

Medium term dynamics and inequalities under epidemics

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Abstract

We are concerned by the dynamic demographic and economic consequences of epidemics, and to this end, we consider a general overlapping generations model which allows for several epidemic configurations. People live for three periods, successively as children, junior adults and senior adults. A junior adult has an exogenous number of children and is perfectly altruistic in that is he only cares for the survival of his children and the social position they will get. He invests in his own health and education, and in the health and education of his children. Because we take into account both child and adult mortality, we are in principle able to investigate the implications of epidemics for any age-mortality profile. First, we fully analytically characterise the short run and long run economic and demographic properties of the model, which allows us to do the same for the distributions of human capital and thus income. Second, we analyse the consequences of one-period long epidemics in two polar cases: an epidemic hitting only children Vs an epidemic only killing adults. Both are shown to have permanent demographic and economic effects. In contrast to epidemics only killing children, ‘adult’ epidemics are additionally shown to distort the income distribution in the medium run, creating more poverty. Such distributional effects vanish in the long run.

To analyse the medium term effects of HIV/AIDS, we assume that the epidemic hit junior adults, increase the number of deaths among children and reduces fertility. Then, we show that the size of the total population will decrease in the medium term, and that the share of the active population in the total population will also lower. In the active population, the proportion of people with a high level of human capital will decrease and the proportion holding a low level of human capital will increase. Finally output per worker and per capita will decrease.

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1. Introduction

Though the study of the economic effects of epidemics has always been of interest to many economists (see for example Hirshleifer, 1987), the more recent HIV/AIDS pandemic and its apparent massive demographic effects, especially in sub-Saharan Africa, has suggested an exceptionally abundant empirical and theoretical economic literature. Unfortunately, there is no common view of either the short or long run consequences of such an epidemic on economic growth so far.

On an empirical ground, the impact of AIDS on economic growth has been investigated in many studies³. Using cross-country data, Bloom and Mahal (1997) find a statistically insignificant coefficient on the AIDS variable and conclude that AIDS has had little impact on growth. The explanation seems to be that, by killing large numbers of people, AIDS is reducing population pressure on existing land and capital, thus raising labour productivity. It is possible that the 1980–1992 period examined in this study is too early in the epidemic to fully assess the effect of AIDS on growth. However, a more recent paper by Young (2005) comes to the even stronger conclusion that the AIDS epidemic will increase the per capita consumption and output of surviving people over the levels, which would have been reached without the epidemic.

On the other hand McDonald and Roberts (2006) apply similar but more elaborated econometrics than Bloom and Mahal to the more recent period 1984-1999. These authors work with an elaboration of the Solow model where production uses four factors: labour, physical capital, health capital and education capital. This model is estimated on a panel of 112 countries, over the period 1960 to 1998, with the data observed at 5 yearly intervals. Health capital per capita is measured by the life expectancy at birth or by the infant mortality rate. Both measurements give similar results. The authors conclude to a strong effect of the stock of general health on average income in developing countries. Average health itself depends on HIV/AIDS prevalence and the proportion of population at risk of malaria. They conclude that the marginal impact on income per capita of a 1% increase in HIV prevalence rate is minus 0.59% in Africa.

Such a disagreement on the growth effects of AIDS is also apparent in the empirical literature which studies the impact of the Spanish flu (see the excellent work of Brainerd and Siegler, 2003), and we will see that it goes with a similar discrepancy in the related theory.

However, disagreement does not extend to the demographic effects of AIDS in the medium term. HIV/AIDS primarily affects the most productive age group of men and women between 15 and 49 years—the main breadwinners and heads of households raising families and supporting the elderly—and their children. All studies conclude that the total population of countries severely hit by AIDS will be much lower in 20 or 25 years than if the epidemic had not taken place. Figure 1 (United Nations, 2004) presents the projected population size from 1995 to 2025, taking into account the demographic impact of AIDS as well as the hypothetical projected population without AIDS, for the 38 most affected African countries. In 1995, their population stood at 553 million, 6 million less than it would have been without AIDS. By 2025, the population of these 38 African countries will reach 983 million, that is, 156 million (or 14 per cent) fewer than without AIDS. This number can be decomposed between 98 million additional deaths between 1995 and 2025, and 58 million children who will not be born because of the early deaths of women of reproductive age. In the most severe case, Botswana, where currently more than one in three adults is HIV positive, population is expected to decline within the next few years

Death affects more the adult population of working age than younger or older populations. However, the same study by the United Nations writes “Approximately one fourth to one third of children born to HIV-positive women are likely to acquire infection from their mothers. Paediatric HIV infection is expected to have a substantial impact on mortality during infancy and childhood, particularly among older children (above age one)... Children who acquire the HIV virus from their mothers during childbirth or breast feeding usually do not survive long enough to enrol in school.... Children die young from HIV owing to mother-to-child transmission and to the weakened ability of infected mothers to care for their infants and young children”. Cohen (1998) notes: “Child mortality rates are already higher today than they would have been without AIDS in some high prevalence countries. Thus child mortality rates are estimated as being 75% higher in Botswana in 1996. By the year 2010 child mortality rates are expected to be twice as high in Botswana, 4 times greater in Zimbabwe and about twice as high in Zambia and Malawi” (Figure 2).

Finally, Figure 3 (United Nations, 2004) displays the age pyramid of Botswana, the country with the highest adult HIV prevalence, in 2000 and as projected for 2025, with and without AIDS. In 2000, the impact of AIDS on the age structure of Botswana’s population is still

³ For an excellent review see United Nations (2004).

mild. But by 2025, more than half of the potential population aged 35-59 would have been lost to AIDS. In comparison, one third of the population aged less than 15 years old is expected to be lost to AIDS. Cohen (2002) notes that for Malawi one of the most important consequences of AIDS is a change in the age pyramid of the population, with a narrowing of the distribution in the working age population, and a consequent problem with respect to age dependency, with larger numbers of youthful and elderly dependents.

At the theoretical level, the discrepancy in the evaluation of the effects of an epidemic on economic growth is especially neat in the benchmark growth models, as clearly reflected in Barro and Sala-I-Martin (1995), chapter 5. Two models are considered in this chapter. Both use two production factors: physical capital and human capital. The economy is on a reference balanced growth path when an epidemic, which takes place at date 0, destroys a part of the human capital, but leaves physical capital intact. In the first model, the sector producing the human capital uses the same technology as the sector producing consumption goods and physical capital; it is therefore a one-sector model. However, investments in both factors must be non-negative (irreversibility). Then the epidemic creates an imbalance between the two factors. The economy reacts by setting the investment in physical capital to zero, but also by reducing households' consumption. The correction of the imbalance and the reduction in consumption increase the growth rate of the production of the physical good above its reference value. Of course, this growth rate will decline monotonically over time until it reaches its initial value.

The second case considered by Barro and Sala-I-Martin is the celebrated Lucas-Uzawa model (see for example Lucas, 1988). Education, the sector producing human capital, only uses this factor as input. The production of consumption good and physical capital uses both factors. Then, an epidemic increases the scarcity of human capital, and the wage rate. The high cost of operation for the education sector will motivate people to allocate human capital to production of goods, rather than to education, the sector that produces the relatively scarce factor. This effect tends to retard the economy's growth rate. The growth rate of gross output (including the production of new human capital) will decrease at the time of the epidemic, then it will increase monotonically over time until it reaches its reference value.

Hence, the predictions of the two-sector model for economic growth are exactly the opposite of those of the one-sector prototype. Incidentally, the latter delivers the same prediction as the

even more standard Solow model. In such a model, the initial effect of an epidemic is to increase the amount of capital per worker and output per worker. After the initial shock, the economy will gradually converge back to its steady-state, and the growth rate of output per worker will be less than its steady state value during this transition. Despite the divergent predictions, all these textbook models have some common characteristics:

(i) The disembodied nature of human capital

All the models listed above consider that human capital, which aggregates the education level of the population and sometimes its health status, is similar to physical capital. However, human capital (education and health) is embodied in individuals, inducing possible big differences concerning the mechanisms of investment in physical capital. For instance, the death of a child or an elderly has no effect on the level of the human capital used in production. Its economic effects will be very different from the death of workers in their twenties or thirties, which brings the destruction of human capital progressively brought up in them through child rearing, formal education and learning on the job⁴.

(ii) The importance of parental decisions in education and health expenditures

Another specificity of the human capital (education and health) is that the amount of it embodied in a person strongly results from decisions taken by his parents. Bowles and Gintis (2002) quote a series of empirical results for the United States. A son born in the highest income decile has a probability of 22.9% to reach the same decile and a probability of 2.4% to reach the lowest income decile. A son born in the lowest income decile has a probability of 1.3% to reach the highest decile and a probability of 31.2% to reach the lowest decile. Grawe and Mulligan (2002) review cross-country evidence showing that countries with lower public provision of human capital experience smaller

⁴ Brainerd and Siegler make an interesting remark, which unfortunately they do not use in their quantitative analysis: « In a typical influenza epidemic, the majority of the victims are young children and the elderly, giving the age profile of mortality a distinct ‘U’ shape. A distinguishing characteristic of the 1918 epidemic was that it disproportionately killed men and women with ages 15 to 44, so that the age profile of mortality instead followed a ‘W’ pattern. ... For both whites and non whites, the male mortality rate for those ages 15 to 44 exceeded the female mortality rate by 50-75 per cent in 1918, in contrast to the non-epidemic years in which the death rates by gender are virtually identical. The death rate for non whites also exceeds that of whites, although the ‘W’ pattern characterises the age-specific death rates of both races”.

intergenerational mobility. For instance, less developed countries exhibit strong intergenerational transmission. The connection between the absence of intergenerational mobility and education is well documented. Bowles and Gentis show that this situation can also be linked to the health of children, which is itself a function of their parents' income (see also Case, Lubotsky and Paxson, 2001).

One important implication of property (ii) is the following: when young adults die, not only do they reduce the amount of labour and human capital used in production, but they also leave orphans behind them. To show how this effect can be disastrous, we can quote the following extract of an article published by *The Economist* (2003) ``... one-in-ten sub-Saharan children is now an orphan. A third of these are the result of AIDS. Orphaning rates above 5% worry UNICEF because they exceed the capacity of local communities to care for parentless children. So do places such as Zambia, where almost 12% of children are AIDS orphans.... Orphans tend to be poorer than non orphans, and to face a higher risk of malnutrition, stunting and death — even if they are free of HIV themselves. Orphans are less likely to attend school because they cannot afford the fees but also because step-parents tend to educate their own children first”. Case, Pakson and Ableidinger (2004) give interesting complements to this view. Orphans live in foster families who discriminate against them and in favour of the children of the family head. The probability of the school enrolment of an orphan is inversely proportional to the degree of relatedness of the child to the household head. Gertler, Levine and Martinez (2003) show that parental loss does not operate only through a reduction in household resources. Parental presence, including the loss of mentoring, the transmission of values and emotional and psychological support, plays an important role in investment in child human capital.

The report by the United Nations (2004) adds that the health and nutritional status of orphans are also likely to suffer. In a study of 312 communities in 13 Indonesian provinces, it was found that children whose mothers had died were more likely to die than children who had not lost a parent. Bereaved children were generally less healthy than children whose parents had lived. In a study of children's health in the north-western United Republic of Tanzania, it was found that adult deaths led to increased morbidity and reduced height for age of children under five in the household. Finally the report notes: “The effects of lowered investment in the human capital of the younger generation will affect economic performance over future decades, well beyond the time frame of most economic analyses”.

Our paper deals with the economic and demographic effects of AIDS in the medium term that is one generation after the time when the epidemics started and in a period when the number of death has lowered⁵. Few studies investigate this horizon, and prefer focusing on the short term or the long run. We will especially be interested by the modifications in inequality that AIDS will induce in the medium term.

Our paper develops a discrete time, perfect foresight endogenous growth model of a small open economy which incorporates the two crucial aspects of human capital formation mentioned above. Hereafter, we shall take human capital in the broad sense of education **and** health. The demographic and economic properties of the model are fully analytically investigated, which is yet a contribution to the literature as it will be clear in the next review section. In our model, people live for three periods, successively as children, junior adults and senior adults. A junior adult has an exogenous number of children and is perfectly altruistic that is he only cares for the survival of his children and the social position they will get. He invests in his own health and education, and in the health and education of his children. The probability for a child to reach a high level of human capital is independent of the levels of the human capital of his parent, under the conditions that he survives and that his parent survives and is able to bring him up. Thus, we have eliminated the traditional channel of the cultural heritage to focus on alternative channels which work through education and health and investments in both. The probabilities of survival of a child and of a junior adult depend on the amounts of money spent by the junior adult for his own human capital and for the one of his children.

The credit market is incomplete: parents cannot finance spending on their children by borrowing against their higher expected income, which will result from this spending. So, health and education spending and the probabilities of survival will be low if parents are poor. Moreover, if a parent dies and if his children become orphans, their probabilities of survival will be lowered. Finally, we will assume that an orphan has a lower probability to reach a high level of human capital than a child brought up by living parents.

An interesting feature of our paper is to distinguish the mortality of children from that of parents, each depending on specific education and health spending. Investing in his children human capital will increase their survival rate until they reach the age when they can

⁵ The United Nations report gives the estimated and projected excess deaths due to AIDS in the 53 countries where the rates of prevalence are the highest, from 1990 to 2025. The number of deaths increases at an increasing rate until 2003. Then it increases at a decreasing rate, reaches a maximum in 2022 or 2023, and decreases afterward.

procreate, and so will contribute to increase the growth rate of population. Of course the ratio between the active and inactive population that is between junior adults and children will depend on the amounts of money spent on the education and health of these two kinds of population.

Another feature of the paper is to consider a new dimension of inequality, namely inequality in front of death. Inequality between children has several causes. First, the children of less educated parents who have survived and who bring them up have a higher probability of dying before growing adults because their parents spend less on their health and education. Secondly, less educated parents spend less on their own education and health and have a higher probability to die and to be unable to bring their children up. Orphans have a higher probability of dying young, and if they survive of being less educated.

We shall define an epidemic as an increase in the death rate of a generation of people lasting for only one period. We consider two kinds of epidemics. The first one kills a given proportion of children while the second kills a proportion of junior adults. We shall show that the two epidemics have completely different dynamic demographic and economic implications. Ultimately, we will build a relatively simple but quite global economic theory of epidemics with embodied human capital and with a comprehensive accounting of inequality in front of death.

The paper is organised as follows. The second section reviews the related literature to clarify our contributions. The third section presents the model and its short run equilibrium. The model has a property of decomposability. The equilibrium values of the choices of the agents can be computed first. These values contribute to the determination of the sizes of the various populations, but are independent of them. The fourth section is devoted to the transitory dynamics and the long run equilibrium of demographic variables. The fifth section investigates the economic and demographic effects of an epidemic hitting either children or junior adults. Then it tries to evaluate the medium-terms effects of AIDS by combining the effects of these two kinds of epidemic and of a reduction in fertility. The sixth section concludes.

2. Relation to the existing literature

There are several papers developing computable general equilibrium models to investigate the effects of AIDS, and giving an important role to the increase in the number of orphans. For

example, Bell, Devarajan and Gersbach (2003) develop such a model applied to the South African case. The authors emphasise the formation of human capital and transmission mechanism across generations and conclude to a very negative effect of the epidemic on long-run growth, with a large proportion of families and their offspring falling in a poverty trap. So, a transitory shock can have permanent effects.

A similar model was developed by Corrigan, Glomm and Mendez (2004), who also conclude that the growth effects of AIDS are large. The policies investigated by the authors are to make AIDS patients well enough to live more or less normal and productive lives, which would include being more able to care for their children. However, the authors conclude that such policies such as changing subsidies for AIDS related medical care have relatively small growth effects. In their paper, children receive a different level of education if their parents are healthy or ill. However, when they grow up and become adult, the available human capital of their cohort is reallocated in an egalitarian way between all its members. This redistribution, which forsakes the assumption of embodiment of the investment in education, simplifies the simulation of the model, but contradicts the optimisation program of the parents that does not anticipate it. In the paper by Bell, Devarajan and Gersbach children are ranked by increasing human capital then divided into a finite number of classes. The reallocation of the human capital occurs inside each of these classes. Thus, these authors approximate a continuous distribution by a discrete distribution. As this approximation can be as precise as desired, this solution is more acceptable than the previous one.

Our paper takes a broader perspective: we are concerned by the dynamic and long run demographic and economic consequences of epidemics, and to this end, we consider a general model which allows for several epidemic configurations. In particular, because we take into account both child and adult mortality, we are in principle able to investigate the implications of epidemics for any age-mortality profile. In order to derive fully analytical results, we shall precisely tackle two polar cases: an epidemic hitting only children Vs an epidemic only killing adults. The comparison of these two cases will be eloquent enough. Moreover, our treatment of human capital formation meets the two crucial characteristics outlined in the introduction (roughly, embodiment and ‘paternalism’).

Finally it should be noted that the papers focusing on AIDS usually comment on the changes in the distributions of human capital and income possibly following the epidemic although

they do not fully investigate them. The only theoretical paper we know, which investigates the links between health spending, mortality and the persistence of inequality across generations, is by Chakraborty and Das (2005). These authors base their analysis of the persistence of poverty on the fact that poor parents invest less in their own health and so have a high probability of dying. Thus, they save little and leave to their children a small bequest if they survive and a still smaller bequest if they die. The paper assumes that parents only care for the health of their children if they are themselves alive when their children grow. However, parents cannot buy annuities against the saving they will leave in the case of their premature death (so, in this situation, children get an unplanned bequest). An extension of the paper introduces the possibility of investing, not only in the health of parents, but in the education of children too. The productivity of labour depends on both these investments. Nonetheless, these authors do not consider investments in the health of children nor their survival probability.

3. The model: behaviour of the agents and temporary equilibrium

We consider a discrete time, perfect foresight dynamic model of a small open economy. People live for three periods, successively as children, junior adults and senior adults. We will start by examining the choices of a junior adult in an given period denoted t . In a second paragraph we will describe the temporary equilibrium of the model in this period. To ease the exposition and to be able to bring out a fully analytical characterization, we shall refer to a single good, health care. The latter should be taken in the much broader sense of any investment raising human capital (including education).

3.1. The choices of a junior adult

A junior adult enters period t with an endowment in human capital h . Healthcare is the only good existing in the economy. It is produced by firms, which use human capital as their unique input and which operate under constant returns. We will assume that the productivity of human capital is equal to 1 and that firms make no profit. Thus, h can also be interpreted as the earnings of the agent. The agent sets his saving s and his investment in health l for the period, under the budget constraint

$$(1) \quad h = s + l$$

Spending on health has an effect on the lifetime of the agent. His probability of being alive in period $t+1$ (as a senior adult) is $\pi(l)$. At the end of period t the agent will have an exogenous number n of children. Senior adults receive no wages. This assumption will simplify the model in directions that we are not very interested to investigate. The agent will invest e_{+1} in the health of each of his children. The probability for each of them to be alive at the beginning of period $t+2$ will depend on this investment. If the agent is alive in period $t+1$ and can take care of his children, this probability will be $\lambda(e_{+1})$. If he is dead and if his children are orphans, this probability will be $c\lambda(e_{+1})$, with $0 < c \leq 1$. The saving of the agent in period t , s , is lent on the international capital market at the exogenous and constant capitalisation rate $R > 1$. The budget constraint of the agent in period $t+1$ is:

$$(2) \quad Rs = ne_{+1}$$

We notice that the amount invested by the agent in the health of his children will be the same if the agent dies or stays alive at the end of period t . This investment is equal to the capitalisation of the saving made in period t . The intertemporal budget constraint of the agent is

$$(3) \quad Rh = lR + ne_{+1}$$

To simplify the model we will assume that human capital can take only two values: h^- and h^+ , with: $0 < h^- < h^+$. We will assume that a child who has living parents and who stays alive has a probability p of obtaining a human capital of h^+ and a probability $1-p$ of obtaining a human capital of h^- . An orphan who stays alive has the probability q of obtaining the high level of human capital and $1-q$ of obtaining the low level of human capital. We assume that $0 \leq q < p \leq 1$.

Our junior adult has the following utility function in period t

$$(4) \quad U \equiv n\lambda(e_{+1})\{\pi(l)v[p(h^+ - h^-) + h^-] + [1 - \pi(l)]vc[q(h^+ - h^-) + h^-]\}$$

The junior adult is wholly altruistic. His utility only depends on the expected human capital accumulated by his children who will reach the adult age. If the junior adult reaches the age of senior adult, he will bring his children up, which will increase their probability of survival and their expected levels of human capital. vh^+ (vh^-) represents the satisfaction a child brings to his parent when he reaches the adult age with the level of human capital h^+ (h^-).

We assume that $\nu > 0$. When the child dies this satisfaction is 0. We will introduce the following notations

$$(5) \quad r_1 = \nu[p(h^+ - h^-) + h^-], \quad r_2 = \nu c[q(h^+ - h^-) + h^-] \quad \text{and} \quad r = r_1 / r_2 - 1.$$

The utility function of our junior adult in period t becomes, after having removed a constant multiplicative term, $U \equiv \lambda(e_{+1})[\pi(l)r + 1]$. r represents the premium in satisfaction brought by children, when their parent stays alive, or if one prefers, the utility for parents of staying alive. In this case, the probability of survival of each child is higher (by a factor $1/c$) and his expected level of human capital is higher too. r , is an increasing function of the inequality in earnings, $(h^+ - h^-) / h^-$, which is expected for the next period. In the following exercises of comparative static, we will assume that h and r can change independently. Finally, our junior adult must solve in period t the program

$$(6) \quad \underset{l, e_{+1}}{\text{Max}} \lambda(e_{+1})[\pi(l)r + 1]$$

$$Rh = lR + ne_{+1}$$

$$l, e_{+1} \geq 0$$

Before solving this program we must give precise specifications of the survival functions:

$$(7) \quad \lambda(e_{+1}) = (Ae_{+1})^{1-\alpha} / (1-\alpha), \quad \text{if } 0 \leq Ae_{+1} \leq (1-\alpha)^{1/(1-\alpha)}$$

$$\lambda(e_{+1}) = 1, \quad \text{if } Ae_{+1} \geq (1-\alpha)^{1/(1-\alpha)}$$

$$(8) \quad \pi(l) = (Bl)^{1-\beta} / (1-\beta), \quad \text{if } 0 \leq Bl \leq (1-\beta)^{1/(1-\beta)}$$

$$\pi(l) = 1, \quad \text{if } Bl \geq (1-\beta)^{1/(1-\beta)}$$

with: $0 < \beta, \alpha < 1$, $A, B > 0$. In the rest of the paper we will assume that we are always inside the intervals where both functions are strictly increasing. Deaton (2003) notices that health spending, the health state and the longevity of an individual are increasing and concave functions of his income: for instance the probability for somebody of dying between the ages of 50 and 60 is a decreasing convex function of his income. This concavity is a possible explanation of the impact of inequality on the average health state in a country, and it implies that some redistribution of income can increase average health.

With the survival functions given above, program (6) becomes

$$(9) \quad \underset{l, e_{+1}}{\text{Max}} (Ae_{+1})^{1-\alpha} [r(Bl)^{1-\beta} / (1-\beta) + 1] / (1-\alpha)$$

$$Rh = lR + ne_{+1}$$

$$l, e_{+1} \geq 0, \quad Ae_{+1} \leq (1-\alpha)^{1/(1-\alpha)}, \quad Bl \leq (1-\beta)^{1/(1-\beta)}$$

We make the following assumptions

Assumption 1. *The parameters of the model must satisfy the constraints*

$$(10) Bh^+ \leq (1-\beta)^{1/(1-\beta)} \left[1 + \frac{1-\alpha}{1-\beta} (1+1/r) \right]$$

$$(11) Ah^+ \leq \frac{n}{R} (1-\alpha)^{1/(1-\alpha)}$$

This assumption is needed to guarantee the solvability of the optimisation problem considered. Now, we can establish the following lemmas. Lemma 1 is precisely about the latter property.

Lemma 1. *Program (9) has a unique solution defined by the two equations*

$$(12) \frac{h}{l} - \frac{1-\alpha}{r(Bl)^{1-\beta}} = 1 + \frac{1-\alpha}{1-\beta}$$

$$(13) e_{+1} = \frac{R}{n} (h-l)$$

Proof. Equation (13) is the constraint in program (9). We use this constraint to eliminate e_{+1} from the objective function. Equation (12) is the first order conditions of the so-transformed objective function. Let us define the function $y(l) \equiv \frac{h}{l} - \frac{1-\alpha}{r(Bl)^{1-\beta}}$. We have $y(0) = +\infty$,

$$y(h) = 1 - \frac{1-\alpha}{r(Bh)^{1-\beta}} < 1 + \frac{1-\alpha}{1-\beta}, \quad y(+\infty) = 0. \quad y(l) \text{ has a unique minimum, which is negative, for}$$

$$l^\beta = \frac{rh}{(1-\alpha)(1-\beta)} B^{1-\beta}. \quad \text{Thus, equation (12) defines a unique value for } l, \text{ which is positive}$$

and smaller than h .

We have to check that this solution satisfies $Bl \leq (1-\beta)^{1/(1-\beta)}$. This is equivalent to

$$y\left[\frac{(1-\beta)^{1/(1-\beta)}}{B}\right] \leq 1 + (1-\alpha)/(1-\beta), \text{ which results from inequality (10). We also have to}$$

$$\text{check that } Ae_{+1} = A \frac{R}{n} (h-l) \leq (1-\alpha)^{1/(1-\alpha)} \quad \text{or} \quad Al \geq Ah - \frac{n}{R} (1-\alpha)^{1/(1-\alpha)}. \text{ This condition is}$$

satisfied because of inequality (11). \square

The two following lemmas describe in detail the characteristics of the optimal decisions taken by a junior adult, first concerning investment in his own health, then concerning investment in the health of his offspring.

Lemma 2. *a) A well-endowed junior adult invests more in his health than a poorly endowed junior adult. b) The investment of a junior adult in his own health increases with his earnings*

and when there is an increase in the utility for parents of being alive. c) The investment of a junior adult in his own health increases with the scale parameter of his survival function. d) The investment of a junior adult in his own health is independent of the scale parameter of the survival function of his children.

Proof. We use the following equation $y(l) \equiv \frac{h}{l} - \frac{1-\alpha}{r(Bl)^{1-\beta}} = 1 + \frac{1-\alpha}{1-\beta}$, which determines l and the properties of the function $y(l)$, which were established in the proof of lemma 1. \square

Lemma 3. a) A well-endowed junior adult invests more in the health of his children than a poorly endowed junior adult. His total investment is independent of the number of his children. b) The investment of a junior adult in the health of his children increases with his earnings and decreases when there is an increase in the utility for parents of being alive. c) The investment of a junior adult in the health of his children decreases with the scale parameter of his survival function. d) The investment of a junior adult in the health of his children is independent of the scale parameter of the survival function of these children.

Proof. We use the results of the previous lemma and either the expression of ne_{+1} , which is given by equation (13), or the following expression, which results from a combination of

$$\text{equations (12) and (13) } ne_{+1} = R(1-\alpha) \left[\frac{l}{1-\beta} + \frac{l^\beta}{rB^{1-\beta}} \right]. \square$$

The model has several worth-mentioning properties. First, and as announced in the introduction section, our model entails inequality in front of death. Children of parents with low human capital have a higher probability of dying before growing. Moreover, such parents tend to spend less in their own health care (and education), and hence face a lower survival probability with the subsequent negative effect on the human capital of the resulting orphans. Second, the investment decisions taken by the junior adults are sensitive to exogenous changes in their survival function (Property c of Lemma 2 and 3) but not to shifts in the survival function of their children (Property d of Lemma 2 and 3). Put in other words, an epidemic hitting young adults will have an impact on the investment decisions of these individuals while an epidemic hitting their own children will not.

The consequences of varying life expectancy are extensively studied in the literature. Our model has some interesting predictions regarding this issue. In the standard theory relying on Blanchard-Yaari structures, life expectancy (or the mortality rate) is exogenous. A downward

shift in life expectancy generally decreases the marginal return to investment in this framework, implying less investment either in physical capital (as in the standard Blanchard model, 1985) and/or human capital (as in Boucekkine, de la Croix and Licandro, 2002). In our model, life expectancy is no longer exogenous. When an epidemic shortens the life expectancy of junior adults, healthcare expenditure decreases for reasons similar to the ones we just gave and life expectancy decreases by more than what results from the direct effect of the epidemic.

Actually, our set-up has more subtle predictions concerning children's health care: first, health care expenditures in the benefit of children go up under 'adult' epidemics, and second, the investment decisions of the parents are sensitive to a drop in their own life expectancy but not to a drop in the life expectancy of their children. The first property is very easy to accept given the age specificity of the epidemic considered. The second property could be challenged. For example one could think that he should increase his health expenditures in the benefit of his children when they are subject to an age specific epidemic, precisely because his utility is entirely determined by the expected human capital accumulated by children who will reach the adult age. Nonetheless, because his lifetime earnings are pre-determined, such an increase in the health expenditures of children would imply a decrease in his own health care. Such a trade-off would arise in any model where children have no direct contribution to households' earnings: a child-specific epidemic does not affect earnings, and so rising health expenditure in favour of children is necessarily detrimental to adults' or elderly' health care. In our model, the trade-off is settled in the most neutral way: no extra health care for none.

The next section is devoted to the explicit study of the dynamics of populations and income distributions induced by these properties. Indeed, one of the important advantages of our simplified set-up is to allow for a full analytical appraisal of the latter dynamics. Before, we shall close the model and present its temporary equilibrium.

3.2. The temporary equilibrium of the model

The equilibrium values of investments in health by well-endowed or poorly-endowed junior adults are given by equations (12) and (13), where we only have to specify the respective endowment in human capital, h^+ or h^- , of these agents. The savings of both kinds of junior adults, s^+ and s^- , can easily be derived from equation (2).

3.2.1. Demographic variables

The population alive in period t includes N^{2+} and N^{2-} junior adults with human capital endowments respectively equal to h^+ and h^- . It also includes N^{3+} and N^{3-} senior adults. Finally, it includes N^{1+} , N^{1-} children who have parents with respective human capital h^+ , h^- , and N^{1o+} , N^{1o-} orphans with respectively high and low bequests. The parents of the two first kinds of children are the senior adults of the period. So, we have:

$$(14) \quad N^{1+} = nN^{3+} \quad \text{and} \quad N^{1-} = nN^{3-}$$

The populations N^{1o+} , N^{1o-} , N^{2+} , N^{2-} , N^{3+} and N^{3-} are predetermined in period t . The number of well-endowed (poorly-endowed) senior adults which will be alive in period $t+1$ is equal to the number of junior adults with the same endowment who are alive in period t , time their rate of survival

$$(15) \quad N_{+1}^{3+} = \pi(l^+)N^{2+}, \quad N_{+1}^{3-} = \pi(l^-)N^{2-}$$

If we use equation (14) in period $t+1$ (notice that the total number of children in this period is equal to the number of junior adults in period t times n), we get the equations

$$(16) \quad N_{+1}^{1o+} = nN^{2+} - nN_{+1}^{3+} \quad \text{and} \quad N_{+1}^{1o-} = nN^{2-} - nN_{+1}^{3-}$$

The numbers of well-endowed and poorly-endowed junior adults in period $t+1$ are

$$(17) \quad N_{+1}^{2+} = \lambda(e^+)(pN^{1+} + qcN^{1o+}) + \lambda(e^-)(pN^{1-} + qcN^{1o-}),$$

$$N_{+1}^{2-} = \lambda(e^+)(N^{1+} + cN^{1o+}) + \lambda(e^-)(N^{1-} + cN^{1o-}) - N_{+1}^{2+}$$

3.2.2. Balance of trade and international borrowing

In period t , human capital in the country is equal to $N^{2+}h^+ + N^{2-}h^-$. This expression also gives the quantity of health good domestically produced that is domestic output. The national demand for health good is $N^{2+}l^+ + N^{2-}l^- + (N^{1+} + N^{1o+})e^+ + (N^{1-} + N^{1o-})e^-$. The excess of supply over demand is equal to the surplus of the trade balance BT . If we use equations (1), (2), (14), (15) and (16) we can write this surplus as

$$(18) \quad BT = (N^{2+}s^+ + N^{2-}s^-) - (N^{3+}s_{-1}^+ - N^{3-}s_{-1}^-)R - [N_{-1}^{2+}(1 - \pi(l_{-1}^+))s_{-1}^+ + N_{-1}^{2-}(1 - \pi(l_{-1}^-))s_{-1}^-]R$$

The first term represents saving by junior adults. The second term represents the disaving (interests included) by senior adults. The last term represents the disaving of the dead, or if one prefers by the orphans.

If we use equation (13) this expression can be rewritten

$$(19) \quad BT = (N^{2+} s^+ + N^{2-} s^-) - (N_{-1}^{2+} s_{-1}^+ + N_{-1}^{2-} s_{-1}^-) R$$

The second term of the right-hand side represents assets held by nationals at the beginning of period t . The first term represents assets held by nationals at the end of period t . They will be inflated by the factor R at the beginning of period $t+1$. Thus, national assets grow at the same rate as the population of junior adults. We will show that in the steady state this rate is lower than n , the number of children by junior adults, and we will assume that $n < R$. So, the discounted value of national assets (debt) will tend to 0 when time increases indefinitely.

4. Dynamics and long run equilibrium

We will start by examining the equations giving the dynamics of populations. Then, in a second paragraph, we will investigate the properties of this dynamics, when the environment of the economics is kept unchanged.

4.1. The dynamics of populations

There are N^{2+} and N^{2-} junior adults alive in period $t \geq 0$. They will have n children each. These children will either become N_{+2}^{2+} and N_{+2}^{2-} junior adults with earnings respectively equal to h^+ and h^- in period $t+2$, or they will die at the end of period $t+1$. D_{+2} represents the supplementary number of junior adults who would exist in period t if no children die before reaching the age of junior adult, that is if the survival rate function λ were identical to 1. We will investigate the dynamics of the model for $t \geq 2$. The states of the economy in periods 0 and 1 are assumed to be given. We have the fundamental relationship:

$$(20) \quad \begin{pmatrix} N_{+2}^{2+} \\ N_{+2}^{2-} \\ D_{+2} \end{pmatrix} = An \begin{pmatrix} N^{2+} \\ N^{2-} \\ D \end{pmatrix} = \begin{pmatrix} a_{11} & a_{12} & 0 \\ a_{21} & a_{22} & 0 \\ 1-a_{11}-a_{21} & 1-a_{12}-a_{22} & 1 \end{pmatrix} n \begin{pmatrix} N^{2+} \\ N^{2-} \\ D \end{pmatrix}$$

with

$$a_{11} = \lambda(e_{+1}^+) \{ \pi(l^+) p + [1 - \pi(l^+)] c q \}$$

$$a_{21} = \lambda(e_{+1}^+) \{ \pi(l^+) (1-p) + [1 - \pi(l^+)] c (1-q) \}$$

$$a_{12} = \lambda(e_{+1}^-) \{ \pi(l^-) p + [1 - \pi(l^-)] c q \}$$

$$a_{22} = \lambda(e_{+1}^-) \{ \pi(l^-)(1-p) + [1 - \pi(l^-)]c(1-q) \}$$

and with $N^{2+}(0)$, $N^{2-}(0)$ and $D(0)$ given if t is even and $N^{2+}(1)$, $N^{2-}(1)$ and $D(1)$ given if t is odd.

Lemma 1, 2 and 3 imply that these parameters satisfy the constraints $0 < a_{12} < a_{11} < 1$, $0 < a_{22} < a_{21} < 1$, $a_{12} + a_{22} < a_{11} + a_{21} < 1$ and $a_{11}a_{22} - a_{12}a_{21} = c(p-q)\lambda(e_{+1}^+)\lambda(e_{+1}^-)[\pi(l^+) - \pi(l^-)] > 0$.

The elements of each column of A are positive and sum to 1. So they can be interpreted as proportions, or as conditional probabilities for instance for a child of a well-endowed junior adult to be well-endowed or poorly-endowed or dead two periods later.

More precisely, $a_{11} - a_{12}$ is the difference between the probabilities for a child to reach a high level of human capital if his parent is well-endowed versus if his parent is poorly-endowed. $a_{21} - a_{22}$ is the difference between the probabilities for a child to reach a low level of human capital if his parents are well-endowed versus if his parents are poorly endowed. The difference between the probabilities for a child to die if his parents are well-endowed versus if his parents are poorly endowed is $-(a_{11} - a_{12}) - (a_{21} - a_{22})$. The fate of children is independent of the social position of their parents when $a_{11} - a_{12} = a_{21} - a_{22} = 0$.

Matrix A in period t only depends on health spending set by junior adults, l^+ , l^- , e_{+1}^+ and e_{+1}^- . These spending are functions of the values taken by a series of exogenous variables in period t : the foreign interest rate R , the scale parameters of the survival functions of children and young adults A and B , the incomes of the junior adults h^+ and h^- and the number of their children n .

Equation (20) gives the dynamics of the numbers of junior adults and of the dead, N^{2+} , N^{2-} and D for $t \geq 2$, when the values of these variables are given in periods 0 and 1. Equation (15) gives the dynamics of the numbers of senior adults $N^{3+} = \pi(l^+)N_{-1}^{2+}$, $N^{3-} = \pi(l^-)N_{-1}^{2-}$ for $t \geq 1$. Equation (14) gives the dynamics of the number of non orphan children $N^{1+} = nN^{3+}$ and $N^{1-} = nN^{3-}$ for $t \geq 1$. Finally, the numbers of orphans in period $t \geq 1$ are given by equations (16) $N_{+1}^{1o+} = nN^{2+} - nN_{+1}^{3+}$ and $N_{+1}^{1o-} = nN^{2-} - nN_{+1}^{3-}$.

We define $P = N^{2+} + N^{2-} + D$ as the potential population of junior adults. It would be equal to the effective population if all children reached the age of junior adult. Equation (20) shows that this potential population grows at rate n : $P_{+2}^2 = nP^2$. The number of dead people is equal to the difference between the potential population and the number of junior adults: $D = P - (N^{2+} + N^{2-})$. Thus, we just have to investigate the dynamics of the numbers of living junior adults N^{2+} and N^{2-} , which is given by

$$(21) \begin{pmatrix} N^{2+}(t+2) \\ N^{2-}(t+2) \end{pmatrix} = \mathbf{B}n \begin{pmatrix} N^{2+}(t) \\ N^{2-}(t) \end{pmatrix} = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix} n \begin{pmatrix} N^{2+}(t) \\ N^{2-}(t) \end{pmatrix}$$

with $N^{2+}(0)$ and $N^{2-}(0)$ given if t is even and $N^{2+}(1)$ and $N^{2-}(1)$ given if t is odd. In the rest of the paper we will assume that t is even.

4.2. Characterization of the demographic dynamics

We will assume in this section that all the parameters and exogenous variables stay constant over time for $t \geq 0$. We will also assume that t is even. Then, matrix \mathbf{A} will stay constant over time, and the dynamics of the model will be limited to the sizes of the various components of population (including the dead). Let us introduce the new variable

$$(22) \Delta \equiv (a_{11} + a_{22})^2 - 4(a_{11}a_{22} - a_{12}a_{21}) = (a_{11} - a_{22})^2 + 4a_{12}a_{21} > 0$$

We have the lemma

Lemma 4. *a) The eigenvalues of matrix \mathbf{B} , ρ_1 and ρ_2 , are real and such that $1 > \rho_1 > \rho_2 > 0$. Their expressions are*

$$(23) \rho_1 = (a_{11} + a_{22} + \sqrt{\Delta})/2 \quad \text{and} \quad \rho_2 = (a_{11} + a_{22} - \sqrt{\Delta})/2$$

b) Let us denote by $V_1 = \begin{pmatrix} v_{11} \\ v_{21} \end{pmatrix}$ and $V_2 = \begin{pmatrix} v_{12} \\ v_{22} \end{pmatrix}$ the right-hand column eigenvectors of \mathbf{B} and

by $V = (V_1 \quad V_2)$ the matrix of these eigenvectors. A determination of these eigenvectors is

$$(24) V = \begin{pmatrix} 2a_{12} & -2a_{12} \\ a_{22} - a_{11} + \sqrt{\Delta} & -a_{22} + a_{11} + \sqrt{\Delta} \end{pmatrix}$$

V_1 can be normed such that its components are positive and sum to 1. V_2 can be normed such that its first component is negative, its second component is positive and the sum of both components is equal to 1.

c) Let $W = \begin{pmatrix} w_{11} & w_{12} \\ w_{21} & w_{22} \end{pmatrix}$ be the inverse of $V : VW = I$. Then, we have

$$(25) \quad W = \frac{1}{4a_{12}\sqrt{\Delta}} \begin{pmatrix} -a_{22} + a_{11} + \sqrt{\Delta} & 2a_{12} \\ -a_{22} + a_{11} - \sqrt{\Delta} & 2a_{12} \end{pmatrix}$$

d) The elements of matrix W satisfy the constraints

$$(26) \quad w_{11} > w_{12} > 0 \quad \text{and} \quad w_{21} < 0 < w_{22}$$

The proof is in the appendix. We can now establish the following crucial proposition which neatly characterizes the demographic dynamics and the evolution of human capital (and thus income) distributions over time.

Proposition 1. *Assume, to fix the ideas, that $N^{2+}(0) + N^{2-}(0) = 1$. Then:*

a) *The dynamic paths followed by the sizes of the cohorts of both kinds of junior adults, are linear combinations of two geometric series with rates equal to the growth rate of potential population n times the eigenvalues of matrix B*

$$(27) \quad N^{2+}(t+2) = (\rho_1 n)^{t/2+1} v_{11} [w_{11} N^{2+}(0) + w_{12} N^{2-}(0)] + (\rho_2 n)^{t/2+1} v_{12} [w_{21} N^{2+}(0) + w_{22} N^{2-}(0)]$$

$$(28) \quad N^{2-}(t+2) = (\rho_1 n)^{t/2+1} v_{21} [w_{11} N^{2+}(0) + w_{12} N^{2-}(0)] + (\rho_2 n)^{t/2+1} v_{22} [w_{21} N^{2+}(0) + w_{22} N^{2-}(0)]$$

In the long run the populations of both kinds of junior adults will grow at a rate equal to the growth rate of the potential population of junior adults times the largest eigenvalue of matrix B (which is smaller than 1). The long run size of each group depends on the initial condition, $N^{2+}(0)$. However, the long run proportions of the two groups of junior adults are independent of the initial conditions, and are precisely proportional to the two components of the eigenvector associated to the largest eigenvalue of matrix B .

b) *Let us assume that its share of junior adults holding a high level of human capital in the initial population is decreased. In the long run, the sizes of both groups of junior adults will drop. In the short run, the number of junior adults holding a high level of human capital and the total size of the population of junior adults will unambiguously go down. In contrast, the number of junior adults holding a low level of human capital may increase in the short run.*

The proof is in the appendix. Proposition 1 has several important implications, which will be illustrated later on in our application to epidemics next section. First of all, Property a) shows the ability of the model to generate hysteresis. This should not be though seen as a surprising result: this is a natural outcome in demographic models: initial demographic shocks are likely to have long lasting echo effects. Such effects may be dampened after a while, for example if fertility markedly changes some generations after the initial shock, but it seems out of question that persistence is a fundamental property of demographic dynamics. Second, our model features that an initial change in the income distribution of the population may distort this distribution in the short and medium terms but not in the long run. This is a very important property as we will see in the application to epidemics. Actually, one of the debates around AIDS (especially in sub-Saharan Africa) is its impact on income inequality either in the short or long run. Our benchmark model delivers a very clear message in this respect as explained hereafter.

5. The demographic and economic effects of epidemics

We define an epidemic as an increase in the death rate of a generation of people lasting for only one period. Two kinds of epidemics will be considered in this paper. First, the scale parameter A of the survival function of children is decreased by a fixed amount. Secondly, the parameter B of the survival function of junior adults is decreased. The epidemic hits people irrespectively of their endowment in human capital or of their social background. We will assume that nothing can be done against the epidemic itself and the number of death it directly causes. In both cases, we will start from a reference balanced growth path with a total population of junior adults equal to 1.

We first define precisely such a balanced path. We can deduce from the expressions of matrices V and W given in Lemma 4 that $w_{11}v_{11} + w_{12}v_{21} = 1$, and $w_{21}v_{11} + w_{22}v_{21} = 0$. Assume that the initial population of junior adults, $N^{2+}(0) + N^{2-}(0)$, is equal to 1, and suppose we norm eigenvector V_1 in such a way that the sum of its two components is equal to 1. If the vector of the initial values of the populations of the two kinds of junior adults is equal to the eigenvector of the transition matrix associated to its largest eigenvalue $\begin{pmatrix} N^{2+}(0) \\ N^{2-}(0) \end{pmatrix} = V_1$, the population of junior adults will follow the balanced growth path

$$(29) \begin{pmatrix} N^{2+}(t+2) \\ N^{2-}(t+2) \end{pmatrix} = (\rho_1 n)^{t/2+1} V_1$$

Proposition 1 shows that this steady state is relatively asymptotically stable. This will be our reference balanced growth path. We now move to our analysis of epidemics. For a better understanding, recall that total domestic output in our model is given by

$$(30) Y(t) = N^{2+}(t)h^+ + N^{2-}(t)h^- .$$

5.1. An epidemic hitting children

The epidemic takes place in period 1 and kills a given proportion of children. So, the population of junior adults alive in period 2 will be reduced by the same proportion. However, the ratio between the numbers of well-endowed and poorly endowed junior adults will be unchanged. The second effect will be that the population of junior adults will be reduced by a constant proportion in every *even* period by the children, grandchildren, etc. who will not be born because of the death of their forebear. Domestic output will be reduced by the same proportion in even periods.

Let us investigate the problem at a more formal level. The value of parameter A is decreased by $dA < 0$ in period 1. According to Lemma 2 and 3 junior adults do not change their investment decisions. Equations (20) and (21) show that matrix B is reduced by a factor $(1-\alpha)dA/A$ in period 0. So, the populations of both kinds of senior adults in every even period starting in period 2 is reduced by the same proportion. These populations remain unchanged in odd periods.

Equations (14), (15) and (16) show that in *even* periods the numbers of senior adults and of children of each category, are unchanged. These numbers are reduced by the factor $(1-\alpha)dA/A$ in *odd* periods starting in period 3. The only demographic change in period 1 is the death of children caused by the epidemic. Thus, the third consequence of the epidemic of period 1 is an echo effect, which permanently changes the demographic structure of the population. The share of junior adults is reduced in every even period and increased in every odd period. Thus, even if domestic output per worker remains the same in these periods, domestic output per capita decreases in even periods and increases in odd periods.

As we can see, such an epidemic has some important demographic and economic effects either in the short or long run by inducing a permanent demographic composition effect and a change in output per capita (but not per worker). Nonetheless, the epidemic is shown to be

neutral at all temporal horizons in terms of the income distribution among junior adults. The next section shows that ‘adult’ epidemics can in contrast distort such a distribution.

5.2. An epidemic hitting junior adults

The epidemic takes place in period 0 and kills a proportion of junior adults at the end of the period. The number of children alive in period 1 will be unchanged but the proportion of orphans among them will be higher. The number of senior adults alive in period 1 will be lower as a result of the epidemic. So, in the model, the value of parameter B is decreased by $dB < 0$ in period 0⁶. Junior adults living in this period perfectly understand the consequences of the epidemic when they make their decisions. They will reduce their investment in their own health, and their survival rates at the end of the period will decrease by more than what results from the epidemic. Junior adults will also increase their investment in the health of their children in period 1, which will improve the survival rates of children in period 1. Thus, matrix B has been changed in period 0, and consequently the populations of junior adults in period 2. The relative variations in the populations of juniors adults holding a high level and a low level of human capital, in this period is

$$(31) \quad \frac{dN^{2+}(2)}{N^{2+}(2)} = \frac{v_{11}da_{11} + (1-v_{11})da_{12}}{\rho_1 v_{11}}$$

$$(32) \quad \frac{dN^{2-}(2)}{N^{2-}(2)} = \frac{v_{11}da_{21} + (1-v_{11})da_{22}}{\rho_1(1-v_{11})}$$

The relative changes in the total population of junior adults and in the domestic output per worker are

$$(33) \quad \frac{dN^{2+}(2) + dN^{2-}(2)}{N^{2+}(2) + N^{2-}(2)} = \frac{v_{11}(da_{11} + da_{21}) + (1-v_{11})(da_{12} + da_{22})}{\rho_1}$$

$$(34) \quad \frac{\frac{dY(2)}{N^{2+}(2)h^+ + N^{2-}(2)h^-} - \frac{dN^{2+}(2) + dN^{2-}(2)}{N^{2+}(2) + N^{2-}(2)}}{N^{2+}(2)N^{2-}(2)(h^+ - h^-)}}{\left[\frac{dN^{2+}(2)}{N^{2+}(2)} - \frac{dN^{2-}(2)}{N^{2-}(2)} \right]} =$$

⁶ The assumption that the decrease in the value of parameter B that is in the probability of survival, is the same for junior adults with a high as with a low level of human capital, is debatable. There are indications that people with a relatively high schooling level are more exposed to the risk of being hit by AIDS because they have more sexual partners (Cogneau and Grimm, 2005). There are also indications that these people are more aware of the risks of AIDS than less educated people and understand faster the usefulness of not engaging in risky behaviour, for instance they are more responsive to campaigns of information, and prevention (de Walque, 2004). The United Nations (2004) quotes several studies showing that poor and uneducated people are more likely to engage in risky behaviour and to acquire HIV/AIDS.

The following lemma is an extension of lemmas 2 and 3.

Lemma 5. *Let us consider a junior adult with endowment h , and a decrease in the coefficient of his survival function by $dB < 0$. His probability of survival and the probability of survival of each of his children will change by*

$$(35) \quad \frac{d\pi(l)}{\pi(l)} = \frac{(1-\beta)h/l}{\beta h/l + 2 - \alpha - \beta} \frac{dB}{B} < (1-\beta) \frac{dB}{B} < 0$$

$$(36) \quad \frac{d\lambda(e_{+1})}{\lambda(e_{+1})} = -\frac{l}{h-l} \frac{(1-\alpha)^2(1-\beta)}{r(Bl)^{1-\beta}(\beta h/l + 2 - \alpha - \beta)} \frac{dB}{B} =$$

$$-\frac{[(1-\alpha)l]^2}{h(h-l)} \frac{1}{(1-\beta)r\pi(l)} \frac{d\pi(l)}{\pi(l)} > 0$$

Proof. We deduce from equation (13)

$$\frac{de_{+1}}{e_{+1}} = -\frac{dl}{h-l}.$$

We deduce from equation (12)

$$\left(\beta \frac{h}{l} + 2 - \alpha - \beta\right) \frac{dl}{l} = \frac{(1-\alpha)(1-\beta)}{r(Bl)^{1-\beta}} \frac{dB}{B} = \left[(1-\beta) \frac{h}{l} - 2 + \alpha + \beta\right] \frac{dB}{B} < 0.$$

If we differentiate equation (8) and use the previous equation we get equation (35). We deduce from equation (7)

$$\frac{d\lambda(e_{+1})}{\lambda(e_{+1})} = (1-\alpha) \frac{de_{+1}}{e_{+1}} = -(1-\alpha) \frac{l}{h-l} \frac{dl}{l}$$

If we substitute the above expression of dl/l we get equation (36). \square -

An epidemic decreases the probability of survival of junior adults, first because it increases the death rate of this population, secondly because it reduces the spending of this population on its own health. This epidemic increases the probability of survival of children (conditionally on the facts that they are orphans or that their parents are alive) because parents spend more on the health of their children. The following lemma will be used in the proof of Proposition 2.

Lemma 6. Consider a junior adult with endowment h who invests l in his own health. When parameters c and q change, the expression

$$(37) E = (1-\alpha) \frac{l}{h} \left[1 - \frac{1-\alpha}{1-\beta} \frac{h}{h-l} \right] = \frac{[(1-\alpha)l]^2}{(1-\beta)r\pi(l)h(h-l)}$$

has a positive lower bound \underline{E} and an upper bound \bar{E} smaller than 1.

Proof. Equation (5) and the conditions on the parameters imply that r is positive and has an upper bound. Equation (12) shows that l has a positive lower bound.

Equation (12) shows that l has an upper bound smaller than h . Thus, E has an upper bound smaller than 1. \square

The following proposition will give the changes, taking place in period 2, in the total population of junior adults, and in the population of workers holding, respectively, a high level and a low level of human capital, induced by an epidemic taking place in period 0.

Proposition 2. If the reduction in the probability of survival of orphans, $1-c$, and if the probability for an orphan to reach a high level of human capital, q , are low enough, we have the following results.

- a) In period 2 the total population of junior adults increases.
- b) The population of junior adults holding a high level of human capital decreases, and the population of junior adults with a low level of human capital increases. Thus, the proportion of junior adults with a low endowment of human capital in the total population increases. Consequently, domestic output per worker decreases.
- c) The numbers of each kind of children and senior adults are unchanged.

The proof is in the appendix. When an epidemic takes place, well-endowed junior adults will spend more on the health of their children. This will contribute to increasing the proportion of these children who will survive in period 2. However, more of these children will grow as orphans whose the probability of survival is reduced by a factor $1-c$. If c is near enough to 1, the first effect will dominate and the number of junior adults alive in period 2 will be higher.

In period 2, the number of junior adults who were orphans will increase and the number of those who were brought up by their parents will decrease. If the probability for an orphan to

reach a high level of human capital, q , is low enough, the number of junior adults with a high level of human capital, alive in period 2, will become lower. The two assumptions of Proposition 2 mean that orphans are more disadvantaged in their probability of reaching a high level of human capital than in their probability of dying before adult age.

Proposition 2 is a crucial characterisation of the medium term distributional effects of ‘adult’ epidemics. In contrast to the epidemic only killing children, considered before, the distributional consequences are significant in the medium run. More young adults will get less educated two periods after the epidemic and output per worker goes down: the economy is clearly impoverished (with respect to the reference balanced growth path) at this time horizon⁷. Thus, the demographic and economic effects are clearly much more potentially dangerous when the epidemic hits junior adults than when it only affects children. This is of a course a natural outcome since adults are the working individuals in the economy. However, our model already makes nontrivial contributions at this stage: it neatly shows the huge differences between ‘child’ Vs ‘adult’ epidemics in all respects, and in particular, it forward puts the differences in distributional consequences, which is not treated so explicitly in the related literature.

The analysis of periods posterior to period 2 is cumbersome. We know that, in the long run, the shares of junior adults holding respectively a high level and a low level of human capital that is the income distribution will go back to their balanced growth values. So, in contrast to some contributions in the AIDS-related literature (like Bell et al., 2003), the model predicts a kind of corrective dynamics which will bring some key variables to the corresponding balanced growth corresponding values. But we cannot even conclude on the long run change in the total population of junior adults without further assumptions. However, we can note that just like ‘child’ epidemics and for the same reasons, we have some permanent effects, notably on the demographic composition of the economy.

5.3. A first analysis of the medium-term effects of AIDS

AIDS mostly hits junior adults. However, many children of contaminated mothers get HIV and die. So, we can interpret AIDS as, first hitting the junior adults of period 0, and then

⁷ However, the share of the active population in the total population increases and we do not know if output per capita increases or decreases.

hitting their children born at the end of period 0 who become infected in period 1⁸. Thus we can cumulate the analysis of the two previous paragraphs. The relative decreases in the populations of junior adults and of children who survive the AIDS epidemic, if health spending did not change, respectively are $(1 - \beta)dB/B < 0$ and $(1 - \alpha)dA/A < 0$. As some of the children of people having got AIDS survive, the second decrease should be smaller than the first.

We will limit our analysis to the medium-run that is to the effects of AIDS in period 2. Then, the numbers of senior adults and children of every kind are unaffected by the epidemic. On one hand, the death of children in period 1 induces a proportional decrease in the population of both kinds of junior adults in the following period. Thus, the output per worker remains unchanged, but the output per head decreases. On the other hand, the death of junior adults in period 0 induces an increase in the total population of junior adults in period 2. The number of junior adults with a high level of human capital decreases; the number of junior adults holding a low level of human capital increases. So, output per worker decreases but the evolution of output per head is undetermined.

One of the most robust stylised facts is that AIDS induces important diminution in total population in the medium-term as in the long-run. To obtain this result our model must assume that AIDS kills enough children and not too many junior adults. Under this assumption we get the following effects of the HIV/AIDS epidemic in the medium term. The numbers of senior adults and children of every kind are unaffected but the number of junior adults decreases. In this last population, the proportion of people with a high level of human capital decreases and the proportion holding a low level of human capital increases. Finally output per worker and output per capita decrease.

The condition under which these results are obtained, which is that AIDS must kill a large number of children, is unconvincing. However, AIDS also reduces the fertility of women. First, women die when they are in reproductive ages and secondly, women who survive become more cautious about having sex for fear of infection, and because as others die out of the workforce, female labour becomes more valuable (Young, 2005). The next paragraph will

⁸ The discretisation of time used in the model implies that children who die from AIDS die after their parents, actually in period 1 when their parents died in period 0. This is only a technical simplification, which excludes

show that assuming a decrease in fertility will keep all the previous results unchanged, but under more reasonable assumption.

5.4. A reduction in fertility

A junior adult living in period 0 will have, at the end of this period, a number of children reduced by the amount $dn < 0$. In the following periods fertility will be restored to its initial level. According to Lemma 2 and 3 this junior adult will keep health spending on him unchanged. He will also keep health spending on the whole of his children unmodified. So, a junior adult with an endowment of human capital h^+ will increase his investment in the health of each of his children by $de_{+1}^+ = -e_{+1}^+ dn/n > 0$. The probability of survival of this child will increase by $d\lambda(e_{+1}^+) = (1-\alpha)\lambda(e_{+1}^+)de_{+1}^+/e_{+1}^+ = -(1-\alpha)\lambda(e_{+1}^+)dn/n > 0$.

The number of junior adults alive in period 2 will change in reaction to two opposite effects. It will tend to decrease because of the lower number of children born at the end of period 0, but it will tend to increase because parents will spend more on the health of each of their children. We can compute the total effect by differentiating equation (21)

$$(38) \begin{pmatrix} dN^{2+}(2) \\ dN^{2-}(2) \end{pmatrix} = \left[Bdn + n \begin{pmatrix} da_{11} & da_{12} \\ da_{21} & da_{22} \end{pmatrix} \right] \begin{pmatrix} N^{2+}(0) \\ N^{2-}(0) \end{pmatrix}$$

If we use equation (20) we get

$$(39) \begin{pmatrix} da_{11} & da_{12} \\ da_{21} & da_{22} \end{pmatrix} = -(1-\alpha)Bdn/n$$

Thus

$$(40) \begin{pmatrix} dN^{2+}(2) \\ dN^{2-}(2) \end{pmatrix} = \alpha B \begin{pmatrix} N^{2+}(0) \\ N^{2-}(0) \end{pmatrix} dn = \alpha \begin{pmatrix} N^{2+}(2) \\ N^{2-}(2) \end{pmatrix} dn/n < 0$$

the case when children die before their parents.

So, the consequence of a decrease in fertility in period 0 will be to reduce the population of both kinds of senior adults in period 2 and every following even period by the proportion $\alpha dn/n$.

The effects of a temporary reduction in fertility are very similar to those of an epidemic hitting children. The ratio between the numbers of well-endowed and poorly endowed junior adults will be unchanged, but the population of junior adults will be reduced by a constant proportion in every *even* period. The numbers of senior adults and of children of each category will be reduced by the factor $\alpha dn/n$ in *odd* periods starting in period 3. The only demographic change in period 1 will be the reduction in the number of children resulting from the decrease in fertility. So, the share of junior adults in the total population is reduced in every even period and increased in every odd period. Thus, even if the domestic output per worker remains the same in these periods, domestic output per capita decreases in even periods and increases in odd periods.

6. Conclusion

This paper investigates the medium term effects of an epidemic, which are the effects one generation after the time when the epidemic started to expand to large segments of the population. In the medium term the number of deaths directly caused by the epidemic has decreased, but the economy still suffers the consequences of the epidemic, for instance because of the orphans who died young or lost the opportunity to receive good education. The effects of an epidemic will be very different if it hits children or the active population. In the first case, the size of the active population, its fraction of total population and domestic output per capita, will be depressed. However, the composition of the active population by levels of education and skill will remain unchanged. In the following periods, the economy will go through a succession of repeated contractions in even periods and expansions in odd periods.

An epidemic hitting the active population will have the opposite effects on the size of the active population, which will increase in the medium term. So, the fraction of this population in total population will expand. Moreover, in the medium term, the larger active population will be, in average, less educated, and its output per worker will be lower. So, output per worker will be depressed. Progressively, this unbalance in the composition of the active population will disappear, and its average productivity will increase and converge to the level

it would have had if the epidemic had not taken place. This last conclusion is similar to the one reached by the Lucas-Uzawa model, reminded in the introduction.

To analyse the medium term effects of HIV/AIDS, we assumed that the epidemic first hit junior adults. However, it also increases the number of deaths among children and reduces the rate of fertility because women die in reproductive ages or because they decide to have fewer children for health and economic reasons. Then, we showed that the size of total population will decrease in the medium term, and that the share of the active population in the total population will also be lower. In the active population, the proportion of people with a high level of human capital will decrease and the proportion holding a low level of human capital will increase. Finally output per worker and per capita will decrease.

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APPENDIX

Proof of Lemma 4

a) The eigenvalues of matrix B are the roots of the characteristic equation

$$S(\Lambda) \equiv \rho^2 - (a_{11} + a_{22})\rho + (a_{11}a_{22} - a_{12}a_{21}) = 0$$

The discriminant of this equation is $\Delta > 0$. So, the two eigenvalues of B are distinct and real.

Their product is given by $S(0) \equiv a_{11}a_{22} - a_{12}a_{21} > 0$. Moreover we have

$$S(1) \equiv 1 - (a_{11} + a_{22}) + (a_{11}a_{22} - a_{12}a_{21}) = (1 - a_{11})(1 - a_{22}) - a_{12}a_{21}$$

As we have $1 - a_{11} > a_{21}$ and $1 - a_{22} > a_{12}$, we can conclude that $S(1) > 0$. Thus, the two eigenvalues of matrix B are strictly included between 0 and 1.

b) We have

$$(a_{11} + a_{22} + \sqrt{\Delta})v_{11} / 2 = \rho_1 v_{11} = a_{11}v_{11} + a_{12}v_{21}, \text{ so}$$

$$(a_{22} - a_{11} + \sqrt{\Delta})v_{11} = 2a_{12}v_{21}$$

We also have

$$(a_{22} - a_{11} - \sqrt{\Delta})v_{12} = 2a_{12}v_{22}$$

So, a determination of the eigenvectors is given by equation (24). The two components of V_1 are positive and we can norm this eigenvector by setting $v_{11} + v_{21} = 1$. Moreover the sum of the two components of V_2 is positive and we can norm this eigenvector by setting

$$v_{12} + v_{22} = 1$$

c) We deduce from $VW = I$

$$2a_{12}(w_{11} - w_{21}) = 1$$

$$2a_{12}(w_{12} - w_{22}) = 0$$

$$(a_{22} - a_{11})(w_{11} - w_{21}) + \sqrt{\Delta}(w_{11} + w_{21}) = 0$$

$$(a_{22} - a_{11})(w_{12} - w_{22}) + \sqrt{\Delta}(w_{12} + w_{22}) = 1$$

$$\text{so } w_{11} = \frac{1}{4\sqrt{\Delta}} \frac{\sqrt{\Delta} + a_{11} - a_{22}}{a_{12}} > 0 \text{ and } w_{12} = \frac{1}{2\sqrt{\Delta}} > 0$$

$$\text{and } w_{21} = \frac{1}{4\sqrt{\Delta}} \frac{-\sqrt{\Delta} + a_{11} - a_{22}}{a_{12}} < 0 \text{ and } w_{22} = \frac{1}{2\sqrt{\Delta}} > 0$$

d) The inequalities are easy to check. For example, $w_{11} > w_{12}$ is equivalent to

$\sqrt{\Delta} > 2a_{12} + (a_{22} - a_{11})$. A sufficient condition for this inequality is

$$\Delta \equiv (a_{11} - a_{22})^2 + 4a_{12}a_{21} > (a_{22} - a_{11})^2 + 4a_{12}(a_{12} + a_{22} - a_{11}), \text{ or}$$

$a_{11} + a_{21} > a_{12} + a_{22}$, which is true. \square

Proof of Proposition 1

a) Let \mathbf{P} be the diagonal matrix with elements ρ_1 and ρ_2 . Then (21) can be rewritten

$$\begin{pmatrix} N^{2+}(t+2) \\ N^{2-}(t+2) \end{pmatrix} = \mathbf{B}n \begin{pmatrix} N^{2+}(t) \\ N^{2-}(t) \end{pmatrix} = V\mathbf{P}Wn \begin{pmatrix} N^{2+}(t) \\ N^{2-}(t) \end{pmatrix} = V(n\mathbf{P})^{t/2+1}W \begin{pmatrix} N^{2+}(0) \\ N^{2-}(0) \end{pmatrix}$$

In the long run, under $N^{2+}(0) + N^{2-}(0) = 1$, we have

$$N^{2+}(t+2)/(\rho_1 n)^{t/2+1} \xrightarrow{t \rightarrow \infty} v_{11} [(w_{11} - w_{12})N^{2+}(0) + w_{12}]$$

$$N^{2-}(t+2)/(\rho_1 n)^{t/2+1} \xrightarrow{t \rightarrow \infty} v_{21} [(w_{11} - w_{12})N^{2+}(0) + w_{12}]$$

This establishes directly property a).

b) The dynamics of populations can be written

$$N^{2+}(t+2) = (\rho_1 n)^{t/2+1} v_{11} [(w_{11} - w_{12})N^{2+}(0) + w_{12}] + (\rho_2 n)^{t/2+1} v_{12} [(w_{21} - w_{22})N^{2+}(0) + w_{22}]$$

$$N^{2-}(t+2) = (\rho_1 n)^{t/2+1} v_{21} [(w_{11} - w_{12})N^{2+}(0) + w_{12}] + (\rho_2 n)^{t/2+1} v_{22} [(w_{21} - w_{22})N^{2+}(0) + w_{22}]$$

$$N^{2+}(t+2) + N^{2-}(t+2) =$$

$$(\rho_1 n)^{t/2+1} (v_{11} + v_{21}) [(w_{11} - w_{12})N^{2+}(0) + w_{12}] + (\rho_2 n)^{t/2+1} (v_{12} + v_{22}) [(w_{21} - w_{22})N^{2+}(0) + w_{22}]$$

We know from Lemma 4d that $w_{11} > w_{12} > 0$, and $w_{21} < 0 < w_{22}$. Lemma 4b established that

$v_{11}, v_{21}, v_{22} > 0$, $v_{12} < 0$, and $v_{12} + v_{22} > 0$ also hold.

Now notice that, if $N^{2+}(0)$ is decreased, then $N^{2+}(t+2)$ should go down. As $\rho_1 > \rho_2$,

$N^{2+}(t+2) + N^{2-}(t+2)$ drops too if $(v_{11} + v_{21})(w_{11} - w_{12}) + (v_{12} + v_{22})(w_{21} - w_{22}) \geq 0$. The

expressions of matrices V and W given in Lemma 4 show that the left-hand side of this inequality is equal to 0. However, we do not know if $N^{2-}(t+2)$ increases or decreases in the

short run. Indeed, by the same reasoning as just before, this figure would go down if

$v_{21}(w_{11} - w_{12}) + v_{22}(w_{21} - w_{22}) \geq 0$. Unfortunately this expression turns out to be equal to

$-4(a_{12}\sqrt{\Delta})$, which is negative. Therefore anything could happen in the short run as for the number of low human capital junior adults. \square

Proof of Proposition 2

a) The change in the number of junior adults living in period 2, whose parents held a high level of human capital is, according to equation (20)

$$d(a_{11} + a_{21}) = d\left\{\lambda(e_+^+)[\pi(l^+)(1-c) + c]\right\} = \left\{\pi(l^+)(1-c)\left[\frac{d\lambda(e_+^+)}{\lambda(e_+^+)} + \frac{d\pi(l^+)}{\pi(l^+)}\right] + c\frac{d\lambda(e_+^+)}{\lambda(e_+^+)}\right\}\lambda(e_+^+)$$

We use equation (36) and get

$$d(a_{11} + a_{21}) = \left\{\pi(l^+)(1-c)\left[1 - \frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)}\right] - c\frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)}\right\} \lambda(e_+^+) \frac{d\pi(l^+)}{\pi(l^+)}$$

Equation (35) shows that $d(a_{11} + a_{21}) > 0$ is equivalent to

$$\pi(l^+)(1-c)\left[1 - \frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)}\right] - c\frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)} < 0$$

We use equation (12) and get

$$\pi(l^+)(1-c)\left\{1 - (1-\alpha)\frac{l^+}{h^+}\left[1 - \frac{1-\alpha}{1-\beta}\frac{l^+}{h^+ - l^+}\right]\right\} < c(1-\alpha)\frac{l^+}{h^+}\left[1 - \frac{1-\alpha}{1-\beta}\frac{l^+}{h^+ - l^+}\right]$$

$$1 < \left[1 + \frac{c}{1-c}\frac{1}{\pi(l^+)}\right](1-\alpha)\frac{l^+}{h^+}\left[1 - \frac{1-\alpha}{1-\beta}\frac{l^+}{h^+ - l^+}\right]$$

Lemma 6 shows that the product of the two last terms of the right-hand side has a positive lower bound. So, for c near enough to 1, the inequality is satisfied.

A similar computation shows that $d(a_{12} + a_{22}) > 0$. Then, equation (33) establishes part a of the proposition.

b) We have

$$d(a_{11}) = d\left\{\lambda(e_+^+)[\pi(l^+)(p - cq) + cq]\right\} = \left\{\pi(l^+)(p - cq)\left[1 - \frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)}\right] - cq\frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)}\right\}\lambda(e_+^+)\frac{d\pi(l^+)}{\pi(l^+)}$$

Equation (35) shows that $d(a_{11}) < 0$ is equivalent to

$$\pi(l^+) (p - cq) \left[1 - \frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)} \right] - cq \frac{[(1-\alpha)l^+]^2}{h^+(h^+ - l^+)(1-\beta)r\pi(l^+)} > 0$$

We use equation (12) and get

$$1 > \left[1 + \frac{cq}{p - cq} \frac{1}{\pi(l^+)} \right] (1-\alpha) \frac{l^+}{h^+} \left[1 - \frac{1-\alpha}{1-\beta} \frac{l^+}{h^+ - l^+} \right]$$

$$\text{or } 1 + \frac{cq}{p - cq} \frac{1}{\pi(l^+)} < \frac{1}{E}$$

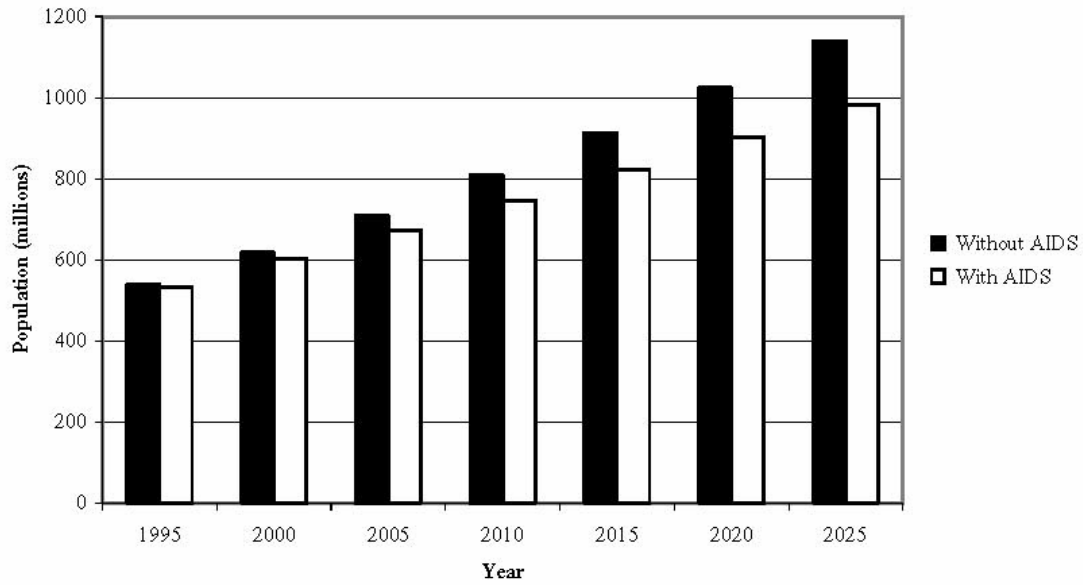
According to lemma 6, a sufficient condition for this inequality to hold is

$$1 + \frac{cq}{p - cq} \frac{1}{\pi(l^+)} < \frac{1}{\bar{E}}, \text{ with } 1/\bar{E} > 1$$

For q near enough to 0, the inequality is satisfied. A similar computation shows that $d(a_{12}) < 0$. Then, equation (31) establishes part b of the proposition. \square

Figure 1

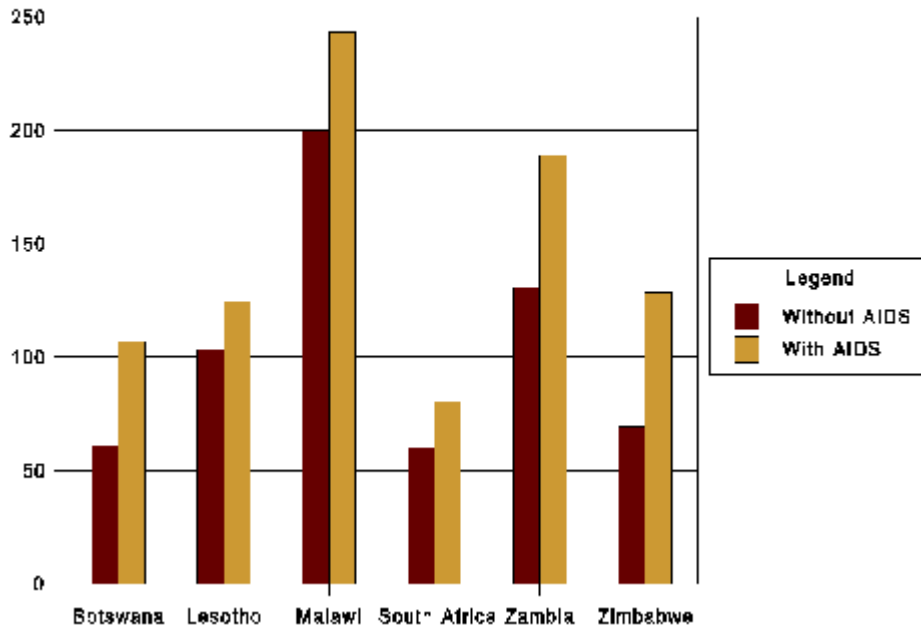
Figure 2. Estimated and projected population size with and without AIDS, 38 African countries, 1995-2000 to 2020-2025



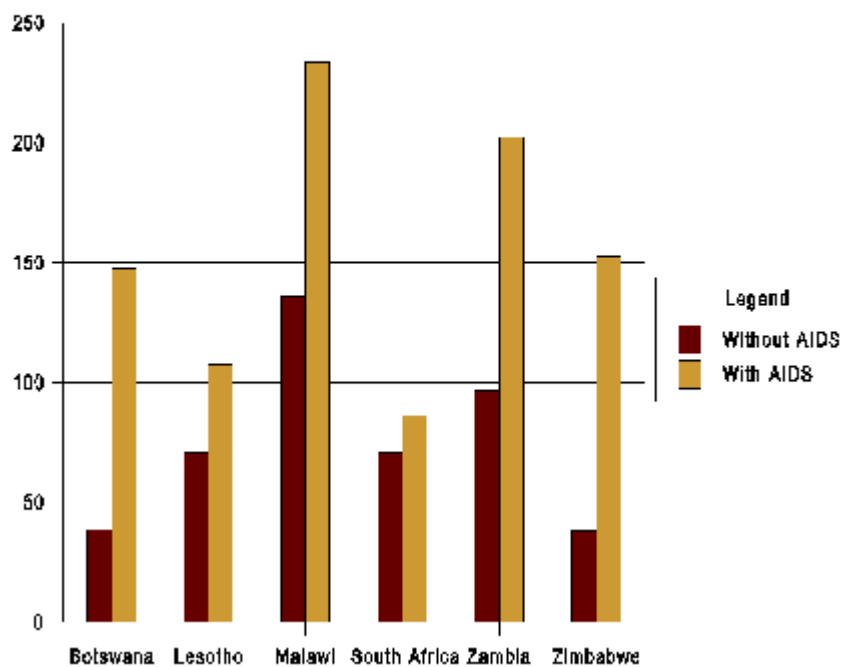
Source: World Population Prospects: The 2002 Revision, CD-ROM (United Nations, Department of Economic and Social Affairs, Population Division publication, Sales No. E.03.XIII.8).

Figure 2

**Child Mortality Rate* With and Without
AIDS, Southern Africa: 1996**



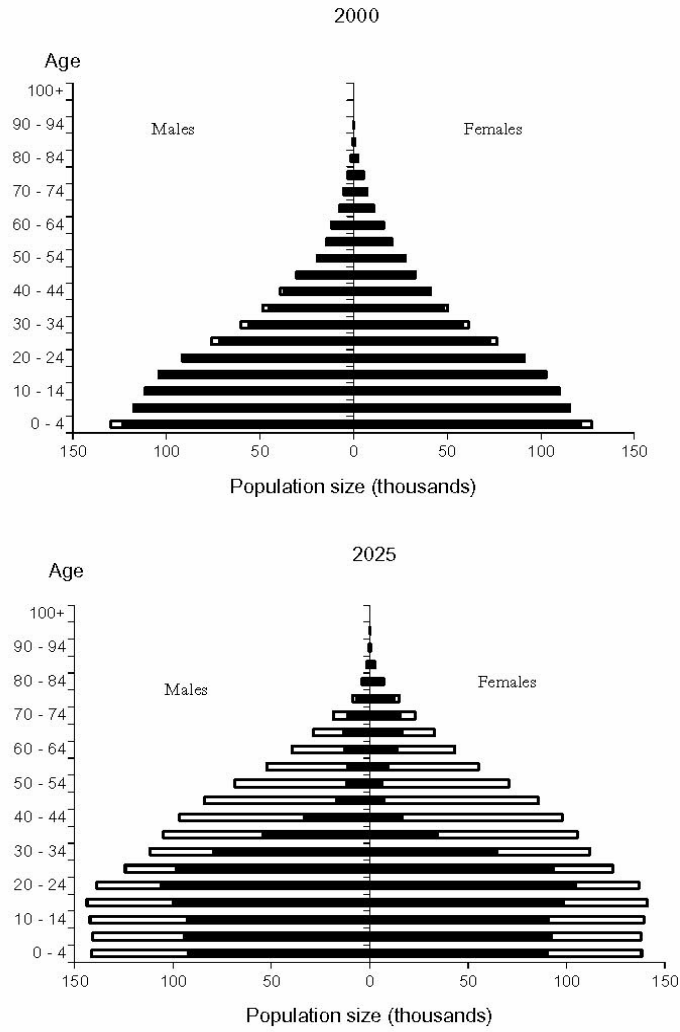
**Child Mortality Rate* With and Without
AIDS, Southern Africa: 2010**



*Child mortality rate is the number of children dying before age 5 per 1,000 live births.
Source: International Programs Center - Population Division US Bureau of the Census,
Washington, DC

Figure3

Figure 4. Population size with and without AIDS, Botswana, 2000 and 2025



Source: *World Population Prospects: The 2002 Revision*, CD-ROM (United Nations, Department of Economic and Social Affairs, Population Division publication, Sales No. E.03.XIII.8).

NOTE: Unshaded bars represent the hypothetical size of the population in the absence of AIDS. Shaded bars represent the actual estimated and projected population.