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The Dispersion of Age Differences between Partners and the Asymptotic Dynamics of the HIV Epidemic

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Abstract

In this article, the effect of a change in the distribution of age differences between sexual partners on the dynamics of the HIV epidemic is studied. In a gender and age structured compartmental model, it is shown that if the variance of the distribution is small enough, an increase in this variance strongly increases the basic reproduction number. Moreover, if the variance is large enough, the mean age difference barely affects the basic reproduction number. We therefore conclude that the local stability of the disease-free equilibrium relies more on the variance than on the mean.

1 Introduction

Thirty years after the discovery of the first confirmed clinical cases, the HIV epidemic is not yet under control. Worldwide, UNAIDS (2010) estimates that 2.6 million adults and children were newly infected with HIV in 2009. These cases affect Africa disproportionately, and especially Sub-Saharan Africa, which accounts for 69% of the new infections (1.8 million in 2009, according to UNAIDS, 2010). This article is concerned with a demographic explanation of the differences in the evolution of the epidemic that have been observed across regions. More precisely, we study the effect of the distribution of age differences between sexual partners on the long-run dynamics of the epidemic and on its endemic nature.

The age mixing, or age differences, among marital partners is particularly widespread in Africa compared to other parts of the world. Spijker (2011) illustrates this pattern by providing statistics on the distribution of married couples by age differences using the most recent census data from the Integrated Public Use Microdata Series. In Africa, the proportion of couples having more than 8 years of age difference ranges from 22.5% (South Africa, 1996) to 80.3% (Guinea, 1996) while the same proportion ranges from 6% (China, 1990) to 26.2% (Malaysia, 1980) in Asia, and from 17.5% (Chile, 1992) to 28% (Panama, 1990) in Latin America. A large amount of literature documents the particular frequency of age mixing in Sub-Saharan Africa. Historically, age mixing has been commonplace in Africa (Casterline et al., 1986) as a result of practices such as polygamy, the remarrying of widows and the premature marrying of young girls. Studies have shown that age differences persist nowadays throughout Africa[1] both within marital and non marital partnerships, and both within casual and regular relationships (Auvert et al., 2001, Gregson et al., 2002). It has been found that about 40%

[1] See Luke (2003) for a literature review on age mixing and possible reasons why it is widespread in the African context.

to 50% of young girls are involved in partnerships with a partner who is five to nine years older (Gregson et al., 2002, Kelly et al., 2003, Konde-Lule et al., 1997). Greater age differences are also common as between 16% to 27% of the young girls partnerships involving an age difference of ten years or more (Konde-Lule et al., 1997; Gregson et al., 2002; Kelly et al., 2003). This age mixing persists for older women as the majority of the married women of age 15 to 44 years old, studied in Boerma et al. (2003), have a husband who is at least six years older. Studying male non marital unions, Luke (2005) finds that 70% of the sampled men are five or more years older than at least one of their recent partners and 20% are ten years or more older.

Grounded on the empirical evidence that the HIV prevalence rate is much greater among young women than among young men (e.g. Buvé et al., 2001, Glynn et al., 2001, Gouws et al., 2008), a growing body of research examines age differences between partners as a potential risk factor of HIV infection. Some articles document the association between age difference between sexual partners and the increased risk of HIV infection (Gregson et al., 2002, Kelly et al., 2003). The increase in risk is significant as documented by Kelly et al. (2003), who finds that the 15-29 year old women engaged in partnership with a partner 5 to 9 years older or 10 years or more have a respective risk of infection of 1.1 and 1.28 times higher than that of their counterparts having partners 0 to 4 years older. Related papers have shown that partnerships involving large age differences are less likely to adopt safe practices than their counterparts, as women in long-term partnerships involving age difference of more than 5 years (Blanc and Wolff, 2001) and men engaged in non marital partnerships involving age difference of 10 years or more (Luke, 2005) are less likely to use a condom than their counterparts.

The importance of age differences between partners on the diffusion and persistence of the epidemic was first brought up by Anderson et al. (1992). Through numerical simulations, the authors showed that the epidemic spreads more rapidly when there is infectious contact between generations. An in-

tuition has been proposed by Brouard (1994) who stressed the importance of the variance of the distribution. The latter could be one of the explanatory causes of a markedly higher prevalence of HIV in Africa. Whatever the mean, if the variance is very low, one can imagine that there would only be minimal transmission of the virus from the first cohorts of a given gender to be affected by the epidemic to the younger cohorts of the same sex. Thus, the dynamics of HIV infection would be epidemic in nature. On the other hand, if there is a significant variance, transmission of the disease to younger cohorts is potentially significant and hence the dynamics are likely to be endemic.

The objective of our article is to propose a formal framework to evaluate the impact of the distribution of age differences between partners on the dynamics of the epidemics. We will proceed in three steps. First, we seek to show that the distribution of age differences between sexual partners has not been modified by the emergence of HIV. This analysis is performed on a sample of African countries given data constraint. However one could argue that if such a scenario has prevailed, that is, if people have changed their matching preferences as a protective behavior against HIV, it is much more likely to have occurred in the region that exhibits the highest levels of prevalence in the world. Using the distribution of age differences for married couples, we show that its mean and variance have not undergone significant variation over time.

Secondly, this preliminary evidence is used to establish a theoretical model in which the distribution of age differences between partners is exogenous to the path of the epidemic. Our model, which is both age- and gender-structured, is an extension of Anderson et al.'s (1992) framework, which allows us to take into account the unique nature of epidemics involving sexually transmitted diseases. We study the stability of the disease-free equilibrium. One important element of our model is the contact function that incorporates the distribution of age differences between partners. Unlike most models

in the literature, our function is necessarily non-separable, which makes it impossible to calculate the basic reproduction number, R_0, explicitly. Nevertheless, by using the operators theory, we are able to establish the local properties as well as some global properties of R_0.

Finally, we assume that the distribution of age differences between partners is characterized by a given distribution and we analyze the effect of both the mean and the variance on R_0. Numerical computations show that variance plays a crucial role as R_0 strongly increases with the variance if it is sufficiently low. Moreover, if the variance is large enough, the mean age difference barely affects R_0. We conclude that, whatever the mean age difference, the disease-free equilibrium will thus have a greater chance of being stable if the variance is small.

This paper is organized as follows. Section 2 presents our empirical evidence. Section 3 describes the dynamic model and section 4 presents our theoretical results. Our numerical results are developed and commented in section 5. Section 6 concludes.

2 Empirical evidence

This section examines the distribution of age-difference between spouses, especially the evolution of its mean and variance over time. In industrialized countries like Sweden, the average age-difference has been found to be stable among the cohorts born between 1883 and 1942, despite a decrease in the age at marriage (Bergstrom and Lam, 1994). In Sub-Saharan Africa where the epidemic has reached tremendously high levels and where the age-difference has been pointed out as a risk-factor of HIV infection, one might wonder whether individuals have adjusted their behavior toward a reduction in the age difference since the onset of the epidemic, as a self-protective device. Data from the Demographic and Health Surveys[2] conducted in Sub-Saharan

[2]These surveys are publicly available at http://www.measuredhs.com

Africa suggest that this scenario is very unlikely.

In order to find out whether the AIDS epidemic has changed matching behaviors and shifted individuals' preferences toward fewer age mixing, we use the distribution of age differences for married couples. As time series of spousal age differences are not available, we obtained data from the self-reported age differences in the most recent Demographic and Health Surveys conducted in Sub-Saharan Africa. In these surveys, women respondents who are currently married are asked to report their current age, the current age of their partner and the year in which they got married. Given the spousal age difference and the marriage year, we are able to establish the empirical distribution of spousal age differences for each marriage year. The year in which the marriage was celebrated is an indicator of the time period in which the individual made her decision about partner selection. Consequently, it provides more accurate information about individual behaviors than any cross sectional analysis.

We restrict the sample to women who married when aged between 15 and 25 years old for two reasons. Firstly, it is the most common age interval in which women marry. In Lesotho, for instance, this sub-sample accounts for 90% of the total sample. Secondly, and more importantly, this sample restriction allows us to rule out heterogeneities in the marital pattern from our analysis. Indeed, women who were married after reaching age 25 might have been previously married to someone else, or might have different preferences in terms of partner selection compared to women who get married at younger age.

To obtain a first indicator as to whether the spread of AIDS in Africa has induced changes in the choice of partner, we draw the distribution of spousal age differences for a low-prevalence and a high-prevalence country and for two distinct samples: women who married before 1990 and those who married after 1990. Figure 1 charts the empirical distributions for Lesotho which is one of most affected countries in Sub-Saharan Africa, since 23.6% of its adult

population was HIV infected in 2009 (UNAIDS, 2010). Similarly, Figure 2 charts the distributions for Niger, a country which has one of the lowest infection rates on the continent as its adult HIV prevalence rate reached 0.8% in 2009 (UNAIDS, 2010).

Figure 1, about here.

Figure 2, about here.

Taking 1990 as a benchmark year, the two distributions are very similar, suggesting that there was no adjustment in behavior after populations became informed about the HIV/AIDS epidemic and its ways of transmission.

The Demographic and Health Surveys are standardized nationally representative household surveys that collect data in various African countries based on a standardized questionnaire. We are thus able to generalize our analysis by using a large set of countries[3] in order to test whether the distribution of the spousal age differences is constant over time.

We use the survey to compute the mean and the coefficient of variation of the distribution of the spousal age differences by country and by marriage year. Figures 3 and 4 provide the dynamics of the mean and the coefficient of variation[4], respectively, for each country of the sample.

Figure 3, about here.

Figure 4, about here.

There is no clear pattern suggesting a change in the distribution of the age differences over time, except for Ghana and Malawi, where one could notice a downward trend in the mean from the mid-1980s onwards. The mean

[3]The countries, with the date of the DHS survey, are the following: Burkina Faso (2003), Cameroon (2004), Democratic Republic of Congo (2007), Ethiopia (2005), Ghana (2003), Guinea (2005), Kenya (2003), Lesotho (2004), Liberia (2007), Mali (2006), Malawi (2004), Niger (2006), Rwanda (2005), Senegal (2005), Swaziland (2006/07), Zambia (2007) and Zimbabwe (2005/06).

[4]The coefficient of variation is the standard deviation divided by the mean.

of age differences decreases from 1985 in Ghana and from 1986 in Malawi, but these decreases are not statistically significant. If we go back to the individual data, and implement a T-test to test for the difference between the population mean of age differences in these years and at the end of the period, we find that in both cases, we cannot reject the null hypothesis that the population mean is equal in 1985 and in 2003 in Ghana (and in 1986 and in 2005 for Malawi).

To test the stability of the distribution of spousal age differences over time, we use a linear fixed effects model to successively estimate the mean and the coefficient of variation of the spousal age differences at the country-year level using as independent variables the marriage year and a dummy variable that takes value one if the marriage was celebrated before 1990 and zero otherwise. Empirical results presented in Table 1 suggest that the marriage year and the act of getting married before the spread of the AIDS epidemic have no statistically significant effect on the dependent variables, i.e. the mean (column 1) and the coefficient of variation (column 2).

Dependent variable	mean	coefficient of variation
Marriage year	−0.0311	0.0021
	(0.021)	(0.002)
1 if marriage before 1990	0.0234	0.0467
	(0.244)	(0.027)
Constant	70.6746	−3.315
	(41.161)	(3.198)
Country effects	Yes	Yes
Number of observations	604	600
Number of countries	17	17

Table 1

Linear fixed effects estimates

(in parentheses: robust standard errors, clustered at the country level)

These stylized facts suggest that controlling for country-specific effects, distributions of spousal age differences are stable over time and that the onset of the epidemic disease does not imply any adjustment in preferences regard-

ing the age difference between spouses. Therefore, grounded on this empirical evidence, the next section will consider the dispersion of age differences as an exogenous parameter of the model.

3 An age-structured mathematical model

3.1 The model

Our model can be seen as an extension of the model developed by Anderson et al. (1992), which describes the spread of a sexually transmitted epidemic disease in a multi-group model. Multi-group modeling is due to the gender specific variables we use. Our main departure from the work of these authors lies in the definition of the boundary conditions that characterize the birth process. Indeed, we assume that the latter depends on sexual behaviors and that, as a consequence, it is intrinsically linked to the spread of the epidemic disease.

For each gender $g \in \{f, m\}$ where f corresponds to the population of women while m corresponds to the population of men, let $S_g(t,a)$ and $I_g(t,a)$ denote, respectively, the (chronological) age-specific density at time $t \in \mathbb{R}^+$ of susceptible and infective individuals of age $a \in [0, \omega]$ where $\omega > 0$ denotes the maximal length of life. Their dynamics are given by the following system of equations:

$$\frac{\partial S_g(t,a)}{\partial t} + \frac{\partial S_g(t,a)}{\partial a} = -S_g(t,a)\left[\mu(a) + \lambda_g(t,a)\right], \quad (1)$$

$$\frac{\partial I_g(t,a)}{\partial t} + \frac{\partial I_g(t,a)}{\partial a} = -I_g(t,a)\left[\mu(a) + \mu_1(a)\right] + S_g(t,a)\,\lambda_g(t,a), \quad (2)$$

where $\mu(a)$ and $\mu_1(a)$ are, respectively, the age-specific mortality rate of individuals at age a and the over-mortality rate of infected individuals at age a. The probability of an individual of gender g and age a being infected at time t is modeled by the so-called force of infection denoted by $\lambda_g(t,a)$.

Before discussing the specific form of the force of infection, let us introduce some notations. Let $N_g(t,a) = S_g(t,a) + I_g(t,a)$ denote the density of

individuals of gender g and age a at time t. Using (1) and (2), we obtain:

$$\frac{\partial N_g(t,a)}{\partial t} + \frac{\partial N_g(t,a)}{\partial a} = -\mu(a) N_g(t,a) - I_g(t,a) \mu_1(a). \tag{3}$$

Consider also the per head age-specific variables that are defined as:

$$s_g(t,a) = \frac{S_g(t,a)}{N_g(t,a)} \text{ and } i_g(t,a) = \frac{I_g(t,a)}{N_g(t,a)}.$$

One important feature of our model is that the probability of being infected depends on the age of the partner, denoted a'. The minimum age at which individuals become sexually active is denoted $a_0 \in [0, \omega)$. Furthermore, homosexual relationships are not considered in our model. These assumptions imply that the force of infection is of criss-cross type and is given by:

$$\lambda_g(t,a) = \int_{a_0}^{\omega} \beta_g(a,a') \rho_g(t,a,a') i_{-g}(t,a') da'. \tag{4}$$

Note that the probability of being infected has three components. The function $\beta_g(a,a')$ is the infectiousness of the disease, i.e. the probability of being infected when having an infected partner of age a'. The component that will be crucial for our analysis is denoted by $\rho_g(t,a,a')$ and represents the average number of partners of age a' and of opposite gender $-g$ per individual of age a and gender g (see Anderson et al., 1992). Lastly, the force of infection depends on $i_{-g}(t,a')$, the proportion of infectious individuals among those of age a' and gender $-g$.

Let us notice that we allow for time-dependence in the average number of partners. More precisely, we assume that such a function can be characterized by the product of two functions: $c_g(t,a)$, the rate of partner change for an individual of age a and gender g at time t, and $J_g(t,a,a')$, a mixing function indicating, at time t, the probability that an individual of age a and gender g chooses a partner of age a'. It satisfies $\int_{a_1}^{a_2} J_g(t,a,a')\, da' = 1$. We hence have:

$$\rho_g(t,a,a') = c_g(t,a) J_g(t,a,a').$$

Functions $c_g(t,a)$ and $J_g(t,a,a')$ are linked to each other through the following constraint:

$$c_f(t,a)\,J_f(t,a,a')\,N_f(t,a) = c_m(t,a')\,J_m(t,a',a)\,N_m(t,a'). \tag{5}$$

Following Anderson et al. (1992), we shall assume that function $\rho_g(t,a,a')$ can be non-autonomous (i.e. time dependent) for one gender only, namely for men. Then assume that, for women, the mixing function is time independent, and reads as: $J_f(t,a,a') \equiv J_f(a,a')$. As a consequence, the age-specific force of infection for women takes the following form:

$$\lambda_f(t,a) = \int_{a_0}^{\omega} \beta_f(a,a')\,c_f(a) J_f(a,a') i_m(t,a')\,da'. \tag{6}$$

It therefore remains to model the age-specific force of infection for men. Following Anderson et al. (1992), we will assume that the mean rate of partner change for men is given by:

$$c_m(t,a') = \frac{\int_{a_1}^{a_2} c_f(a)\,J_f(a,a')\,N_f(t,a)\,da}{N_m(t,a')},$$

while the mixing function is computed according to (5). As a consequence, one obtains that

$$\lambda_m(t,a) = \int_{a_0}^{\omega} \beta_m(a,a')\,c_f(a')\,J_f(a',a)\,\frac{N_f(t,a')}{N_m(t,a)} i_f(t,a')\,da'. \tag{7}$$

Let us now describe the boundary conditions that characterize the birth process. Let $b(a)$ be the probability of age a susceptible and infected women who have a sexual partner, to give birth to a child. Furthermore, in order to simplify the model, assume that there is no vertical transmission of the disease (i.e. all children are born susceptible). Using the time independence assumption for $\rho_f(a,a')$, the boundary conditions read:

$$\begin{cases} S_g(t,0) = \sigma_g \int_0^{\omega} b(a)\,N_f(t,a) \int_{a_0}^{\omega} \rho_f(a,a')\,da'da, \\ I_g(t,0) = 0, \end{cases} \tag{8}$$

where σ_g is the secondary sex ratio that satisfies $\sigma_g \left[N_f(t,0) + N_m(t,0)\right] = N_g(t,0)$. We thus assume that birth depends on contact behavior, which is the same as the one involved in the transmission of the disease. Moreover, the system is described by the following initial data:

$$\begin{cases} S_g(0,a) = S_g^0(a), \\ I_g(0,a) = I_g^0(a), \end{cases} \tag{9}$$

with $S_g^0(a)$, $I_g^0(a) \in L^1(0,\omega)$ and $S_g^0(a)$, $I_g^0(a) \geq 0$ a.e. in $[0,\omega]$.

In summary, the model we consider consists in equations (1), (2), (6) (7), (8) and initial data (9).

3.2 A simplified model

In order to deal with the above age-structured model, we have to consider the possibility of an exponentially growing population. This is due to the linear assumption on the demographic parameter. In the absence of disease, namely $I_g \equiv 0$, the dynamics of the population is driven by the following linear age-structured system of equations given for each gender:

$$\begin{cases} \frac{\partial N_g(t,a)}{\partial t} + \frac{\partial N_g(t,a)}{\partial a} = -\mu(a) N_g(t,a), \\ N_g(t,0) = \sigma_g \int_0^\omega b(a) N_f(t,a) \int_{a_0}^\omega \rho_f(a,a')\, da' da, \\ N_g(0,a) = N_g^0(a) \quad g \in \{m,f\}. \end{cases} \tag{10}$$

For this kind of equations, one can expect a Malthusian growth for the population. This remark will allow us to simplify the model considered above and especially the force of infection for men given by (7). Indeed, if we assume that for each class of age and each gender, the number of infective $I_g(t,a)$ remains small with respect to the age-specific total number of individuals $N_g(t,a)$, then $N_g(t,a)$ arising in (7) can be approximated by the solution of the diseased free population (10). The latter system is well known and the equation for N_f is referred as the Lotka-McKendrick equation. We refer to

the textbooks of Webb (1984) and of Iannelli (1995) for a complete study of this kind of equations. The Lotka-McKendrick equation for N_f reads as

$$\begin{cases} \frac{\partial N_f(t,a)}{\partial t} + \frac{\partial N_f(t,a)}{\partial a} = -\mu(a) N_f(t,a), \\ N_f(t,0) = \sigma_f \int_0^\omega b(a) N_f(t,a) \int_{a_0}^\omega \rho_f(a,a') da' da, \\ N_f(0,a) = N_f^0(a). \end{cases} \tag{11}$$

The equation is well known to satisfy the so-called Asynchronous exponential growth. This means that if we introduce the Malthusian parameter $\gamma \in \mathbb{R}$ of the population, which corresponds to the $\gamma \in \mathbb{R}$ solution of:

$$1 = \sigma_f \int_0^\omega b(a) e^{-\int_0^a (\mu(z)+\gamma)dz} \int_{a_0}^\omega \rho_f(a,a') da' da, \tag{12}$$

one obtains that

$$\lim_{t\to\infty} e^{-\gamma t} N_f(t,a) = \overline{N}_{f0} e^{-\int_0^a (\mu(z)+\gamma)dz},$$

for the topology of $L^1(0,\omega)$. Here $\overline{N}_{f0} \geq 0$ is some given number depending on $N_f^0(a)$, through a suitable projector operator. From this asymptotic property, one can drive a similar behavior for N_m. Indeed simple computation shows that

$$\lim_{t\to\infty} e^{-\gamma t} N_m(t,a) = \overline{N}_{m0} e^{-\int_0^a (\mu(z)+\gamma)dz},$$

where $\overline{N}_{m0}$ is related to $\overline{N}_{f0}$ through the following relation

$$\overline{N}_{m0} = \frac{\sigma_m}{\sigma_f} \overline{N}_{f0}. \tag{13}$$

This remark allows us to simplify formally the epidemic system under consideration and especially (7). Indeed if for each class of ages and each gender, the number of infective people $I_g(t,a)$ remains small with respect to the age-specific total number of individuals $N_g(t,a)$, then one obtains, at least for large t, that

$$\frac{N_f(t,a')}{N_m(t,a)} \approx \frac{e^{\gamma t} \overline{N}_{f0} e^{-\int_0^{a'} (\mu(z)+\gamma)dz}}{e^{\gamma t} \overline{N}_{m0} e^{-\int_0^a (\mu(z)+\gamma)dz}}$$

and therefore the age-specific force of infection for men becomes

$$\lambda_m(t,a) = \frac{\sigma_f}{\sigma_m} \int_{a_0}^{\omega} \beta_m(a,a')\, c_f(a')\, J_f(a',a)\, e^{\int_{a'}^{a} \mu(z)dz + \gamma(a-a')} i_f(t,a')\, da'. \tag{14}$$

Note that this simplification makes sense for growing populations, i.e. for positive Malthusian parameter.

This simplification is studied further and validated using numerical simulations as presented in the figures below. To perform numerical investigations, we assume that the mixing function takes the following form:

$$\begin{cases} J_f(a,a') = \dfrac{e^{-\frac{(a-a'+\nu)^2}{2\sigma^2}}}{\int_{a_1}^{a_2} e^{-\frac{(a-\alpha+\nu)^2}{2\sigma^2}} d\alpha} \text{ for } a, a' \in [a_1, a_2], \\ \\ 0 \text{ otherwise,} \end{cases}$$

where $\nu > 0$ stands for the mean age difference between men and women and $\sigma > 0$ for the standard deviation that measures the dispersion of age differences within couples. The function describing the mean rate of partner change is also taken from Anderson et al. (1992) and reads as:

$$c_f(a) = \begin{cases} \eta \text{ for } a \in [a_1, a_2], \\ \\ 0 \text{ otherwise.} \end{cases}$$

Figures 5 and 6 represent the forces of infection for men computed as a solution of the system composed of equations (1), (2) and either (4) or (14). More precisely, the continuous curve describes the L^1-norm of (4) with respect to time while the dotted line corresponds to that of (14). The chosen parameter sets imply an eradication of the disease in Figure 5, and the convergence towards an endemic equilibrium point in Figure 6. These computations show that when the time is sufficiently large, the two forces of infection have the same behavior.

Figure 5, about here.

Figure 6, about here.

Finally, our simplification allows us to deal with age-specific prevalence $i_g(t,a)$, to derive an expression for R_0 and to study its dependence with respect to various parameters. Let us also mention that our simplification keeps some information on the Malthusian parameter of the total population, namely parameter γ, and also on the sex-ratio parameters σ_g. More specifically, using the above simplification and using the independent variables $i_f(t,a)$ and $i_m(t,a)$, the system we will consider reduces combining equations (3) and (10), to the following one:

$$\begin{cases} \frac{\partial i_g(t,a)}{\partial t} + \frac{\partial i_g(t,a)}{\partial a} = (1 - i_g(t,a))\left[\lambda_g(t,a) - \mu_1(a) i_g(t,a)\right] \quad t > 0,\ a \in (0,\omega) \\ \\ i_g(t,0) = 0 \quad g \in \{m,f\}, \\ \\ i_g(0,.) = i_g^0(.) \in L^1(0,\omega;\mathbb{R}^+) \quad g \in \{m,f\}. \end{cases} \tag{15}$$

In the above system of equations, λ_f and λ_m are respectively given by (6) and (14).

4 Basic reproduction number

This section aims at deriving some basic mathematical properties of (15) together with (6) and (14). The local dynamics are studied by analyzing the spectral radius of a linear operator of a related system. The difficulty comes from the fact that in contrast to most papers in the literature, we do not assume the separability of the rates of infection. It is therefore not possible to derive an explicit expression for the spectral radius of the next generation operator. Spectral theory provides however well-known tools to obtain properties for the spectral radius. We establish some of its properties that will allow us, in the last part, to obtain some properties about the dynamics of some specified contact rate functions.

Because of the biological definition of i_g, we introduce the following state Banach lattice spaces $X = L^1(0,\omega;\mathbb{R}) \times L^1(0,\omega;\mathbb{R})$ endowed together with

the usual product norm as well as

$$\mathcal{C} = \left\{ \begin{pmatrix} i_f \\ i_m \end{pmatrix} \in X : \ \ 0 \leq i_g \leq 1 \ a.e. \ \ g \in \{f, m\} \right\}. \tag{16}$$

In order to derive mathematical properties of (15), let us rewrite the system of equations using the following form:

$$\begin{cases} \frac{\partial i_g(t,a)}{\partial t} + \frac{\partial i_g(t,a)}{\partial a} = (1 - i_g(t,a)) \left[\Lambda_g[i_{-g}(t,.)](a) - \mu_1(a) i_g(t,a) \right], \\ \\ i_g(t,0) = 0, \ \ \forall g \in \{m, f\}, \\ \\ \begin{pmatrix} i_f(0,.) \\ i_m(0,.) \end{pmatrix} = \begin{pmatrix} i_f^0(.) \\ i_m^0(.) \end{pmatrix} \in \mathcal{C}, \end{cases} \tag{17}$$

wherein we have set for each $g \in \{f, m\}$,

$$\Lambda_g[\varphi](.) = \int_0^\omega \gamma_g(., a')\varphi(a')da', \ \forall \varphi \in L^1(0, \omega; \mathbb{R}),$$

for some functions $\gamma_g \equiv \gamma_g(a, a')$ coming from (6) and (14). Functions $\gamma_g \equiv \gamma_g(a, a')$ stand for the so-called rate of infection from contacts between an infective individual of age a' and a susceptible individual of age a (see Li et al., 2008). The above system of equations will be studied by using the following set of assumptions.

Assumption 1 *Assume that* $\mu_1 \in L_+^\infty(0, \omega; \mathbb{R}^+)$ *and, for each* $g \in \{f, m\}$, *functions* γ_g *belong to* $L^\infty((0, \omega) \times (0, \omega); \mathbb{R}^+)$.

As a consequence, Λ_g defined above becomes a bounded linear operator $\Lambda_g : L^1(0, \omega; \mathbb{R}) \to L^\infty(0, \omega; \mathbb{R})$. Next, the functional framework is defined as follows. Let us recall first that $X = L^1(0, \omega; \mathbb{R}) \times L^1(0, \omega; \mathbb{R})$ is a Banach Lattice partially ordered with its positive cone X^+ defined by:

$$X^+ = L^1(0, \omega; \mathbb{R}^+) \times L^1(0, \omega; \mathbb{R}^+).$$

Moreover following the standard notion[5], for each $(\varphi, \psi) \in X$, the symbol $\varphi \leq \psi$ means that $\psi - \varphi \in X^+$. Our first Lemma establishes the existence

[5]We refer notably to Clément et al. (1987), Grobler (1987, 1995), Meyer-Nieberg (1991), Schaefer (1974) for some results on linear positive operators and Banach Lattices.

of a weak solution of system (17). Let $\alpha > 0$ be given such that:

$$\alpha > \mu_1(a) + \Lambda_g[1](a), \quad g \in \{f, m\}, \ a.e.\ a \in (0, \omega). \tag{18}$$

Then, consider the linear operator $A : D(A) \subset X \to X$ defined by

$$D(A) = \left\{ \varphi = \begin{pmatrix} \varphi_f \\ \\ \varphi_m \end{pmatrix} \in W^{1,1}(0, \omega; \mathbb{R})^2 : \ \varphi(0) = (0, 0) \right\},$$

and

$$A \begin{pmatrix} \varphi_f \\ \\ \varphi_m \end{pmatrix} = \begin{pmatrix} -\varphi_f' \\ \\ -\varphi_m' \end{pmatrix},$$

and the nonlinear operator $F : \mathcal{C} \to X$ defined by

$$F \begin{pmatrix} \varphi_f \\ \\ \varphi_m \end{pmatrix} = \begin{pmatrix} (1 - \varphi_f) \left(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f \right) \\ \\ (1 - \varphi_m) \left(\Lambda_m[\varphi_f] - \varphi_m \right) \end{pmatrix}.$$

Then, using $u(t) = (i_f(t, .), i_m(t, .))$, the system (17) can be rewritten as the following abstract Cauchy problem:

$$\begin{cases} \frac{du(t)}{dt} = Au(t) + F(u(t)), \quad t > 0 \\ \\ u(0) = \varphi \in \mathcal{C}. \end{cases} \tag{19}$$

Note that given the choice of α (see (18)), for each $(\varphi, \psi) \in \mathcal{C}^2$ such that $\varphi \leq \psi$, one obtains that:

$$F(\varphi) + \alpha\varphi \leq F(\psi) + \alpha\psi.$$

Consequently, system (19) is equivalent to

$$\begin{cases} \frac{du(t)}{dt} = (A - \alpha) u(t) + (F + \alpha)(u(t)), \quad t > 0 \\ \\ u(0) = \varphi \in \mathcal{C}. \end{cases} \tag{20}$$

We directly deduce the following result.

Lemma 1. *Let Assumption 1 be satisfied. Then, the operator $(A, D(A))$ is the infinitesimal generator of a C_0-positive semigroup $\{T_A(t)\}_{t \geq 0}$ on X.*

There exists a unique strongly continuous semiflow $\{U(t;\,.\,) : C \to C\}_{t\geq 0}$ *such that for each* $\varphi \in C$*, the map* $t \to U(t;\varphi)$ *is a mild solution of system (19), that is*

$$U(t;\varphi) = T_A(t)\varphi + \int_0^t T_A(t-s)F\left(U(s;\varphi)\,ds, \quad \forall t \geq 0.\right.$$

Moreover for each $(\varphi, \psi) \in C^2$ *one has*

$$\varphi \leq \psi, \;\; \Rightarrow \;\; U\left(t;\varphi\right) \leq U\left(t;\psi\right), \;\; \forall t \geq 0.$$

Proof. The proofs of similar results can be found in Webb (1985), Busenberg et al. (1991) and Feng et al. (2005). A key ingredient is given by the positivity of the semigroup generated by A, namely

$$T_A(t)\varphi(a) = \begin{cases} \varphi(a-t) \text{ if } t < a \\ \\ 0 \text{ if } t > a \end{cases}, \;\; \forall \varphi \in X.$$

□

Let us now study the local dynamics in the neighborhood of the so-called disease free equilibrium (DFE, hereafter) that corresponds to the stationary solution $i_g(t,a) \equiv 0$. We now aim at proving that the linear stability of the DFE is related to the so-called basic reproduction number. The corresponding linearized equation around the DFE is given by:

$$\begin{aligned} \frac{\partial u_f(t,a)}{\partial t} + \frac{\partial u_f(t,a)}{\partial a} &= -\mu_1\left(a\right)u_f + \Lambda_f[u_m(t,.)](a), \\ \frac{\partial u_m(t,a)}{\partial t} + \frac{\partial u_m(t,a)}{\partial a} &= -\mu_1\left(a\right)u_m + \Lambda_m[u_f(t,.)](a), \end{aligned} \tag{21}$$

together with

$$\begin{aligned} u_f(t,0) &= u_m(t,0) = 0, \\ \left(u_f, u_m\right)(0,.) &= \left(u_f^0, u_m^0\right) \in X. \end{aligned} \tag{22}$$

In order to study this linear equation, we consider the linear operator $\widehat{A} : D\left(\widehat{A}\right) \subset X \to X$ and the bounded linear operator $B : X \subset X \to X$ defined

by

$$D\left(\widehat{A}\right) = D(A), \;\; \widehat{A} = \begin{pmatrix} -\frac{d}{da} - \mu_1(a) & 0 \\ 0 & -\frac{d}{da} - \mu_1(a) \end{pmatrix},$$

and

$$B = \begin{pmatrix} 0 & \Lambda_f \\ \Lambda_m & 0 \end{pmatrix}.$$

Then, by setting $u(t) = (u_f(t,.), u_m(t,.))$, system (21)-(22) can be written as follows:

$$\frac{du(t)}{dt} = \left(\widehat{A} + B\right) u(t), \; t > 0, \;\; u(0) = u_0 = \begin{pmatrix} u_f^0 \\ u_m^0 \end{pmatrix} \in X.$$

In order to study some properties of the above linear problem, let us first establish the following result:

Theorem 1. *The linear operator $\widehat{A}+B : D(A) \subset X \to X$ is the infinitesimal generator of positive C_0-semigroups $\{T_{\widehat{A}+B}(t)\}_{t\geq 0}$ on X. We also have the fixed-point formulation:*

$$T_{(\widehat{A}+B)}(t) = T_{\widehat{A}}(t) + \int_0^t BT_{\widehat{A}+B}(s)ds, \;\; \forall t \geq 0,$$

and

$$\omega_{ess}(\widehat{A}+B) = -\infty, \;\; \omega_0(\widehat{A}+B) = s(\widehat{A}+B) \in \sigma\left(\widehat{A}+B\right). \qquad (23)$$

Here, $\omega_{ess}(\widehat{A}+B)$ denotes the essential growth rate of $\{T_{(\widehat{A}+B)}(t)\}_{t\geq 0}$, while $\omega_0(\widehat{A}+B)$ and $s(\widehat{A}+B)$ respectively denote the growth rate of $T_{\widehat{A}+B}(t)$ and the spectral bound of $(\widehat{A}+B)$.

Proof. It is easy to see that

$$T_{\widehat{A}}(t)\varphi = \begin{cases} 0 \text{ if } t > a \\ \\ e^{-\int_{a-t}^{a} \mu_1(s)ds}\varphi(a-t) \text{ if } a > t. \end{cases}$$

This proves that $T_{\widehat{A}}(t)$ is a nilpotent semigroup and therefore, we obtain that $\omega_{ess}\left(\widehat{A}\right) = -\infty$. To prove the other part of (23), using results obtained by Greiner (1984) as well as Voigt's perturbation result (Voigt, 1994), we need to prove that for each $t > 0$, the operator $BT_{\widehat{A}}(t)B$ is weakly compact in X.

Recalling that $T_{\widehat{A}}(t) = 0$ for all $t \geq \omega$, it is sufficient to consider the case $t \in (0, \omega)$. Let $t \in (0, \omega)$ be given. Then, we have:

$$BT_{\widehat{A}}(t)B = \begin{pmatrix} C_1 & 0 \\ 0 & C_2 \end{pmatrix},$$

where we have set

$$C_1\varphi_f = \int_0^\omega da\gamma_f(a,.)1_{(t,\omega)}(a)e^{-\int_{a-t}^a \mu(s)ds} \int_0^\omega \gamma_m(a-t,a')\varphi_f(a')da'$$
$$C_2\varphi_m = \int_0^\omega da\gamma_m(a,.)1_{(t,\omega)}(a)e^{-\int_{a-t}^a \mu(s)ds} \int_0^\omega \gamma_f\left(a-t,a'\right)\varphi_m\left(a'\right)da'.$$

Note that operators C_1 and C_2 both act on $L^1(0,\omega)$ and are bounded linear operators. Moreover, they satisfy

$$0 \leq C_i\varphi \leq M \int_0^\omega \varphi(s)ds, \quad \forall \varphi \in L^1_+(0,\omega),$$

for some constant $M > 0$ independent of φ. Using the results of Greiner (1984), we conclude that C_1 and C_2 are both weakly compact operators, and thus $BT_A(t)B$ is also weakly compact. □

Before establishing the local stability of the DFE, let us propose a formal definition of the basic reproduction number and make a remark.

Definition 1 (Basic reproduction number). *Consider the bounded linear operator $T_0 \in L(X)$ defined by $T_0 = \left(-\widehat{A}\right)^{-1} B$ and define the following quantity*

$$R_0 = r\left(T_0\right).$$

Remark 1. One has the following explicit expression for operator T

$$T_0\varphi = \int_0^\omega G_0(.,u)\varphi(u)du, \quad \forall \varphi \in X,$$

where we have set:

$$G_0(a,u) = \int_0^a e^{-\int_{a'}^a \mu_1(s)ds} \begin{pmatrix} 0 & \gamma_f\left(a',u\right) \\ \gamma_m\left(a',u\right) & 0 \end{pmatrix} da'.$$

The next Theorem establishes the local stability of the DFE.

Theorem 2. *Let Assumption 1 be satisfied. Then, the disease free equilibrium is locally asymptotically stable if* $R_0 < 1$, *and is unstable if* $R_0 > 1$.

To prove Theorem 2, we demonstrate two Lemma. We first notice that due to Theorem 1, the local stability of the DFE is related to the location of the real value $s\left(\widehat{A}+B\right)$ with respect to zero. Consider for each $\lambda \in \mathbb{R}$ the bounded linear operator $T_\lambda : X \to X$ defined by:

$$T_\lambda \begin{pmatrix} \varphi_f(a) \\ \varphi_m(a) \end{pmatrix} = \int_0^\omega G_\lambda(a,u) \begin{pmatrix} \varphi_f(u) \\ \varphi_m(u) \end{pmatrix} du,$$

where

$$G_\lambda(a,u) = \int_0^a e^{-(a-a')\lambda - \int_{a'}^a \mu_1(s)ds} \begin{pmatrix} 0 & \gamma_f(a',u) \\ \gamma_m(a',u) & 0 \end{pmatrix} da'.$$

Then, one has the following Lemma.

Lemma 2. *For each* $\lambda \in R$, *the operator* T_λ *is positive and compact. Moreover, for each* $\lambda \leq \lambda'$, *one has*:

$$T_{\lambda'}\varphi \leq T_\lambda \varphi, \quad \forall \varphi \in X^+.$$

Proof. The positiveness is obvious as well as the decreasing property with respect to λ (see for instance Marek, 1970). The compactness follows by noticing that for each $\lambda \in \mathbb{R}$, operator T_λ is regularizing in the sense that it maps the unit ball of X into a bounded set of $W^{1,\infty}(0,\omega;\mathbb{R}^2)$. □

Next, consider the map $R : \mathbb{R} \to [0,\infty)$ defined by

$$R(\lambda) = r(T_\lambda), \quad \forall \lambda \in \mathbb{R},$$

wherein for each $L \in \mathcal{L}(X)$, the quantity $r(L)$ denotes the spectral radius of L. Then, we obtain the following result.

Lemma 3. *Let Assumption 1 be satisfied. Then, the map* $\lambda \mapsto R(\lambda)$ *is continuous, decreasing and satisfies*

$$\lim_{\lambda \to \infty} R(\lambda) = 0,$$

$R(0) = R_0$ *(see Definition 1) and* $R(s) = 1$ *where* $s := s\left(\widehat{A} + B\right)$ *denotes the spectral bound of operator* $\widehat{A} + B$.

Proof. Let us first notice that the map $\lambda \mapsto T_\lambda$ is continuous from $\mathbb{R}$ to $\mathcal{L}(X)$. Since T_λ is compact for each $\lambda \in \mathbb{R}$, we conclude that $\lambda \mapsto R(\lambda)$ is continuous. As a consequence, due to Lemma 2, the map $\lambda \mapsto R(\lambda)$ is decreasing. Then, it is easy to check that

$$\lim_{\lambda \to \infty} \|T_\lambda\|_{\mathcal{L}(X)} = 0,$$

which implies that $R(\lambda) \to 0$ when $\lambda \to \infty$. It is also easy to check that

$$\lambda \in \mathbb{R} \cap \sigma_p\left(\widehat{A} + B\right) \iff 1 \in \sigma_p\left(T_\lambda\right),$$

where σ_p denotes the point spectrum. From this and the positivity, it follows that $R(s) = 1$. $\square$

A direct consequence of Lemma 3 is that if $R_0 < 1$, then $s = s\left(\widehat{A} + B\right) < 0$ and if $R_0 > 1$, then $s > 0$. This completes the proof of Theorem 2.

Let us conclude this section with a result on the existence of endemic equilibria.

Theorem 3. *Let Assumption 1 be satisfied. If* $R_0 > 1$, *then system* (17) *has at least one endemic stationary state, i.e. there exist* $(i_f^e, i_m^e) \in C \cap D(A) \setminus \{0\}$ *such that*:

$$\begin{cases} \frac{di_g^e(a)}{da} = \left(1 - i_g^e(a)\right)\left[\Lambda_g[i^e_{-g}](a) - \mu_1(a) i_g^e(a)\right], \\ i_g^e(0) = 0, \quad \forall g \in \{m, f\}. \end{cases}$$

Proof. Let us recall that as $R_0 > 1$, there exists $\lambda > 0$ such that $R(\lambda) = 1$. Let $\varphi = \begin{pmatrix} \varphi_f \\ \varphi_m \end{pmatrix} \in X^+$ be given such that $T_\lambda \varphi = \varphi$. Consider now the following fixed point problem: find $u \in \mathcal{C} \backslash \{0\}$ such that $u = \left(-\widehat{A} - \alpha\right)^{-1} (F + \alpha)\, u$. Since the operator $\left(-\widehat{A} - \alpha\right)^{-1}$ is positive and F is increasing, one obtains by setting $e = (1, 1)$ that

$$\left(-\widehat{A} - \alpha\right)^{-1} (F + \alpha)\, e \leq \left(-\widehat{A} - \alpha\right)^{-1} \alpha e \leq e.$$

On the other hand, for each $\varepsilon > 0$ one has

$$\begin{aligned}
&\left(-\widehat{A} - \alpha\right)^{-1} (F + \alpha)\, [\varepsilon\varphi](a) \\
&= \varepsilon \int_0^a e^{\alpha(t-a)} \begin{pmatrix} \left(1 - \varepsilon\varphi_f(t)\right) \left(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f\right) + \alpha\varphi_f \\ \left(1 - \varepsilon\varphi_m\right) \left(\Lambda_m[\varphi_f] - \mu_1(t)\varphi_m\right) + \alpha\varphi_m. \end{pmatrix} dt \\
&= \varepsilon \int_0^a e^{\alpha(t-a)} \begin{pmatrix} \Lambda_f[\varphi_m] - \mu_1(a)\varphi_f + \alpha\varphi_f - \varepsilon\varphi_f \left(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f\right) \\ \Lambda_m[\varphi_f] - \mu_1(t)\varphi_m + \alpha\varphi_m - \varepsilon\varphi_f(t) \left(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f\right) \end{pmatrix} dt
\end{aligned}$$

This implies that:

$$\begin{aligned}
&\left(-\widehat{A} - \alpha\right)^{-1} (F + \alpha)\, [\varepsilon\varphi](a) \\
= \;& \varepsilon\varphi(a) + \varepsilon \int_0^a e^{\alpha(t-a)} \begin{pmatrix} \varphi_f(t) \left(\lambda - \varepsilon \left(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f\right)\right) \\ \varphi_m \left(\lambda - \varepsilon \left(\Lambda_f[\varphi_f] - \mu_1(a)\varphi_m\right)\right) \end{pmatrix} dt.
\end{aligned}$$

As a consequence, if $\varepsilon > 0$ is chosen small enough so that

$$\varepsilon\varphi \leq e \text{ and } \varepsilon \begin{pmatrix} \Lambda_f[\varphi_m] - \mu_1(a)\varphi_f \\ \Lambda_f[\varphi_f] - \mu_1(a)\varphi_m \end{pmatrix} \leq \lambda e,$$

one obtains that

$$\left(-\widehat{A} - \alpha\right)^{-1} (F + \alpha)\, [\varepsilon\varphi] \geq \varepsilon\varphi.$$

The above inequality allows us to start a monotone iterative procedure to get complete the proof of the result. □

5 The impact of the dispersion of age differences between partners

In this section, we compute numerically the value of the epidemic threshold, R_0, as a function of the mean and the variance of the distribution of age

differences between partners.

5.1 Parameters and functions of the model

The model is simulated using parameters that are similar to those used in Anderson et al. (1992), including the age-specific mortality and fertility rates displayed in Figure 7.

Figure 7, about here.

For mortality, which is supposed to be similar for men and women, we use the Siler approximation, which is a parametric function that may be used to fit mortality data, and obtain that life expectancy at birth is 55.069 years. Concerning fertility, we obtain a Total Fertility Rate of 7.15. The demographic growth rate of the disease-free population can be computed using the formula given in equation (12), and is equal to $\gamma = 0.076$. Concerning the epidemiological parameters, we suppose that the infectiousness of the disease is age-independent, $\beta_g(a, a') = \beta_g$ and that a susceptible woman has a risk of infection when having a sexual contact with an infected man which is three times higher than those involving a susceptible man and an infected woman. The over-mortality rate of infected individuals is also supposed to be age independent, $\mu_1(a) = \mu_1$, and has been set such that the life expectancy (ignoring other causes of death) is 5 years.

Parameters are given in Table 2.

Sex ratio at birth, σ_f	0.5
Maximal age at death, ω	80
Minimal age of sexual activity, a_0	15
Lower and upper limits ages for fertility, a_1 and a_2	15 and 50
Over-mortality rate, μ_1	0.2
Infectiousness of the disease, β_f and β_m	0.3 and 0.1

Table 2
Parameters of the simulated model

The mean rates of partner change per year as functions of age are pictured at the endemic equilibrium. We use the mean values of ν and σ computed

among our sample of African countries, namely $\nu = 8.78$ and $\sigma = 2.62$. Concerning the parameter of function $c_f(a)$, we follow Anderson et al. (1992) by using $\eta = 3.4$ and $\eta = 5.7$, as depicted in Figure 8 and Figure 9, respectively.

Figure 8, about here.

Figure 9, about here.

Similarly, we compute the average prevalence at the endemic equilibrium as well as the age specific prevalence for men and women. Using $\eta = 3.4$ (Figure 10) and $\eta = 5.7$ (Figure 11), we obtain that the prevalence is equal to 1.5% and 5%, respectively.

Figure 10, about here.

Figure 11, about here.

Two notable features of these figures are that (i) women are proportionally more infected than men and (ii) the mean age of the infected population is lower for women than for men. Both conclusions are consistent with empirical evidence found in previous studies (e.g. Buvé et al.; 2001, Glynn et al., 2001; Gouws et al., 2008; UNAIDS, 2010, chapter 2).

5.2 Numerical results

The numerical simulations aim at evaluating the effect of both the mean and the variance of the age differences on the basic reproduction number. The latter is computed as the exponential of the speed of divergence (or convergence) of the linear system (21). We compute the basic reproduction number as a function of the mean age difference, ν, and the standard deviation, σ using two different values of the parameter of function $c_f(a)$ used in Anderson et al. (1992): $\eta = 3.4$ and $\eta = 5.7$. Results are displayed in Figure 12 and 13 respectively. Both figures clearly show that the epidemic threshold, R_0, is an increasing function of ν and an increasing and concave function of σ. The latter relationship becomes almost flat for values of σ greater than 3.

Figure 12 shows that if the women's rate of partner change is not too large, the standard deviation of age differences is a key parameter. Indeed, we find that if the standard deviation is small enough, the basic reproduction number remains below 1 whatever the value of the mean age difference. Conversely, if the standard deviation is large enough, the basic reproduction number is always greater than 1 even for very low mean age difference.

Figure 12, about here.

Figure 13, about here.

6 Conclusion

In this paper, we have analyzed the effect of a change in the dispersion of age differences between sexual partners on the endemic nature of the HIV epidemic. Once we established empirically that the distribution of age differences in Sub-Saharan Africa had not been modified since the onset of the epidemic, we went on to create an age- and gender-structured dynamic model. We characterized the stability of the epidemic equilibrium and showed that variance plays a crucial role in the determination of the stability properties of this equilibrium. Moreover, the mean age difference has barely any impact on the stability of the disease-free equilibrium if the variance is sufficiently high.

Importantly, our model constitutes a tool in order to evaluate the impact of the mean and the variance of the age differences distribution on the asymptotic dynamics of the HIV epidemics. We show that a larger variance increases the likeliness that the disease-free equilibrium is unstable, and consequently that the epidemic is endemic. This is an asymptotic result that is not necessarily connected with the prevalence rate at a given point in time. It cannot be tested using past prevalence rates, be used to forecast the dynamics of HIV in the next few years in African countries and is not able to

evaluate the various policies that have been launched in the countries of our sample. It rather argues that, everything equal, countries that have a large variance of age difference between partners should be particularly active in the fight against the spread of HIV within the population.

Moreover, in order to focus on the age differences, we have not considered other factors that may influence the dynamics of the epidemics. Our model builds a framework suitable for incorporating other contextual features that could allow for more realism. Especially, our model may be extended by describing precisely the different variables that influence the contact function between generations. We have, indeed, concentrated on the probabilities of having some infectious contacts for an exogenous number of contacts per age. Since this number appears to be important, we must seek to understand the underlying behaviors, which would be a promising avenue of research.

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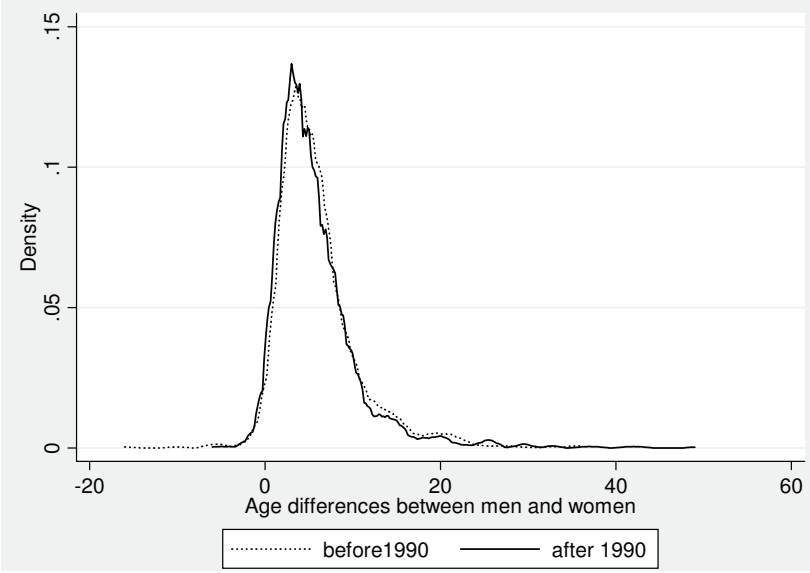

Figure 1: Distribution of age differences between men and women at marriage for Lesotho

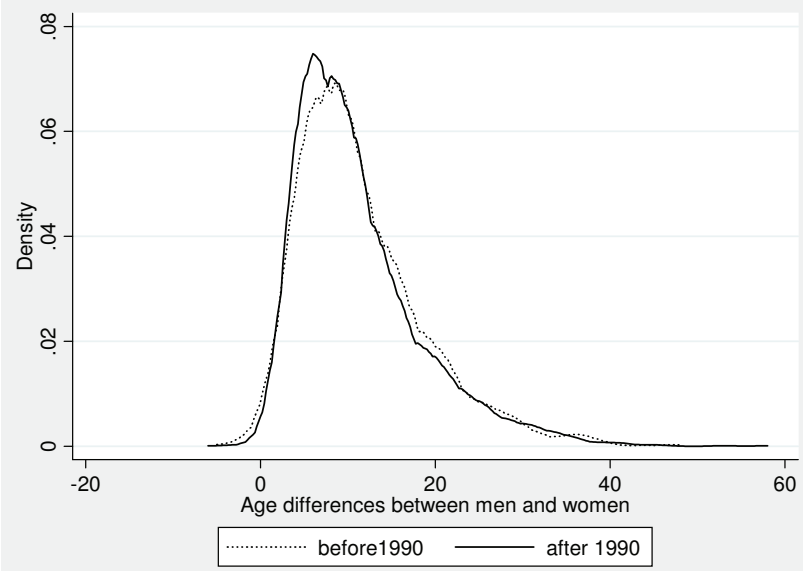

Figure 2: Distribution of age differences between men and women at marriage for Niger

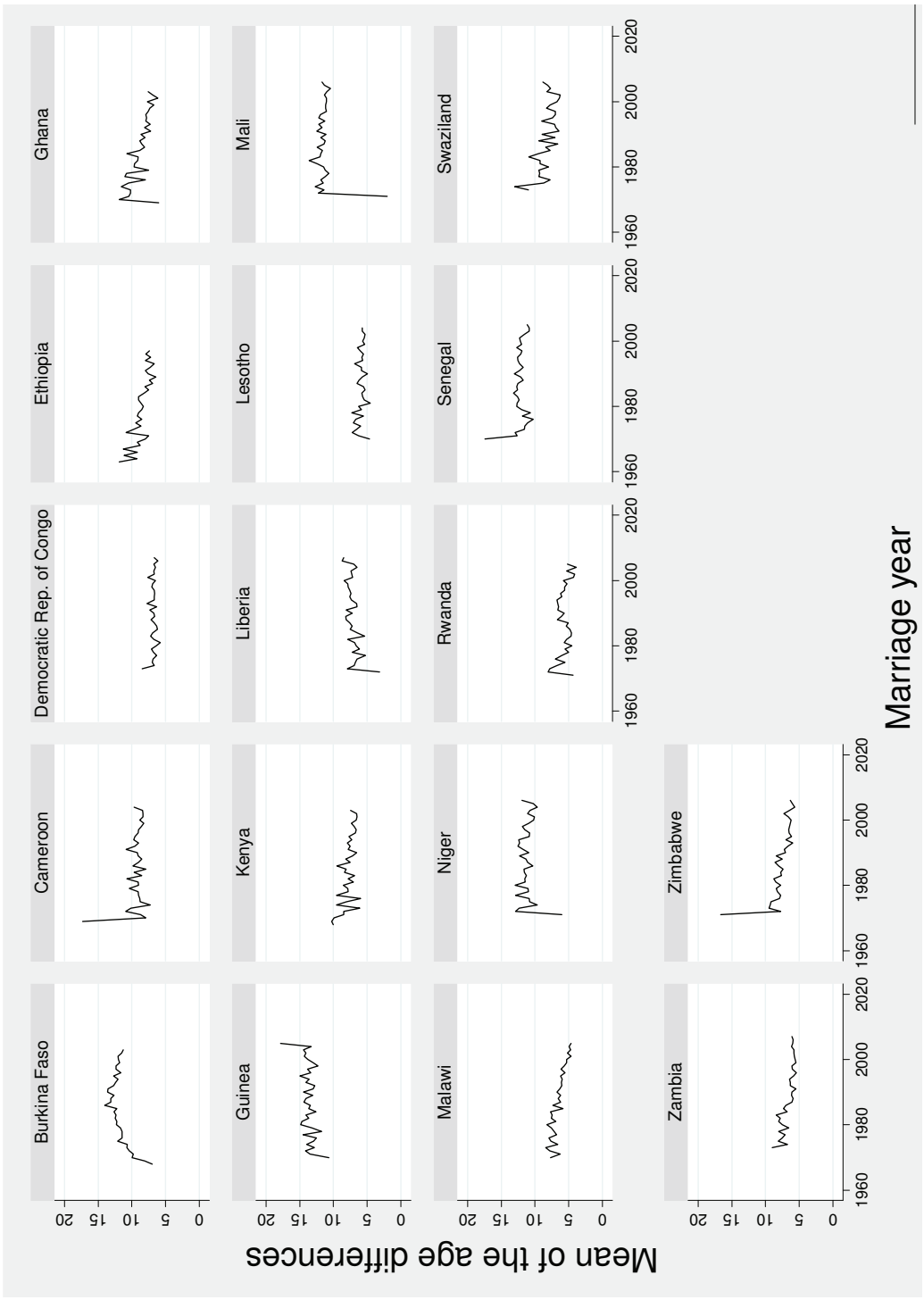

Figure 3: Dynamics of the mean of age differences

Figure 4: Dynamics of the coefficient of variation of the distribution of age differences

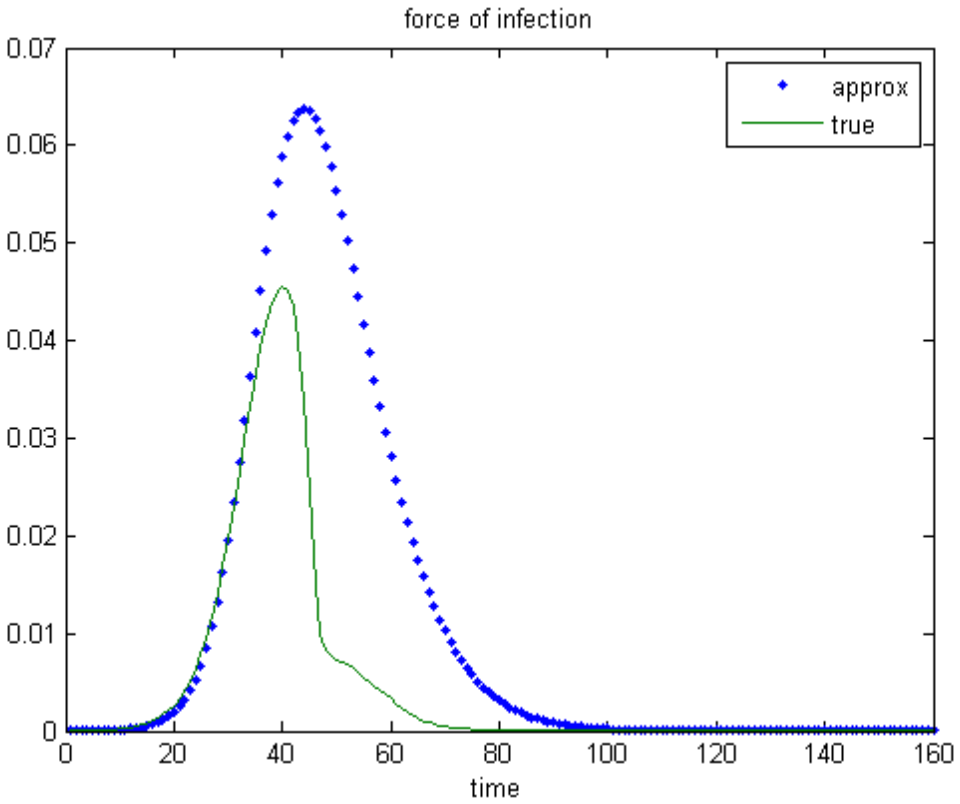

Figure 5: True and approximated forces of infection with asymptotic eradication

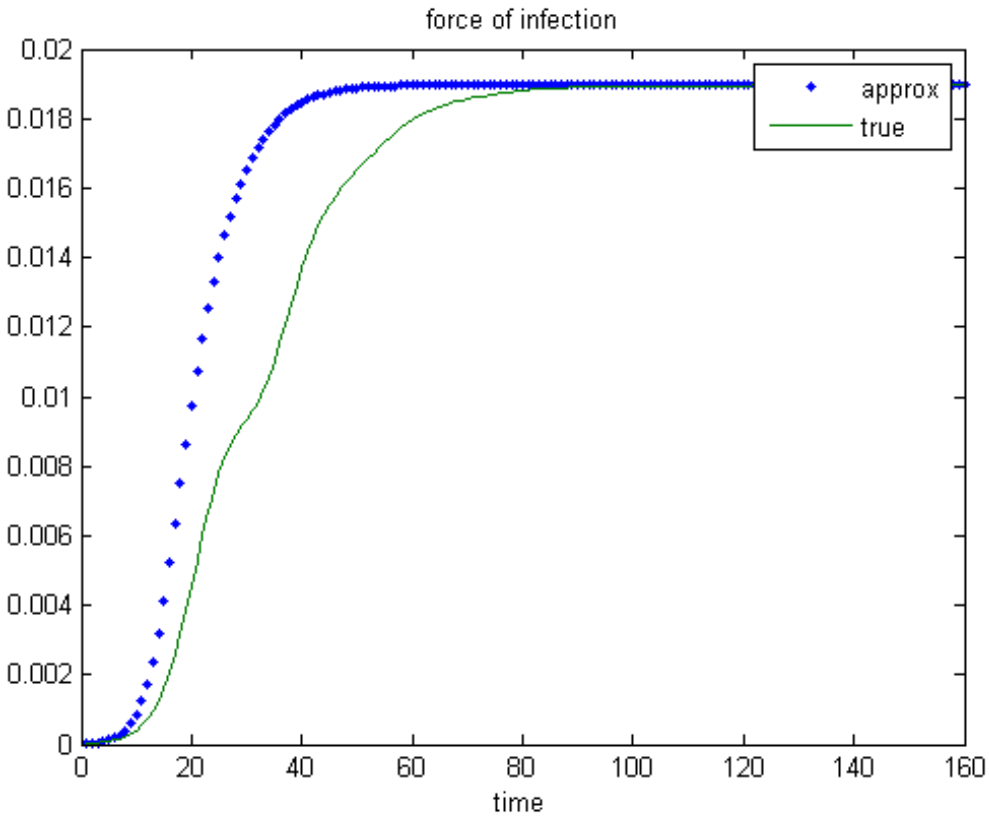

Figure 6: True and approximated forces of infection with an endemic equilibrium

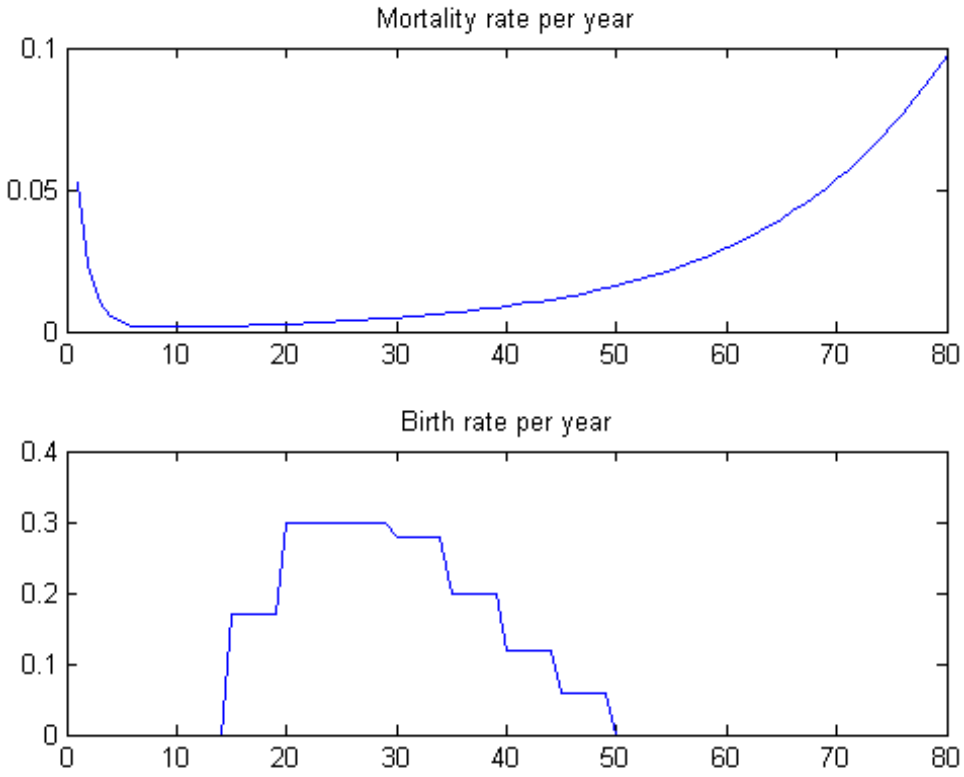

Figure 7: Mortality and fecondity as functions of age

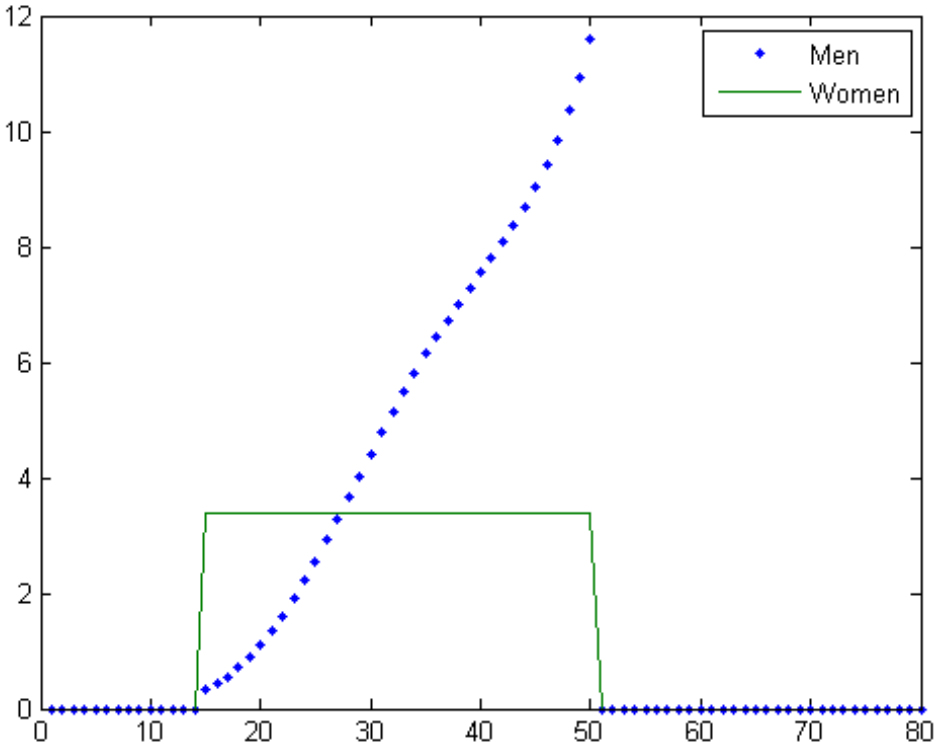

Figure 8: Mean rate of partner change as a function of age for $\eta = 3.4$

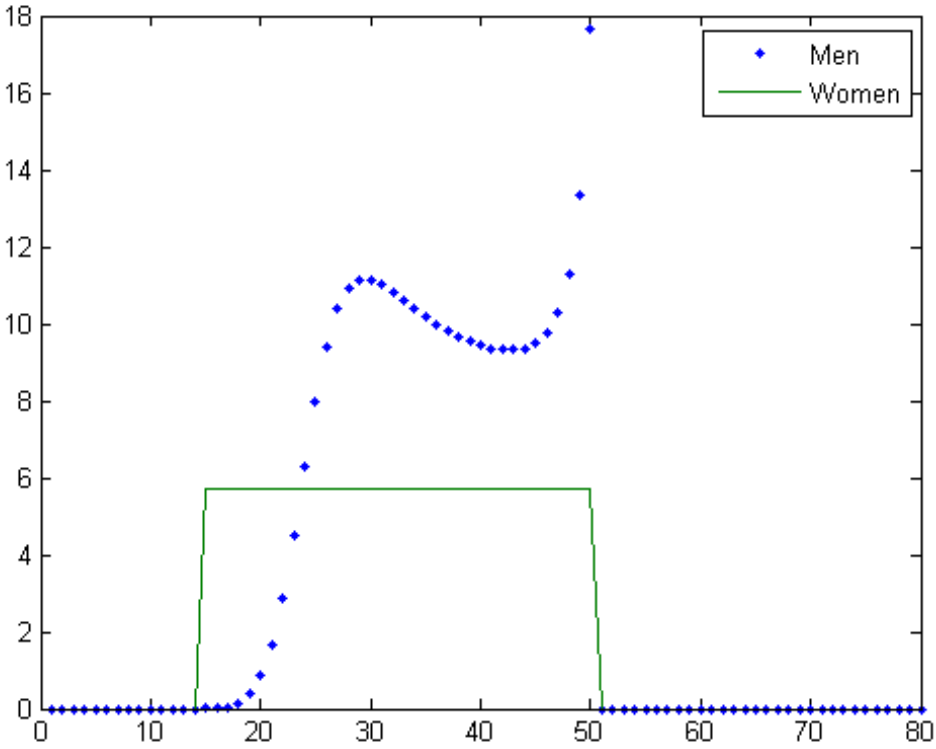

Figure 9: Mean rate of partner change as a function of age for $\eta = 5.7$

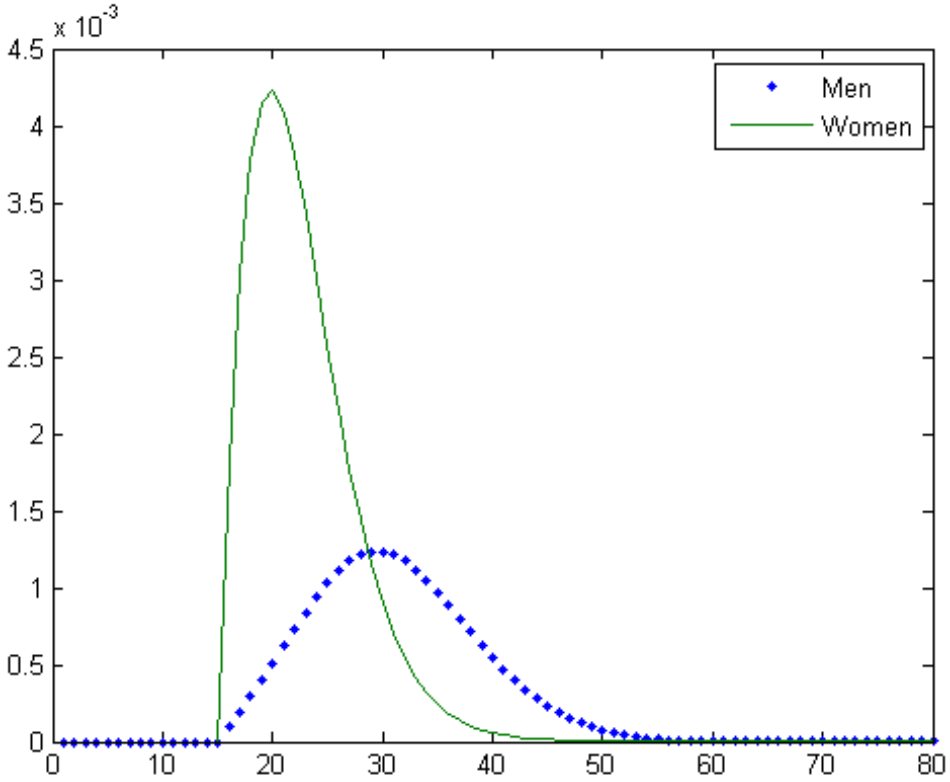

Figure 10: Prevalence rate as a function of age for $\eta = 3.4$

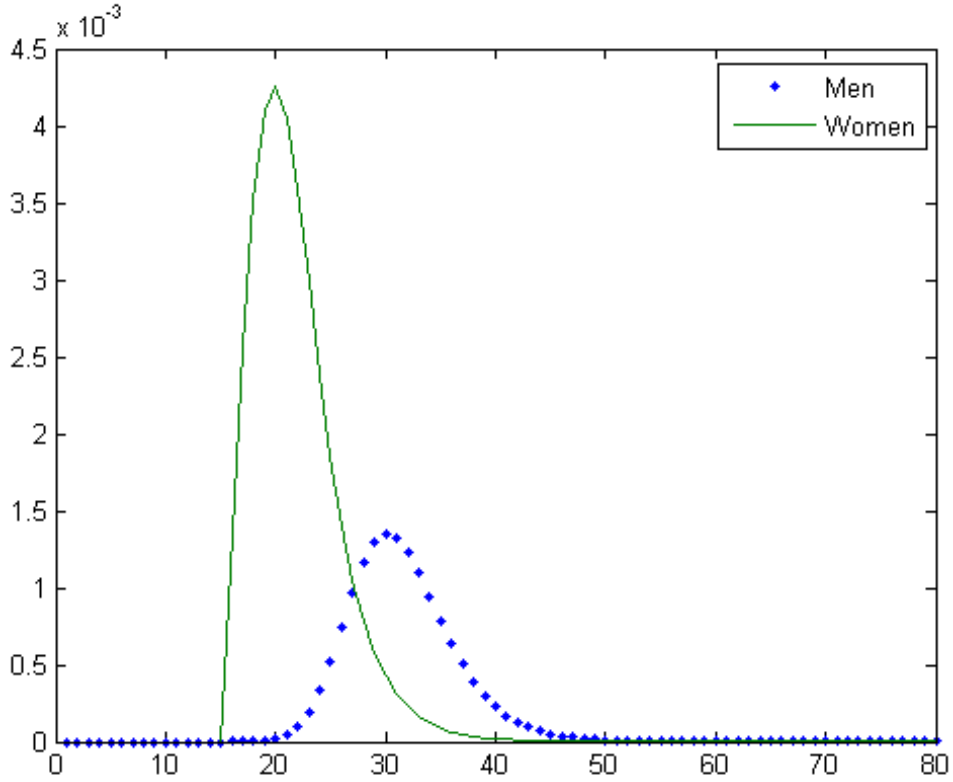

Figure 11: Prevalence rate as a function of age for $\eta = 5.7$

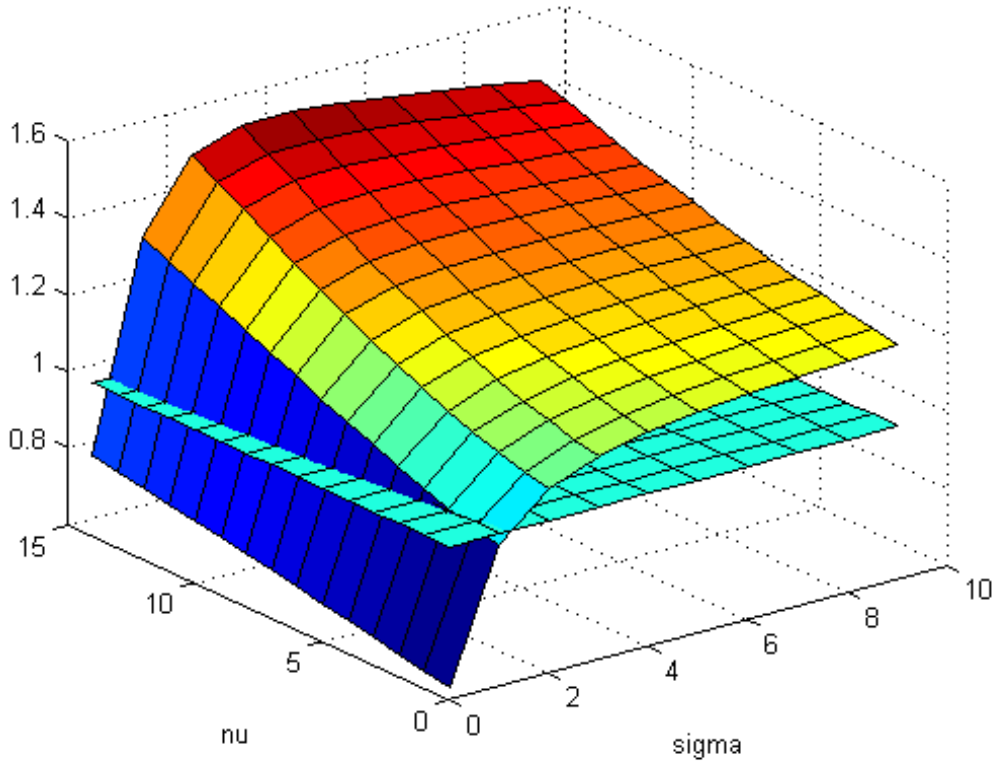

Figure 12: R_0 as a function of ν and σ for $\eta = 3.4$

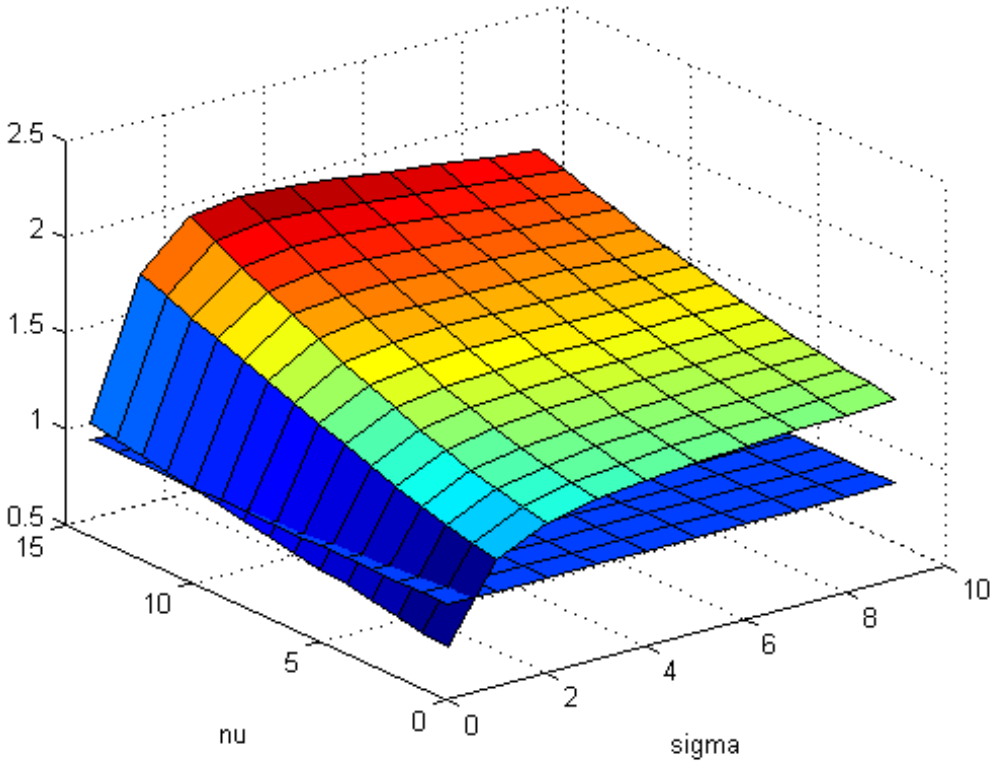

Figure 13: R_0 as a function of ν and σ for $\eta = 5.7$