses is provided, and given to all infected individuals. Varying allocation strategies
(a) Resources remaining after community purchases treatment according to resource allocation strategy.

(b) Population demographics under different resource allocation strategies.

Figure 3.8: Scenario where the community purchases treatment incrementally with available resources each month. We first calculate the number of doses which could be purchased, and then calculate the expenditure based on the fixed allocation strategy. This figure shows the outcomes of three different allocation strategies: all purchased doses of medicine are given to symptomatic consumers, half of the doses are given to each symptomatic group, and all of the purchased doses are given to symptomatic producers. In both the economics and epidemiology, the second two strategies are indistinguishable. The least successful strategy at controlling disease is to treat all symptomatic consumers and no producers.
Figure 3.9: Scenario where the community purchases treatment incrementally with available resources. This figure shows the outcomes of three different allocation strategies: all purchased doses of medicine are given to infected consumers, half of the doses are given to each infected group, and all of the purchased doses are given to infected producers. Symptomatic and asymptomatic infected individuals are equally likely to receive treatment.
Figure 3.10: Community purchases treatment incrementally, treats any infected individual. More expensive treatment.
Figure 3.11: Figure shows epidemiology and economics of community in the case where as many individuals receive treatment as the community can afford that month. In one scenario, all infected consumers are treated first, and then excess doses are given to infected producers. In the second, all infected producers are treated first, and any excess doses are given to infected consumers. Treating producers preferentially leads to a slightly better epidemiological outcome.
Chapter 4

Mathematical Analysis of Model

4.1 Steady State Analysis

We can analyze the long term behavior of our model by asking whether there is an endemic equilibrium, i.e. is there a stable stage distribution where a constant fraction of the population is infected? We look for this by taking all of our differential equations and setting them equal to zero (since the rate of change term is only on one side of the equation), and then use MATLAB to solve the system of six non-linear equations. The only solution is where the population is zero.

4.2 Sensitivity Analysis

In this chapter we will analyze the robustness of the model. In any epidemiological study, there is a margin of error in the parametrization, and it is very important to understand how sensitive the results are to error.

4.2.1 No Disease

The dominant eigenvalue $\rho$ of the projection matrix $M$ gives us the growth rate of the population. The eigenvector $v_\rho$, which corresponds to this eigenvalue, gives the distribution of the population at steady state. The Perron-Frobenious Theorem guarantees for us the existence of a greatest positive eigenvalue and a positive eigenvector which corresponds to it. At steady state now we know the proportion of producers and consumers that remains constant.

We can study the sensitivity of the growth rate to various parameters. We have the well-known sensitivity formula

$$\frac{\delta \rho}{\delta m_i} = \frac{v_i * w_j}{\langle v, w \rangle}$$

where $v, w$ are the left and right eigenvectors corresponding of $M$ corresponding to $\rho$. 
4.2.2 How does model look with our parameter values?

With the parameter values we have chosen, we find that the population grows at a rate $\rho = 1.0013$ monthly and the long term population distribution is $[S_c = 36.7, S_p = 93.01]$ with a ratio of 28% consumers and 72% producers.

4.2.3 Numerical Conclusions

The sensitivity matrix of how $\rho$ responds to changes in the entries of $M$ turns out to be

\[
\begin{pmatrix}
.1812 & .4587 \\
.3234 & .8188 \\
\end{pmatrix}
\]

, for the given parameter values.

The chain rules then gives us that the calculated sensitivity to $\gamma_1$ is $\frac{\delta \rho}{\delta \gamma_1} = -0.2617$, and to $\gamma_2$ is $\frac{\delta \rho}{\delta \gamma_2} = -0.7367$.

Figure 4.1: Plot of annual population growth rate against annual death rate

Figure 4.1 shows the annual population growth rate which would result from each possible death rate. We explore the effects of varying both child and adult death rates.

Figure 4.2 shows the response of the growth rate to changes in death rates. This is the sensitivity we are most interested in, since death rates are what will be most affected as HIV invades the population.
3.6

Figure 4.2: Plot of sensitivity of growth rate to fluctuations in death rate at each given death rate

4.2.4 Model without treatment

The model that includes HIV had non-linear elements, but we perform matrix calculus analysis on the linear portion of the model, without sexual transmission terms. This gives partial information, but it is relevant since we see that, with our chosen parameter values, transmission through interpersonal contact is not what drives the spread of HIV.

With the parameter values we have used, we find that the population without transmission through interpersonal contact would have a monthly population growth rate of $\rho = .9985$, which amounts to an approximate 2% population decrease per year. The long term population distribution is $[S_c, S_p, I_c, I_p, I_{ca}, I_{pa}]$ with each comprising the following percentages of the population $[33.67, 42.78, 0.42, 15.27, 0.27, 7.59]$ in the long term stable distribution.

The chain rule gives the sensitivity of $\rho$ to perturbations in $\gamma_1$ as $\frac{\partial \rho}{\partial \gamma_1} = \frac{\partial \rho}{\partial M_{11}} \cdot \frac{\delta M_{11}}{\delta \gamma_1} = \frac{\delta \rho}{\delta M_{11}} (\zeta_1 - 1)(1 - \beta)$

Similarly, the sensitivity of $\rho$ to $\gamma_2$ is given by $\frac{\partial \rho}{\partial \gamma_2} = \frac{\delta \rho}{\delta M_{22}} \cdot \frac{\delta M_{22}}{\delta \gamma_2} = \frac{\delta \rho}{\delta M_{22}} (\zeta_2 - 1)$
The sensitivity matrix of how \( \rho \) responds to changes in the entries of \( M \) turns out to be

\[
\begin{pmatrix}
0.3567 & 0.4532 & 0.0044 & 0.1617 & 0.0028 & 0.0804 \\
0.4567 & 0.5802 & 0.0057 & 0.2070 & 0.0036 & 0.1029 \\
0.0205 & 0.0260 & 0.0003 & 0.0093 & 0.0002 & 0.0046 \\
0.1219 & 0.1549 & 0.0015 & 0.0553 & 0.0010 & 0.0275 \\
0.0041 & 0.0052 & 0.0001 & 0.0018 & 0.0000 & 0.0009 \\
0.0332 & 0.0422 & 0.0004 & 0.0151 & 0.0003 & 0.0075 \\
\end{pmatrix}
\]

for the given parameter values. The chain rules then gives us that the calculated sensitivity to \( \gamma_1 \) is \( \frac{\delta \rho}{\delta \gamma_1} = 0.3567 * (-1) * (0.994) = -0.3547 \), and to \( \gamma_2 \) is \( \frac{\delta \rho}{\delta \gamma_2} = 0.5802 * 0.995 = 0.577 \), when these parameters are very close to the ones used to create our simulations.

Finally, we want to look at how the model depends on the latency period of the disease. The chain rule tells us that the sensitivity of the population growth rate to the rate at which asymptomatic infected consumers become symptomatic consumers is given by \( \frac{\delta \rho}{\delta \tau_1} = 1 \cdot \frac{\delta \rho}{\delta M_{35}} - (1 - \gamma_3)(1 - beta) \cdot \frac{\delta \rho}{\delta M_{45}}. \) The sensitivity of the population growth rate to the rate at which asymptomatic infected producers become symptomatic is given by \( \frac{\delta \rho}{\delta \tau_2} = 1 \cdot \frac{\delta \rho}{\delta M_{45}} - (1 - \gamma_4) \cdot \frac{\delta \rho}{\delta M_{44}}. \)

For values near the ones used in the simulation, \( \frac{\delta \rho}{\delta \tau_1} = -2.0183e-04 \) and \( \frac{\delta \rho}{\delta \tau_2} = -0.0401. \) Figure 4.5 shows the sensitivity to every possible such rate, while holding the other value fixed at the one used in simulations. Figure 4.4 just shows the population growth rate which would result from each set of parameters \( \tau_1, \tau_2. \)
We vary the latency period (time for disease to progress from asymptomatic to symptomatic) for adults and children individually, while holding the other value fixed at the value used in the model.

4.2.5 Model with treatment by percent

We will only perform sensitivity analysis on the linear portion of the model with treatment by percent (the first type of strategy), and we will do so analogously to the above.

4.2.5.1 Treating only symptomatic

We will show how growth rates change as we vary $\mu_c, \mu_p$, one at a time. In this case, these are fractions of infected symptomatic consumers and producers which will be treated at each time step. Since we only look at the linear portion of the model, we only see the effects of treatment on vertical transmission and death rates in this analysis. The sensitivity of the population growth rate to perturbations in the treatment strategy is given by $\frac{\delta \rho}{\delta \mu_c} = (1 - \beta)(\gamma_5)/(1 - l_1) \cdot \frac{\delta \rho}{\delta M_5}$.

The sensitivity of the population growth rate to perturbations in the treatment of symptomatic producers is given by $\frac{\delta \rho}{\delta \mu_p} = \gamma_6(1 - l_2) \cdot \frac{\delta \rho}{\delta M_6}$. 

Figure 4.4: We vary the latency period (time for disease to progress from asymptomatic to symptomatic) for adults and children individually, while holding the other value fixed at the value used in the model.
Figure 4.5: We vary each death rate between 0 and 1 and look at the sensitivity of the growth rate to perturbations around each value. The graph shows the log of the sensitivity.

Figure 4.6
Figure 4.7

Sensitivity of growth rate to perturbations in treatment allocation strategies

Log of annual population growth rate vs. Percent of symptomatic individuals treated

- Graph showing the relationship between the log of the annual population growth rate and the percentage of symptomatic individuals treated, with two lines representing different scenarios: $\mu_c$ varies, $\mu_p = 1$ (blue line) and $\mu_p$ varies, $mu_c = 1$ (green line).
Chapter 5

Conclusions

We have built a model to study HIV transmission in a small community. We model HIV as a disease with two stages basic stages and without any possibility of recovery or immunity. We also separate the population into two age cohorts, which correspond to our economic model as we.

We use average parameters from a literature search of studies conducted in a number of different regions in Sub-Saharan Africa. This represents the type of community we believe has the greatest need to implement efficient resource allocation strategies.

5.1 Summary of Results

We first demonstrate that our model and parameter choices lead to a reasonable representation of how the HIV epidemic has developed. We run the simulation over thirty years, and see the prevalence rise from approximately 8% to 31% without any intervention. We show that the spread of HIV affects the demography of the population; the fraction of the population that consumers make up increases from 28% to 36% with increasing prevalence.

When we introduce the possibility of ARV treatment, we explore a number of different scenarios. In the first, we assume that only symptomatic individuals will have the opportunity to receive treatment. This is aligned with the current treatment guidelines in most countries in SSA.

An additional consequence of our model is that it shows that sexual transmission cannot be the only mode by which HIV is spreading in Sub-Saharan Africa. We even suggest it cannot be the primary mode of disease spread. As a result of this, treating individuals within a small community with ARVs actually has a limited effect on decreasing prevalence, but it greatly increases life spans and productivity of the community. It also lowers the percent of the community comprised of children under fifteen, which improves the economic and social stability.

Administering ARVs to individuals who are infected (assuming knowledge of seropositivity) is more effective at preventing the spread of disease, but not as much as we expected. It has a very
significant effect on the economic well-being of the community.

In every scenario that we explore, strategies that involve focusing on treating producers leads to better epidemiological and economic outcomes.

5.2 Falsifiable assumptions of the model

As with any mathematical model, there are many simplifying assumptions made. I would like to highlight a few here that have the potential to truly affect the results. It would be extremely important to look at these assumptions closely before applying our model to a particular community.

We assume very good adherence to treatment regimens: that each individual in the community who receives treatment will take the medication as he or she is supposed to, and also that more or less the same individuals are treated at each time interval. Poor adherence to treatment regimens can lead to the evolution of drug resistance in the virus [5]. Furthermore, individuals who do not take their medication consistently or correctly will not experience the health and prevention benefits afforded by ART. Our parameter estimation for these protective rates came primarily from studies conducted in hospitals or other very good treatment programs. If a community does not have adequately trained public health workers, these rates may be overly optimistic. And finally, the impact of administering treatment to the community would not have an immediate effect on productivity. Individuals need to adhere to a course of treatment for some time before they regain their health measurably; it could take as long as three to six years after initiating therapy to achieve the gains in productivity that we assume. Since many of those individuals might not survive that length time, this is not just a lag in the resource equation behind the population dynamics.

We also assume that demographic parameters such as birth and death rates do not change over time. But over the course of 30 years, this is not true for many countries. For instance, in South Africa, the death rate has nearly doubled since 1985, but the birth rate has fallen significantly. I do not believe there is any accurate way to model this phenomenon if trying to plan for the future, but it is worth considering when interpreting the results if planning the intervention strategy for a specific community. The model is probably more accurate on a shorter time span.

The impact of the assumption that older people are producers is not clear. It is very possible that these individuals should be represented in their own cohort as a different kind of consumer. If more were known about a particular community, it may be useful to expand the model into one with age cohorts of approximately 5 years. This would be quite simple to do with our model construction. It would also help to capture the highly specific effects of infant mortality.
5.3 Issues for further study

One of the issues that we did not consider in our model is the effect of administering ART on the latency period an individual might experience. Failing to include this makes our model conclusions very conservative, which we believe is appropriate given that we are trying to draw broad trends. Including the effect that ARVs may increase the latency period would dramatically increase survivorship.

The economic model we use is extremely simplified. We think this is appropriate to capture how disease affects the proportions of able bodied producers in the community, but there are a few choices that could be made. For instance, it could be argued that the consumption rate of sick individuals would increase, because they need more care.

Another possible expansion of the model would be to consider men and women as separate cohorts. This might yield interesting results because men and women have a different impact on the spread of HIV, since only women give birth. The rates of transmission are also somewhat asymmetrical, and gender differences vary by age cohort. In some countries, men have higher overall prevalence rates, but young women ages 15-19 have much higher prevalence rates than young men. This is particularly disturbing since these are young women with high fertility. Another reason to separate these subpopulations is that men and women often have very different economic roles. Men tend to have higher employment rates, and there are many gender-biases in the type of job held. This will cause differences in how HIV impacts their ability to work. Information about a particular community’s social structure would be crucial when trying to capture the true impact.
We normalize all parameters to be rate per month, since a month is the time projection interval we have chosen. For ease of reading, they are left as annual rates here, while in the simulation, they are divided by 12.

For use in the model, we have to use data from the field to estimate parameters, because it is not possible to get exact values. Furthermore, we are not modeling one particular community, but an approximation of what we think is a good representation of the communities we believe would most need to rely on resource allocation strategies.

We are confident relying on data from the field, because we believe any errors will not be of an order of magnitude to affect our results.

<table>
<thead>
<tr>
<th>Country</th>
<th>Annual birth rate per 1,000 people (2011)</th>
<th>Annual death rate per 1,000 people</th>
<th>Infant mortality per 1,000 live births</th>
<th>Life Expectancy at birth in years</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Africa</td>
<td>19.32</td>
<td>17.23</td>
<td>42.67</td>
<td>49.41</td>
</tr>
<tr>
<td>Namibia</td>
<td>21.11</td>
<td>13.09</td>
<td>45.61</td>
<td>52.17</td>
</tr>
<tr>
<td>Zimbabwe</td>
<td>32.19</td>
<td>12.38</td>
<td>28.23</td>
<td>51.82</td>
</tr>
<tr>
<td>Tanzania</td>
<td>37.7</td>
<td>8.6</td>
<td>46.5</td>
<td>53.14</td>
</tr>
<tr>
<td>Mozambique</td>
<td>39.34</td>
<td>12.79</td>
<td>76.85</td>
<td>52.02</td>
</tr>
<tr>
<td>Kenya</td>
<td>31.93</td>
<td>7.26</td>
<td>43.61</td>
<td>63.07</td>
</tr>
<tr>
<td>Congo</td>
<td>40.09</td>
<td>11.25</td>
<td>74.22</td>
<td>55.27</td>
</tr>
<tr>
<td>Zambia</td>
<td>43.1</td>
<td>13.4</td>
<td>70.6</td>
<td>52.57</td>
</tr>
<tr>
<td>Malawi</td>
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<td>12.84</td>
<td>79.02</td>
<td>52.31</td>
</tr>
<tr>
<td>Botswana</td>
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<td>12</td>
<td>10.49</td>
<td>55.74</td>
</tr>
<tr>
<td><strong>AVERAGE</strong></td>
<td><strong>32.72</strong></td>
<td><strong>12.08</strong></td>
<td><strong>51.78</strong></td>
<td><strong>53.75</strong></td>
</tr>
</tbody>
</table>

Figure A.1: This table contains the estimated birth and death rates, as well as the life expectancy at birth of an individual in the country in 2011. Source is CIA World Factbook.
A.0.1 Demographic Parameters

- $\alpha$ is the birth rate per adult. The table above gives a crude birth rate per person, but this depends on age structure of population. Since the age structure changes during our simulation, it does not make sense to use this number as the birth rate. Instead, we use the following information:

  Figure A.2: This table contains the estimated female fertility, under the assumption that each woman lived to her full life expectancy at birth and taking into account fertility at different ages. Source is CIA World Factbook.

  We will assume that the population in each country is approximately fifty percent women; it is not far from this in any country. The average fertility rate is 4.2 per adult woman, so half of that gives 2.1 children born per adult person per their adult life. If we assume a healthy (no AIDS) adulthood to be approximately 45 years, this yields $\alpha = 0.047$ annual, per adult birth rate.

- $\beta$ is the rate at which consumers become producers, or children grow up and become adults. Since the age of adulthood is approximately 15, we set this by default as $\frac{1}{15}$. This agrees closely with definitions used in most of the literature.

- $\gamma_1$ is the death rate for uninfected children, $\gamma_3$ is the death rate for infected but asymptomatic children, and $\gamma_5$ is the death rate for children with AIDS. We are unable to find
good information for death rates of healthy children, probably because child survival depends also on adult health. These rates are extremely community dependent, will change with overall HIV prevalence in the community, and prevalence of other diseases. We calculate in the following way: The life expectancy at birth of a seronegative person in rural Africa is 60 [22] years, 15 of which are in childhood. This would yield a death rate of .0167, and we know the adult death rate to be .0105. If we assume that the overall death rate is a weighted average, then $\gamma_1 = .0367$.

- The percent reduction in death rate due to administering ARV to AIDS infected individual is $l_1$ for consumers, $l_2$ for producers. Death rates for patients receiving ARV are surprisingly high, but this is largely because they do not seek treatment until the disease is quite advanced. This is consistent with our other choice. From our own literature review, and composite ones from other scientists, we use an approximate death rate from a number of different programs in different countries, such as Malawi and Botswana. The variation among retention for various reasons was huge, but loss of patients due to death varied less. Death contributed to 40 percent of attrition overall. We will take 10 percent as a fair representation of a death rate of patients receiving ARV treatment per year, while among adults, that rate is as low as 2.5 percent. So, $l_2\gamma_6 = .025$ and $l_1\gamma_5 = .075$ [23–26]. The parameter is used as follows: if we are treating $I_{pa}$ at a rate $\mu_p$, then the death rate will be $(1 - \mu_p)\gamma_6 + \mu_pl_2\gamma_6$, and for $I_{ca}$ it would be $(1 - \mu_c)\gamma_5 + \mu_cl_1\gamma_5$

A.0.2 Epidemiological Parameters

- $\zeta_1$ and $\zeta_2$ describe the linear rates of disease transmission, for consumers and producers respectively. These include doctors visits, IV drug use, contact with outside of the community, and migration rates into the community. These rates are extremely community specific. We set $\zeta_1 = 0$ for the simulations, because it seems likely that children will have much lower rates of contact with the outside community than adults. This factor could easily be included if it were known for a specific community. $\zeta_2$ would include factors such as rates of sexual contact with individuals outside of the community, and ambient infection rates in the country or region as a whole. In some studies, approximately one third of the producer (adult) population spend the majority of their time living and working outside of the geographic area [13], but returned to their home community regularly. It seems very likely that this segment of the population is often exposed to infection, especially through sexual contact with outside of the community. We will make a very rough lower estimate for this parameter of $\zeta_2 = .005$.

- $\tau_2$ is the rate at which an adult will develop AIDS, or transition from $I_p$ to $I_{pa}$. In Africa, this latency period has been documented as short as 4 years [2], and as long as 9 years [27]. Some of this variation is due to the definition of AIDS onset, but most is probably due to the specifics of the population. We will take $\tau_2 = 1/6$ as a representative estimate.
• $\tau_1$ tells us how quickly a child develops AIDS. While the overall rate for children ages 0 to 15 appears to approximately coincide with our approximate for adults, and we will use $\tau_1 = \tau_2$ in our simulations, it is important to note that the mechanism and distribution in five year cohorts of deaths is very different for children from adults. Infants under age 5 are the group of children most affected by HIV, children between ages 5 and 15 are the age group that suffer the lowest mortality due to AIDS, by an order of magnitude [28]. Of children born to HIV infected mothers, 52 percent of the infected children die by age 2, as opposed to 7.6 percent of uninfected, so the magnitude of the effect is quite clear. [29]

• $\alpha_{41}$ is the rate of non-HIV infected children born to an $I_p$ individual.

• $\alpha_{43}$ is the rate of HIV infected children born to an $I_p$ individual.

• $\alpha_{61}$ is the rate of non-HIV infected children born to an $I_{pa}$ individual.

• $\alpha_{63}$ is the rate of HIV infected children born to an $I_{pa}$ individual.

In Africa, the rate of vertical transmission, call this $r$, ranges from 25 – 35 percent, in the absence of ARV. We are concerned with rural areas where many interventions during birth and afterward are not possible which might bring these rates down, so we will take $r = .35$ for $I_{pa}$. Cesarean sections require a great deal of medical equipment, and breast feeding will be very likely as formula is expensive. We will guess that risk factors are not too different for all levels of infection in the mother, because studies have not been conclusive as to the relationship between CD4+ count and rate of vertical transmission. [30] It would be very easy to fix this if better information were known, since the parameters are decoupled already.

$$\alpha_{61} = .90 \alpha \quad \alpha_{43} = .10 \alpha \quad \alpha_{63} = r \alpha = .35 \alpha$$

• $p_v = .95$ is the protection that ARVs confer against vertical transmission. Rates of protection vary widely, depending on the type and duration of intervention used during pregnancy. We are mostly concerned with populations where. Since we are also concerned in this paper primarily with the health of adults, rather than the distinctly different focus of preventing vertical transmission, we will assume that pregnant mothers are getting combination HAART, rather than just ZDT (which does not return adults to productivity), which is extremely effective at preventing transmission. Then the adjusted birth rates from $I_{pa}$ with treatment are as follows: healthy kids born are now

$$\alpha_{61} = [(1 - \mu_p)(1 - r) + \mu_p(1 - p_v r)]\alpha$$

$$\alpha_{63} = [(1 - \mu_p)r + \mu_p(1 - r)]\alpha$$

• $\iota_a$ is the probability of transmission per month through sexual contact between serodiscordant couples where one partner is suffering from AIDS and is not being treated with ART. One study cites the rate .0897 per year [21]. [31] lists the combined rate from all seropositive individuals as .0564, and states that the transmission from individuals with extremely high viral load has been documented as high as .0903. From this, we think that choosing $\iota_a = .09$ gives an accurate view for transmission from $I_{pa}$

• $\iota_s = .0016$ is the transmission rate through sexual intercourse with individuals who still
have relatively undamaged immune systems, $I_p$. [21]

- $p = .92$ is the reduction in sexual transmission rate from those on ARV [21]. This difference in transmission stems not only from the lowering of viral load in the infected individual, but also from changes in behavior that often follow treatment. Condom use increases. We use rates that refer to heterosexual sex, and do not necessarily incorporate extremely risky behavior, such as visiting sex workers. This data would need to be gathered at a community-specific level. It could make a very big difference to the transmission dynamics.

Transmission between a $S_p$ and $I_{pa}$ who is getting ARV will $= p * \iota_a$

- $\iota_{ns} = .005$ is the probability of transmission per month through non-sexual contact with any infected individual (child or adult). This affects contact with $I_c, I_p, I_{ca}$ or $I_{pa}$. This is a poorly studied, but extremely important mode of transmission, we assume that it is an order of magnitude less than sexual transmission. It is very likely that this is an underestimate, but that would only strengthen our argument. While these transmission events (IVs, rituals) usually occur much more rarely, each contact has a much higher likelihood of transmission than heterosexual intercourse. [32, 33] We make this rate the same for contact with symptomatic and asymptomatic people because while symptomatic individuals are more infectious, many kinds of contact with them will be more rare, while there are a number of traditions that would be particularly likely to occur through contact with an asymptomatic individual (such as widow cleansing).

### A.0.3 Economic Parameters

The economic parameters in this model are in no way meant to be realistic. They are placeholders to reflect an overall impact on the level of functionality of the community due to the HIV epidemic. Looking at the demographic makeup as percent consumers vs percent producers is not quite sufficient to summarize this, because it does not capture the percentage of able-bodied producers. The productivity factor is an overall average measure of able-bodiedness, not an actual measure of (for example) employment. Unemployment in the adult population in many of these countries is extremely high in general [13], and so the actual output of the community would depend on many external economic factors. We want to measure the labor potential and how it is impacted by AIDS, as well as by the provision of ART.

In one large-scale study in South Africa, comparing patients receiving pre-ARV care and those following an ART regimen, individuals who were not yet receiving ART reported significant impairment during the previous work week about twice as often [14]. In another, larger scale study [34], patients enrolled in a public sector program were seen to recover almost 90% of their productivity three to four years before initiation therapy (while asymptomatic).

In the nearly full recovery case, we should to note that there is a significant lag between initiation of therapy and recovery of productivity. We do not have a good way of incorporating this into the model- it was proposed that we build in a lag to the resources equation (calculating it from
the population state three years back)- but I believe this would be inaccurate, as the resources in a subsistence community really depend on the state of the current population.

- $\eta_c = 2, \eta_p = 3$ consumption rates for consumers and producers, respectively. These are chosen arbitrarily, and take into account only that adults consume more food than children.

- $\lambda$ productivity of a healthy producers. AIDS producer who gets ART is at $.85\lambda$ [13]

- $\lambda_h$ is the productivity rate of $I_p$. While we have generally described these individuals as asymptomatic, there is usually a sharp peak in viral load soon after infection when an individual experiences symptoms. Furthermore, as onset of symptoms can be gradual, with a slow increase in days when an individual cannot work leading up to when they might seek treatment. We will take $\lambda_h = .9\lambda$.

- $\lambda_a$ is the productivity rate of an individual who is suffering from AIDS. We will take $\lambda_a = .5\lambda_h$ for our simulations, but this is again just an average.

- $tr$ is the fraction of $\lambda_h$ that a symptomatic producer who gets ARV hopes to regain. As discussed above, a symptomatic individual who gets ARV treatment has been shown to eventually recover up to 90% of his or her productivity 3 – 4 years prior to initiating therapy, so we let $tr = .9$, and the treated $I_{pa}$ are productive at a rate of $tr\lambda_h$.

- If we were to treat producers in the early stages of infection, there is good evidence that they would regain full productivity. So, the productivity of $I_p$ who are put on ART is $\lambda$

To make a more detailed analysis than the one we have here, one would need information about a specific community, and well as the type of ARV treatment program in place there. Some private companies provide HIV therapy and treatment for their workers, and these often have extremely good adherence rates. There are also studies which parse more carefully the incremental loss of productivity leading up to loss of employment [10] in a particular industry. This kind of information would be very helpful if the community depends on a particular industry or crop for the majority of its employment.
Bibliography


