The dispersion of age differences between partners and the asymptotic dynamics of the HIV epidemic

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The dispersion of age differences between partners and the asymptotic dynamics of the HIV epidemic

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In this paper, 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.

Keywords: epidemiology; population dynamics

1. Introduction

Thirty years after the discovery of the first confirmed clinical cases, the HIV epidemic is not yet under control. UNAIDS [28] estimates that worldwide 2.6 million adults and children were newly infected with HIV in 2009. The HIV epidemic affects Africa disproportionately, and especially sub-Saharan Africa, which accounts for 69% of the new infections (1.8 million in 2009, according to [28]). This paper is concerned with a demographic explanation of the differences in the evolution of the epidemic that have been observed across the 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 than in other parts of the world. Spijker [27] illustrated 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 ranged from 22.5% (South Africa, 1996) to 80.3% (Guinea, 1996), while
the same proportion ranged 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 has documented the particular frequency of age mixing in sub-Saharan Africa. Historically, age mixing has been commonplace in Africa [9] as a result of practices such as polygamy, the remarrying of widows and the premature marrying of young girls. Studies have shown that nowadays age differences persist throughout Africa [1] within both marital and non-marital partnerships and within both casual and regular relationships [2,14]. It has been found that about 40–50% of young girls are involved in partnerships with a partner who is 5–9 years older than them [14,19,20]. Greater age differences are also common as between 16% and 27% of young girls are in partnerships involving an age difference of 10 years or more [14,19,20]. This age mixing can also be observed for older women as the majority of the married women aged 15–44 years studied in [5] had a husband who was at least 6 years older than them. Studying male non-marital unions, Luke [23] found that 70% of the sampled men were 5 or more years older than at least one of their recent partners and 20% were 10 years or older.

Grounded on the empirical evidence that the HIV prevalence rate is much greater among young women than among young men (e.g. [8,12,13]), a growing body of research has examined age differences between partners as a potential risk factor of HIV infection. Some articles have documented the association between age difference between sexual partners and the increased risk of HIV infection [14,19]. The increase in risk is significant as documented by Kelly et al. [19], who found that 15–29-year-old women engaged in partnerships with a partner 5–9 years older or 10 years or older than them had a respective risk of infection of 1.1 and 1.28 times higher than their counterparts having partners 0–4 years older than them. Related papers have shown that people in partnerships involving large age differences are less likely to adopt safe practices than their counterparts, as women in long-term partnerships involving an age difference of more than 5 years [4] and men engaged in non-marital partnerships involving an age difference of 10 years or more [23] are less likely to use a condom than their counterparts.

The importance of age differences between partners in the diffusion and persistence of the epidemic was first brought up by Anderson et al. [1]. Through numerical simulations, the authors showed that the epidemic spreads more rapidly when there is an infectious contact between generations. An intuition has been proposed by Brouard [6], 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 be 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, the transmission of the disease to younger cohorts is potentially significant and hence the dynamics are likely to be endemic.

The objective of this paper 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 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 the 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 behaviour 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.

Second, we use this preliminary evidence 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 [1]
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 (DFE). 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, 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 analyse the effect of both the mean and the variance on $R_0$. Numerical computations show that the 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 be the mean age difference, the DFE will thus have a greater chance of being stable if the variance is small.

The paper is organized as follows. In Section 2, we present our empirical evidence. In Section 3, we describe the dynamic model, and in Section 4, we present our theoretical results. In Section 5, we develop and comment upon our numerical results. In Section 6, we give the conclusions.

2. Empirical evidence

In this section, we examine the distribution of age differences between spouses, especially the evolution of its mean and variance over time. In industrialized countries such as 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 [3]. 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 behaviour towards a reduction in the age difference since the onset of the epidemic as a self-protective mechanism. The data collected from the Demographic and Health Surveys conducted in sub-Saharan Africa suggest that this scenario is very unlikely.

In order to determine whether the AIDS epidemic has changed matching behaviours and shifted individuals’ preferences towards fewer age mixings, we used the distribution of age differences for married couples. As the time series of spousal age differences were 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 currently married were 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 were able to establish the empirical distribution of spousal age differences for each marriage year. The year in which the marriage took place was an indicator of the time period in which the individual made her decision about partner selection. Consequently, it provided more accurate information about individual behaviours than any cross-sectional analysis.

We restricted the sample to women who married when aged between 15 and 25 years for two reasons. First, it is the most common age interval at which women marry. In Lesotho, for instance, this sub-sample accounts for 90% of the total sample. Second, and more importantly, this sample restriction allowed us to rule out heterogeneities in the marital pattern from our analysis. Indeed, women who were married after reaching the age of 25 might have been previously married to someone else or might have different preferences in terms of partner selection compared with women who get married at a 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 drew 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 the most affected countries in sub-Saharan Africa, since 23.6% of its adult population was HIV infected in 2009 [28]. 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 [28].
Taking 1990 as a benchmark year, the two distributions were very similar, suggesting that there was no adjustment in the behaviour after the 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 were thus able to generalize our analysis using a large set of countries in order to test whether the distribution of the spousal age differences was constant over time.

We used 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 show the dynamics of the mean and the coefficient of variation, respectively, for each country of the sample.

There was no clear pattern suggesting a change in the distribution of the age differences over time, except for Ghana and Malawi, where a downward trend in the mean from the mid-1980s onwards could be observed. The mean of age differences decreased from 1985 in Ghana and from 1986 in Malawi, but these decreases were 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 in Malawi).

To test the stability of the distribution of spousal age differences over time, we used 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 the value of one if the marriage took place before 1990 and zero otherwise. The 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, that is, the mean (column 1) and the coefficient of variation (column 2).
Figure 4. Dynamics of the coefficient of variation of the distribution of age differences.

Table 1. Linear fixed-effects estimates (in parentheses: robust standard errors, clustered at the country level).

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Mean</th>
<th>Coefficient of variation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marriage year</td>
<td>−0.0311 (0.021)</td>
<td>0.0021 (0.002)</td>
</tr>
<tr>
<td>1 if marriage before 1990</td>
<td>0.0234 (0.244)</td>
<td>0.0467 (0.027)</td>
</tr>
<tr>
<td>Constant</td>
<td>70.6746 (41.161)</td>
<td>−3.315 (3.198)</td>
</tr>
</tbody>
</table>

These stylized facts suggest that controlling for country-specific effects, the distributions of spousal age differences are stable over time and that the onset of the epidemic disease does not imply any adjustment in preferences regarding the age difference between spouses. Therefore, grounded on this empirical evidence, in the next section, we 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. [1], which describes the spread of a sexually transmitted epidemic disease in a multi-group model. In our study, multi-group modelling was implemented due to the gender-specific variables used. 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 behaviours 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:

$$\frac{\partial S_g(t, a)}{\partial t} + \frac{\partial S_g(t, a)}{\partial a} = -S_g(t, a)\mu(a) + \lambda_g(t, a),$$  \hspace{0.5cm} (1)  

$$\frac{\partial I_g(t, a)}{\partial t} + \frac{\partial I_g(t, a)}{\partial a} = -I_g(t, a)\mu(a) + \mu_1(a) + S_g(t, a)\lambda_g(t, a),$$  \hspace{0.5cm} (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 modelled 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 Equations (1) and (2), we obtain

$$\frac{\partial N_g(t, a)}{\partial t} + \frac{\partial N_g(t, a)}{\partial a} = -\mu(a)N_g(t, a) - I_g(t, a)\mu_1(a).$$  \hspace{0.5cm} (3)  

Also consider 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

$$\lambda_g(t, a) = \int_{a_0}^{\omega} \beta_g(a, a')\rho_g(t, a, a')i_{-g}(t, a')\, da'.$$  \hspace{0.5cm} (4)  

Note that the probability of being infected has three components. The function $\beta_g(a, a')$ is the infectiousness of the disease, that is, the probability of being infected when having an infected partner of age $a'$. The component 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 $g$ [1]. 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 note 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
Therefore, the age-specific force of infection for men remains to be modelled. Following Anderson et al. [1], we assume that the mixing function is time independent, and reads as \( J_f(t, a, a') \). Then, we assume that, for women, the age-specific force of infection for women takes the following form:

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

(6)

Following Anderson et al. [1], we assume that function \( \rho_g(t, a, a') \) can be non-autonomous (i.e. time dependent) for one gender only, namely for men. Then, we assume that, for women, the mean rate of partner change for men is given by

\[
c_m(t, a') = \int_{a_1}^{a_2} c_f(a)J_f(a, a')N_f(t, a) \, da
\]

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

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

(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

\[
S_g(t, 0) = \sigma_g \int_0^{\omega} b(a)N_f(t, a) \int_{a_0}^{\omega} \rho_f(a, a') \, da' \, da,
\]

\[
I_g(t, 0) = 0,
\]

(8)

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

\[
S_g(0, a) = S_g^0(a),
\]

\[
I_g(0, a) = I_g^0(a),
\]

(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 that we considered consists of Equations (1), (2), (6), (7) and (8) and initial data (9).
3.2. A simplified model

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

\[
\frac{\partial N_g(t,a)}{\partial t} + \frac{\partial N_g(t,a)}{\partial a} = -\mu(a)N_g(t,a),
\]

\[
N_g(t,0) = \sigma_g \int_0^\omega b(a)N_f(t,a) \int_a^\omega \rho_f(a,a') \, da' \, da,
\]

\[
N_g(0,a) = N_g^0(a) \quad g \in \{m,f\}.
\]

(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 Equation (7). Indeed, if we assume that for each class of ages 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 Equation (7) can be approximated by the solution of the disease-free population (10). The latter system is well known and the equation for \( N_f \) is referred to as the Lotka–McKendrick equation. We refer the reader to the textbooks of Webb [30] and Iannelli [18] for a complete study of this kind of equations. The Lotka–McKendrick equation for \( N_f \) reads as

\[
\frac{\partial N_f(t,a)}{\partial t} + \frac{\partial N_f(t,a)}{\partial a} = -\mu(a)N_f(t,a),
\]

\[
N_f(t,0) = \sigma_f \int_0^\omega b(a)N_f(t,a) \int_a^\omega \rho_f(a,a') \, da' \, da,
\]

\[
N_f(0,a) = N_f^0(a).
\]

(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

\[
1 = \sigma_f \int_0^\omega b(a) e^{-\int_0^a (\mu(z)+\gamma) \, dz} \int_a^\omega \rho_f(a,a') \, da' \, da,
\]

one obtains that

\[
\lim_{t \to \infty} e^{-\gamma t} N_f(t,a) = \tilde{N}_f^0 e^{-\int_0^a (\mu(z)+\gamma) \, dz}
\]

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

\[
\lim_{t \to \infty} e^{-\gamma t} N_m(t,a) = \tilde{N}_m^0 e^{-\int_0^a (\mu(z)+\gamma) \, dz},
\]

where \( \tilde{N}_m^0 \) is related to \( \tilde{N}_f^0 \) through the following relation:

\[
\tilde{N}_m^0 = \frac{\sigma_m}{\sigma_f} \tilde{N}_f^0.
\]

(13)

This remark allows us to formally simplify the epidemic system under consideration and especially Equation (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

\[
\frac{N_f(t, a')}{N_m(t, a)} \approx \frac{e^{\gamma t} N_0}{e^{\gamma t} N_0} \frac{e^{-\int_0^t (\mu(z) + \gamma) \, dz}}{e^{-\int_0^t (\mu(z) + \gamma) \, dz}}
\]

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

\[
\lambda_m(t, a) = \frac{\sigma_f}{\sigma_m} \int_{a_0}^{a_0} \beta_m(a, a') c_f(a') J_f(a', a) e^{\int_0^a \mu(z) \, dz + \gamma (a - a')} i_f(t, a') \, da'. \tag{14}
\]

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

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

\[
J_f(a, a') = \frac{e^{-(a-a'+\nu)^2/2\sigma^2}}{\int_{a_1}^{a_2} e^{-(a-a'+\nu)^2/2\sigma^2} \, da} \quad \text{for } a, a' \in [a_1, a_2],
\]

\[
0 \quad \text{otherwise},
\]

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 [1] 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 present 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 Equation (4) with respect to time, while the dotted line corresponds to that of Equation (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 behaviour.

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 has some information on the Malthusian parameter of the total population, namely parameter \( \gamma \), and also on the sex-ratio parameters \( \sigma_g \). More specifically, using the above simplification and using the independent variables \( i_f(t, a) \) and \( i_m(t, a) \), the system that we will consider reduces by combining Equations (3) and (10) to the following one:

\[
\frac{\partial i_g(t, a)}{\partial t} + \frac{\partial i_g(t, a)}{\partial a} = (1 - i_g(t, a)) \left[ \lambda_g(t, a) - \mu_1(a)i_g(t, a) \right] \quad t > 0, \ a \in (0, \omega),
\]

\[
i_g(t, 0) = 0 \quad g \in \{m, f\},
\]

\[
i_g(0, \cdot) = i_g^0(\cdot) \in L^1(0, \omega; \mathbb{R}^+) \quad g \in \{m, f\}.
\]

In the above system of equations, \( \lambda_f \) and \( \lambda_m \) are, respectively, given by Equations (6) and (14).
4. Basic reproduction number

In this section, we derive some basic mathematical properties of Equation (15) together with Equations (6) and (14). The local dynamics are studied by analysing the spectral radius of a linear operator of a related system. The difficulty arises 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. However, spectral theory provides well-known tools to obtain the 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.
Then, consider the linear operator 

\[ A 1 \]

Assumption the following set of assumptions.

\[ L \alpha > X = \gamma \text{ for some functions } g \text{ wherein we have set for each } g \in \{ f, m \}, \]

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

for some functions \( \gamma_g \equiv \gamma_g(a, a') \) coming from Equations (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 \( a \) [21]. The above system of equations will be studied 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 \( \gamma_g \) belong to \( L^\infty((0, \omega) \times (0, \omega); \mathbb{R}^+) \).

As a consequence, \( \Lambda_g \) defined above becomes a bounded linear operator \( \Lambda_g : L^1(0, \omega; \mathbb{R}) \rightarrow L^\infty(0, \omega; \mathbb{R}) \). Next, the functional framework is defined as follows. Let us first recall 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 of a weak solution of system (17). Let \( \alpha > 0 \) be given such that

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

Then, consider the linear operator \( A : D(A) \subset X \rightarrow X \) 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 \( F : C \rightarrow X \) defined by

\[ F \left( \begin{pmatrix} \varphi_f \\ \varphi_m \end{pmatrix} \right) = \begin{pmatrix} (1 - \varphi_f)(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f) \\ (1 - \varphi_m)(\Lambda_m[\varphi_f] - \varphi_m) \end{pmatrix}. \]
Then, using $u(t) = (i_f(t, \cdot), i_m(t, \cdot))$, system (1) can be rewritten as the following abstract Cauchy problem:

$$\frac{du(t)}{dt} = Au(t) + F(u(t)), \quad t > 0,$$

$$u(0) = \varphi \in C.$$  \hspace{1cm} \text{(19)}

Note that given the choice of $\alpha$ (see Equation (18)), for each $(\varphi, \psi) \in C^2$ such that $\varphi \leq \psi$, one obtains that

$$F(\varphi) + \alpha \varphi \leq F(\psi) + \alpha \psi.$$  \hspace{1cm} \text{(20)}

Consequently, system (19) is equivalent to

$$\frac{du(t)}{dt} = (A - \alpha)u(t) + (F + \alpha)(u(t)), \quad t > 0,$$

$$u(0) = \varphi \in C.$$  \hspace{1cm} \text{(21)}

We directly deduce the following result.

\textbf{Lemma 1} \hspace{0.5cm} \text{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 $\{U(t; \cdot) : C \to C\}_{t \geq 0}$ such that for each $\varphi \in C$, the map $t \to U(t; \varphi)$ is a mild solution of system (19), that is,

$$U(t; \varphi) = T_A(t)\varphi + \int_0^t T_A(t - s)F(U(s; \varphi)) \, ds \quad \forall t \geq 0.$$  \hspace{1cm} \text{(22)}

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

$$\varphi \leq \psi \Rightarrow U(t; \varphi) \leq U(t; \psi) \quad \forall t \geq 0.$$  \hspace{1cm} \text{(23)}

\textbf{Proof} \hspace{0.5cm} \text{The proofs of similar results can be found in [7,11,31]. A key factor is given by the positivity of the semigroup generated by $A$, namely}

$$T_A(t)\varphi(a) = \begin{cases} \varphi(a-t) & \text{if } t < a \\ 0 & \text{if } t > a \end{cases} \quad \forall \varphi \in X.$$  \hspace{1cm} \text{(24)}

Let us now study the local dynamics in the neighbourhood of the so-called DFE that corresponds to the stationary solution $i_g(t, a) \equiv 0$. We now prove 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

$$\frac{\partial u_f(t, a)}{\partial t} + \frac{\partial u_f(t, a)}{\partial a} = -\mu_1(a)u_f + \Lambda_f[u_m(t, \cdot)](a),$$

$$\frac{\partial u_m(t, a)}{\partial t} + \frac{\partial u_m(t, a)}{\partial a} = -\mu_1(a)u_m + \Lambda_m[u_f(t, \cdot)](a),$$  \hspace{1cm} \text{(25)}

$$u_f(t, 0) = u_m(t, 0) = 0,$$

$$(u_f, u_m)(0, \cdot) = (u_f^0, u_m^0) \in X.$$  \hspace{1cm} \text{(26)}
In order to study this linear equation, we consider the linear operator $\hat{A} : D(\hat{A}) \subset X \to X$ and the bounded linear operator $B : X \subset X \to X$ defined by

$$D(\hat{A}) = D(A), \quad \hat{A} = \begin{pmatrix} -\frac{d}{da} - \mu_1(a) & 0 \\ 0 & -\frac{d}{da} - \mu_1(a) \end{pmatrix}$$

and

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

Then, by setting $u(t) = (u_f(t, \cdot), u_m(t, \cdot))$, systems (21) and (22) can be written as follows:

$$\frac{du(t)}{dt} = (\hat{A} + B)u(t), \quad t > 0, \quad u(0) = u_0 = \begin{pmatrix} u_0^f \\ u_0^m \end{pmatrix} \in X.$$ 

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

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

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

and

$$\omega_{\text{ess}}(\hat{A} + B) = -\infty, \quad \omega_0(\hat{A} + B) = s(\hat{A} + B) \in \sigma(\hat{A} + B). \quad (23)$$

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

**Proof** It is easy to see that

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

This proves that $T_{\hat{A}}(t)$ is a nilpotent semigroup and, therefore, we obtain that $\omega_{\text{ess}}(\hat{A}) = -\infty$. To prove the other part of Equation (23), using the results obtained by Greiner [15] as well as Voigt’s [29] perturbation result, we need to prove that for each $t > 0$, the operator $BT_{\hat{A}}(t)B$ is weakly compact in $X$. Recalling that $T_{\hat{A}}(t) = 0$ 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
\[
BT_A(t)B = \begin{pmatrix} C_1 & 0 \\ 0 & C_2 \end{pmatrix},
\]
where we have set
\[
C_1 \varphi_f = \int_0^\omega d\gamma_f(a, \cdot)1_{(t, \omega)}(a) e^{-\int_{a-t}^a \mu(s)ds} \int_0^\omega \gamma_m(a-t, a') \varphi_f(a') da',
\]
\[
C_2 \varphi_m = \int_0^\omega d\gamma_m(a, \cdot)1_{(t, \omega)}(a) e^{-\int_{a-t}^a \mu(s)ds} \int_0^\omega \gamma_f(a-t, a') \varphi_m(a') da'.
\]
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 \( \varphi \). Using the results of Greiner [15], 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 \( T_0 = (-\hat{A})^{-1}B \) and define the following quantity:
\[
R_0 = r(T_0).
\]

**Remark 1** One has the following explicit expression for operator \( T_0 \):
\[
T_0 \varphi = \int_0^\omega G_0(a, u) \varphi(u) du \quad \forall \varphi \in X,
\]
where we have set
\[
G_0(a, u) = \int_0^a e^{-\int_{a'}^a \mu(s)ds} \begin{pmatrix} 0 & \gamma_f(a', u) \\ \gamma_m(a', u) & 0 \end{pmatrix} da'.
\]

The next theorem establishes the local stability of the DFE.

**Theorem 2** Let Assumption 1 be satisfied. Then, the DFE is locally asymptotically stable if \( R_0 < 1 \) and is unstable if \( R_0 > 1 \).

To prove Theorem 2, we present two lemmas. We first note that due to Theorem 1, the local stability of the DFE is related to the location of the real value \( s(\hat{A} + B) \) 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
\[
G_\lambda(a, u) = \int_0^a e^{-(a-a')\lambda-\int_{a'}^a \mu(s)ds} \begin{pmatrix} 0 & \gamma_f(a', u) \\ \gamma_m(a', u) & 0 \end{pmatrix} da'.
\]
Then, one has the following lemma.
Lemma 2. For each \( \lambda \in \mathbb{R} \), the operator \( T_\lambda \) 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 positivity is obvious as well as the decreasing property with respect to \( \lambda \) (see, for instance [24]). The compactness follows by the fact that for each \( \lambda \in \mathbb{R} \), operator \( T_\lambda \) 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
\[
\lim_{\lambda \to \infty} R(\lambda) = 0,
\]
\[
R(0) = R_0 \text{ (see Definition 1) and } R(s) = 1, \text{ where } s := s(\hat{A} + B) \text{ denotes the spectral bound of operator } \hat{A} + B.
\]

Proof. Let us first note that the map \( \lambda \mapsto T_\lambda \) is continuous from \( \mathbb{R} \) to \( \mathcal{L}(X) \). Since \( T_\lambda \) 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
\[
\lambda \in \mathbb{R} \cap \sigma_p(\hat{A} + B) \iff 1 \in \sigma_p(T_\lambda),
\]
where \( \sigma_p \) denotes the point spectrum. From this and the positivity, it follows that \( R(s) = 1 \).

A direct consequence of Lemma 3 is that if \( R_0 < 1 \), then \( s = s(\hat{A} + B) < 0 \), 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 (1) has at least one endemic stationary state, that is, there exist \( (i^e_f, i^e_m) \in C \cap D(A) \setminus \{0\} \) such that
\[
\frac{d i^e_g(a)}{da} = (1 - i^e_g(a)) \Lambda_g[i^e_{g'}(a) - \mu_1(a)i^e_g(a)],
\]
\[
i^e_g(0) = 0 \quad \forall g \in \{m,f\}.
\]

Proof. Let us recall that as \( R_0 > 1 \), there exists \( \lambda > 0 \) such that \( R(\lambda) = 1 \). Let \( \varphi = \frac{\varphi^e}{\|\varphi^e\|} \in X^+ \) be given such that \( T_\lambda \varphi = \varphi \). Consider the following fixed-point problem now: find \( u \in C \setminus \{0\} \) such
that \( u = (-\hat{A} - \alpha)^{-1}(F + \alpha)u \). Since the operator \((-\hat{A} - \alpha)^{-1}\) is positive and \( F \) is increasing, one obtains by setting \( e = (1, 1) \) that

\[
(-\hat{A} - \alpha)^{-1}(F + \alpha)e \leq (-\hat{A} - \alpha)^{-1}\alpha e \leq e.
\]

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

\[
(-\hat{A} - \alpha)^{-1}(F + \alpha)[\varepsilon \varphi](a) = \varepsilon \int_0^a e^{(t-a)} \left( (1 - \varepsilon \varphi_f(t))(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f) + \alpha \varphi_f \right) \left( 1 - \varepsilon \varphi_m(\Lambda_m[\varphi_f] - \mu_1(t)\varphi_m) + \alpha \varphi_m \right) \, dt
\]

\[
= \varepsilon \int_0^a e^{(t-a)} \left( \Lambda_f[\varphi_m] - \mu_1(a)\varphi_f + \alpha \varphi_f \right) \left( -\varepsilon \varphi_f(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f) \right) \Lambda_m[\varphi_f] - \mu_1(t)\varphi_m + \alpha \varphi_m \right) \, dt.
\]

This implies that

\[
(-\hat{A} - \alpha)^{-1}(F + \alpha)[\varepsilon \varphi](a) = \varepsilon \varphi(a) + \varepsilon \int_0^a e^{(t-a)} \left( \varphi_f(t)(\hat{\lambda} - \varepsilon(\Lambda_f[\varphi_m] - \mu_1(a)\varphi_f)) \right) \varphi_m(\hat{\lambda} - \varepsilon(\Lambda_f[\varphi_f] - \mu_1(a)\varphi_m)) \, dt.
\]

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

\[
\varepsilon \varphi \leq e \quad \text{and} \quad \varepsilon \left( \Lambda_f[\varphi_m] - \mu_1(a)\varphi_f \right) \leq \lambda e,
\]

one obtains that

\[
(-\hat{A} - \alpha)^{-1}(F + \alpha)[\varepsilon \varphi] \geq \varepsilon \varphi.
\]

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

\[\blacksquare\]

5. The impact of the dispersion of age differences between partners

In this section, we numerically compute 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

We simulated the model using parameters that were similar to those used in [1], including the age-specific mortality and fertility rates displayed in Figure 7.

For mortality, which is supposed to be similar for men and women, we used 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 obtained 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 it was equal to \( \gamma = 0.076 \). Concerning the epidemiological parameters, we assumed that the infectiousness of the disease is age independent, \( \beta'(a, a') = \beta' \), and that a susceptible woman having a sexual contact with an infected man has a risk of infection which is three times higher than that observed in partnerships involving a susceptible man and an infected woman. The over-mortality rate of the infected individuals was also supposed to be age independent, \( \mu_1(a) = \mu_1 \), and was set such that the life expectancy (ignoring other causes of death) was 5 years.
Figure 7. Mortality and fecundity as functions of age.

Table 2. Parameters of the simulated model.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex ratio at birth, $\sigma_f$</td>
<td>0.5</td>
</tr>
<tr>
<td>Maximal age at death, $\omega$</td>
<td>80</td>
</tr>
<tr>
<td>Minimal age of sexual activity, $a_0$</td>
<td>15</td>
</tr>
<tr>
<td>Lower and upper limits of ages for fertility, $a_1$ and $a_2$</td>
<td>15 and 50</td>
</tr>
<tr>
<td>Over-mortality rate, $\mu_1$</td>
<td>0.2</td>
</tr>
<tr>
<td>Infectiousness of the disease, $\beta_f$ and $\beta_m$</td>
<td>0.3 and 0.1</td>
</tr>
</tbody>
</table>

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

The parameters are given in Table 2.

The mean rates of partner change per year as functions of age were considered at the endemic equilibrium. We used the mean values of $\nu$ and $\sigma$ computed in our sample of African countries, namely $\nu = 8.78$ and $\sigma = 2.62$. Concerning the parameter of function $c_f(a)$, we followed Anderson et al. [1] by using $\eta = 3.4$ and $\eta = 5.7$, as depicted in Figures 8 and 9, respectively.
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 $\eta = 3.4$.

Similarly, we computed 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 found the prevalences to be equal to 1.5% and 5%, respectively.

Two notable features of these figures are that they indicate 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 the empirical evidence reported in previous studies (e.g. [8,12,13, 28, Chapter 2]).

5.2. Numerical results

We performed numerical simulations to evaluate the effect of both the mean and the variance of the age differences on the basic reproduction number. The latter was computed as the exponential of the
speed of divergence (or convergence) of linear system (21). We computed the basic reproduction number as a function of the mean age difference, \( \nu \), and the standard deviation, \( \sigma \), using two different values of the parameter of function \( c_f(a) \) used in [1]: \( \eta = 3.4 \) and \( \eta = 5.7 \). The results are shown in Figures 12 and 13, respectively. Both figures clearly show that the epidemic threshold, \( R_0 \), is an increasing function of \( \nu \) and an increasing and concave function of \( \sigma \). The latter relationship becomes almost flat for values of \( \sigma \) 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 found that if the standard deviation is small enough, the basic reproduction number remains below 1 whatever be 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 a very low mean age difference.
6. Conclusion

In this paper, we have analysed the effect of a change in the dispersion of age differences between sexual partners on the endemic nature of the HIV epidemic. Once we empirically established 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 DFE 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 distribution of age differences on the asymptotic dynamics of the HIV epidemic. We showed that a larger variance increases the likelihood of the DFE being unstable and consequently of the epidemic being endemic. This is an asymptotic result that is not necessarily connected to 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 evaluate the various policies that have been launched in the countries of our sample. It rather argues that, everything being 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 precisely describing the different variables that influence the contact function between generations. We, 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 behaviours, which would be a promising avenue of research.

Notes

1. See [22] for a literature review on age mixing and possible reasons for it being widespread in the African context.
2. These surveys are publicly available at http://www.measuredhs.com.
References


