Aggregate Effects of AIDS on Development

Raül Santaeulàlia-Llopis*
Washington University in St. Louis

First Draft, December 2007
This Draft, April 2008

Abstract

In this paper I study the consequences of the AIDS epidemic for economic development. To this purpose I build a population model that keeps track of the demographic transition by age-specific population groups relating the age distribution of the population of each period to the preceding one via a fertility process, a mortality process and an aging process. I integrate this population model into a standard theory of economic development that determines the income per capita path along the process of industrialization - a transition that structurally shifts capital and labor from a Malthusian-agricultural sector to a neoclassical-industrial sector. This way, I provide a tight time-varying structural relationship between the distribution of the population across age groups and the stage of economic development - in terms of income per capita and agricultural share of output. Then, I use this population model to consistently identify the main channels through which AIDS, raising mortality rates of young adults and lowering fertility rates, affects populations over time: (i) reshapes the age distribution of the population, thinning the ranks of working-age groups (the share of children and old adults per worker raises by as much as 20-25% in highly infected countries), (ii) reduces population growth (by as much as .08% per percentage point of HIV prevalence), and (iii) reduces life expectancy (by as much as 15-20 years). In addition, AIDS also (iv) reduces the individual labor efficiency of the sick with an aggregate loss of 0.3% per percentage point of HIV prevalence. When I incorporate the AIDS epidemic as in (i)-(iv) into a model economy calibrated to an African country unaffected by AIDS, I find that the AIDS epidemic reduces per capita income by as much as 12% at the peak of the epidemic. I find also that the AIDS epidemic can slow down the transition from agriculture to industry by about one century in the worst case scenario: highly infected countries that are at the earliest stage of economic development.

*I am intellectually indebted to my advisor José-Víctor Ríos-Rull. I thank Frank Schorfheide, Urban Jermann, Dirk Krueger, Randall Wright, Hans-Peter Kohler and John Knowles for insightful comments and discussions, and I would like to thank Andrés Rodríguez-Clare, Michele Boldrin, Gustavo Ventura and the seminar participants at the UPenn Money-Macro Workshop, UPenn Population Studies Center, Penn State University, Washington University in Saint Louis, University of Iowa and the Population American Association 2008 Meetings. Universidad Carlos III, Universitat Autònoma de Barcelona, CEMFI, Universitat de València, Banco de Portugal, Institute for International Economic Studies (Stockholm), Institut fur Empirische Wirtschaftsforschung (Zurich), Population Association of America Meetings 2008, World Bank and the Federal Reserve Bank of Minneapolis. Special thanks go to Javier Díaz-Giménez for his red tape, a fundamental input of this project. I would also like to thank my colleagues Gil Shapira, Philip Anglewicz, Shalini Roy and Mallory Leung. I am grateful to The William and Flora Hewlett Foundation and Population Reference Bureau for research support.

1 Introduction

The AIDS epidemic is a well-known human tragedy. AIDS kills and maims people, dramatically reduces the life expectancy of people and distorts the way the population is distributed across age groups due to high mortality rates concentrated in 15–49 year-old adults (see, for instance, Jamison, Feachem, Makgoba, Bos, Baingana, Hofman, and Rogo (2006) and Stanecki (2004)). [1] This is particularly severe in Sub-Saharan Africa (SSA) where the share of HIV infected individuals in the population (the HIV prevalence rate) is 5% in 2007 (see UNAIDS (2007)).

What we do not know very well is how populations reshaped by the AIDS epidemic, that is, reduced, short-lived and less productive populations with rapidly diminishing generations of working age individuals, will fare in terms of economic development. Specifically, by how many years does the AIDS epidemic accelerate or delay the process of industrialization, and how much does the AIDS epidemic increase or decrease consumption per capita? I find the AIDS epidemic delays economic transition from agricultural to industrial regimes by about 105 years, and decreases the consumption per capita by as much as 12% at the peak of the epidemic.

To track and explore the quantitative implications that AIDS has for the development path of the Sub-Saharan African economies, I extend a standard theory of economic development, Hansen and Prescott (2002), with a population model that relates the age distribution of the population of each period to the preceding one via a fertility process, a mortality process and an aging process. This population process captures the main channels through which AIDS, raising mortality rates of young adults and lowering fertility rates, affects populations over time: (i) it reduces population growth (by as much as .08% per percentage point of HIV prevalence), (ii) reshapes the age distribution of the population, thinning the ranks of working-age groups (raising the share of children and old adults per worker by as much as 20-25% in highly infected countries), and (iii) reduces life expectancy (by as much as 15-20 years). In addition, AIDS (iv) shatters the individual labor efficiency of the sick with an aggregate loss of 2.7% for rural Malawi (a country where 80% of the labor force is in agriculture and where 10.2% the rural population is HIV infected).

I incorporate these channels (i)-(iv) into the development theory described in Hansen and Prescott (2002). They postulate a model with three phases of development. First, a long period of stagnation in per capita income in which the economy makes extensive use of a Malthusian-agricultural technology with a nonreproducible factor of production, fixed land, and all economic growth is in population (Malthus phase). Second, at a certain point, the set of production possibilities is large enough to initiate the industrialization process and growth in per capita income occurs. In this model, the structural shift, or transition from agriculture to industry, is jointly determined by a measure of relative efficiency (good policy and institutions), savings, and importantly, the demographic transition. Finally, the economy converges to the standard balanced growth path that makes extensive use of a neoclassical-industrial technology (Solow phase). This set of

[1] The Human Immune Deficiency Virus (HIV) is the cause of the Acquired Immune Deficiency Syndrome (AIDS). Throughout the paper I will use of the term AIDS instead of HIV/AIDS when referring to the disease and the epidemic; however, in the cases where the data are provided only in terms of HIV, I will refer specifically to HIV. A person infected with HIV does not show symptoms of illness or decreases in her/his labor efficiency until she/he develops AIDS. An HIV infected individual is classified as having AIDS if one of two things happens: 1) the CD4 cells drops below 200/cc, or 2) an HIV-related infection or HIV-related cancer develops. The duration between HIV infection and the onset of AIDS varies but averages 9-11 years, and death typically ensues within 1-2 years of symptom onset without antiretroviral drugs. Also, a time line of HIV/AIDS can be found in Appendix A.SSA.

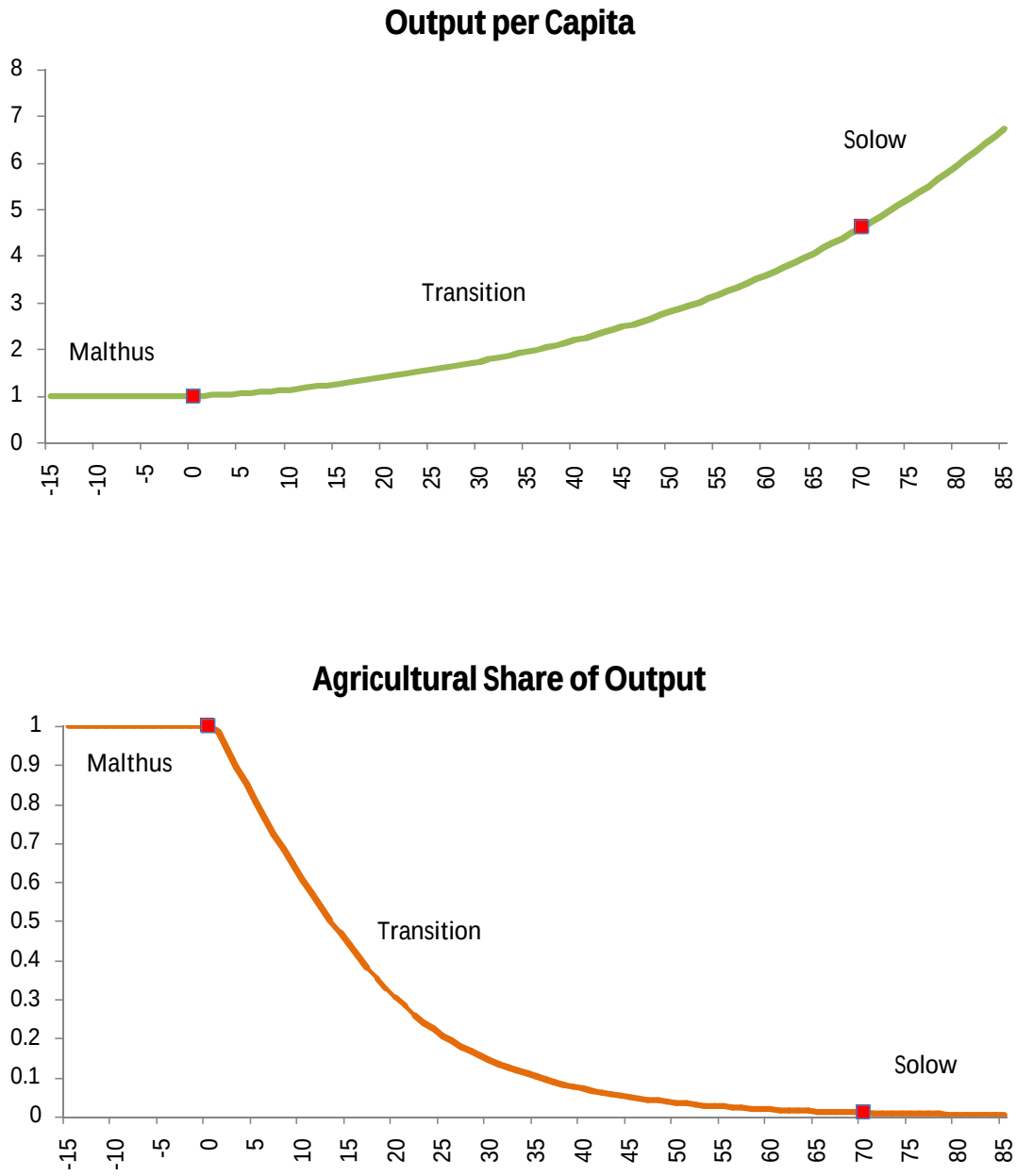

Figure 1: Stylized Development Process: Increase in Output per Capita (Top Panel), and Industrialization (Bottom Panel)

events corresponds to the stylized development process depicted in Figure 1, undertaken by modern industrialized economies, and defined in Kuznets (1965).

The thought experiment is, firstly, calibrate the development model to a country, also a late starter in the process of development as the SSA countries, with a population path not affected by the AIDS epidemic (the no-AIDS scenario). Secondly, specify AIDS as a shock that changes fertility rates and mortality rates by age-specific groups as it does in SSA countries with mature AIDS epidemics. This shock alters the law of motion of the population and generates an alternative population path with AIDS (the AIDS scenario). Finally, incorporate this population path with AIDS into the development model to quantify how much the development path in the AIDS scenario changes with respect to the no-AIDS scenario, in terms of income per capita and industrialization.

While economic development is a process subject to many circumstances that may hold it up, economists have made considerable improvements in understanding it (see an exhaustive review in Aghion and Durlauf (2005)). In particular, population dynamics is regarded as a crucial ingredient that both shapes and is shaped by increases in per capita income. Building on this idea, recent advances in development theory such as Becker, Murphy, and Tamura (1990), Galor and Weil (2000), Jones (2001), Hansen and Prescott (2002) and Lucas (2002), among others, [2] incorporate the 'demographic transition' to study the development experiences of modern industrialized economies. The 'demographic transition' breaks the pre-industrial population pattern of high, fluctuating mortality, and high, steady fertility through a reduction of mortality followed by a more gradual reduction of fertility, see Lee (2003). This results in a spur of population growth crucial for the earliest phases of industrialization (see Simon (1977), and Boserup (1981)). [3] Sub-Saharan countries are currently at the early stages of development, and this way, the AIDS epidemic is diminishing their population when, perhaps, they need it most.

In addition, the work of economic historians and historical demographers has emphasized the idea that changes in the composition of the population may often matter far more than changes in population aggregates for economic development, see Lee (1983), Kelley (1988), Livi-Bacci (1992) and Williamson (2001). In this line, Bloom, Canning, and Sevilla (2002) argue that economies with a large share of working age population may boost economic growth stemming from a reduced spending on dependents that accelerates accumulation of capital. In this context, by reducing the number of young adults aged 15-49 - a disproportionate number of parents and experienced workers, but not the number of dependents - AIDS raises the consumption to income ratio which may have nontrivial consequences for development.

Further, what catalyzes the demographic transition are reductions in mortality rates, that is, increases in life expectancy. Hence, the rise in the size of the population at early stages of industrialization is tied together with more longevity. Gains in life expectancy are considered to raise investment in human and physical capital, which may accelerate development (see, for example, Gallup and Sachs (2001), Kalemli-Ozcan (2002), Soares (2005), Lorentzen, McMillan, and Wacziarg (2005) and Jayachandran and Lleras-Muney (2007)). [4] This way, by reverting the gains in life expectancy the AIDS epidemic may undermine investment of all forms and, hence, strain

[2] See also Greenwood and Seshadri (2002), Fernández-Villaverde (2001), Gollin, Parente, and Rogerson (2002), Gollin, Parente, and Rogerson (2004), Doepke (2004) and Boldrin, Jones, and Khan (2007).

[3] For example, increasing population density may reduce indivisible infrastructure and transportation costs, encourage urbanization, help to enforce property rights (see North (1981)), and ease technological innovation (see Kuznets (1960), Kremer (1993), and Aghion and Howitt (1992)).

[4] Recently, the empirical work of Acemoglu and Johnson (2007) suggests the undercurrent view that life expectancy does not necessarily help to accelerate the process of economic development.

further the process of economic development.

1.1 Related Literature Review

UNAIDS (2006) provides a comprehensive review of an up-to-date research, mostly empirical, conducted to study the effects of AIDS on the level and growth of income per capita (see also Haacker (2004) and Zaba, Whiteside, and Boerma (2004)). Early analysis on the economic impact of AIDS focused on measuring the costs of the epidemics in terms of direct (mainly health) costs and indirect (output foregone) costs (see Cuddington (1993)), and used rudimentary behavioral models with strong assumptions on the direction of the effects of the disease (see Kambou, Devarajan, and Over (1992), Cuddington, Hancock, and Rogers (1994) and Over (1992)). Strikingly, recent more sophisticated empirical studies obtain extremely contradictory results. Pessimistic analyses such as Bonnel (2000), Bell, Devarajan, and Gersbach (2003), and Papageorgiou and Stoytcheva (2007) coexist with Bloom and Mahal (1997) and Werker, Ahuja, and Wendell (2006) which have found an insignificant effect of the AIDS epidemic on the growth rate of per capita income, even after controlling for the endogeneity of HIV prevalence.

Perhaps the best illustration of this conflict is that of the two most recent papers that study the effect of the AIDS epidemic on fertility, Young (2005) and Kalemli-Ozcan (2006). Arguing similarities between AIDS and the Black Death and using the Solow model, where population reductions increase income per capita due to a larger set of resources available for the survivors, Young (2005) finds that the negative effect of AIDS on fertility (a decrease in population growth or the level of future populations) dominates the negative effect of AIDS on the human capital of the offspring of the infected. [5] This way, on net, the AIDS epidemics enhances the future per capita consumption possibilities. Kalemli-Ozcan (2006), while finding a negative relationship between AIDS and schooling, finds, however, a positive relationship between AIDS and total fertility rates which, again within the context of the Solow model, lowers per capita economic growth and welfare for current and future African generations.

In this context, I argue that (i) the Black Death misspecifies the AIDS epidemic, and (ii) the Solow model does not reflect the current development stage of the African economies. First, the Black Death is a sudden demographic impact that swaps people within few years and does not discriminate (see Livi-Bacci (2001)) while the AIDS epidemic also comes unexpected but spreads slowly over time and kills selectively, mainly young adults (see Section 2). This way, although AIDS strikes by surprise, the slow spread and killing allows to learn the odds of contagion and death so that agents may start to modulate their investment decisions accordingly. Second, by focusing on income per capita, the Solow model is silent on the process of industrialization that defines economic development, that is, the structural shift from agriculture to industry which African economies have not completed yet. Importantly, the Solow model does not capture the standing positive relationship between population and the process of industrialization exemplified by the demographic transition experienced by most modern industrialized economies, which is especially relevant at the earliest phases of the industrialization process in which the Sub-Sahara happens to be immersed.

[5] One of the reasons for which a model of educational choice is appealing is in order to evaluate the loss of human wealth (see, for instance, **?** and **?** on the empirical relationship between AIDS and human capital). Here, I am going to abstract from educational choice and evaluate the net effect of the human wealth that resolves the tension between the change in the efficiency units of labor as the population gets infected (that is, reductions in the labor input today and tomorrow) and the behavior of factor prices.

The rest of the paper is organized as follows. In Section 2 I provide some data on the AIDS epidemic and its demographic impact. In Section 3 I describe a theoretical framework that captures the development stage of Africa, pose a population model that keeps track of the relevant aspects of the population that AIDS alters, and define the household problem. The equilibrium is defined in Section 4. Section 5 calibrates the model to an African country unaffected by AIDS. I specify the AIDS shock in Section 6. In Section 7 I discuss my findings. In Section 8 I show how, when I misspecify AIDS as the Black Death and consider the Solow model as the development theory for the Africa, we miss the fact that population reductions slow down the industrialization process. Finally, I conclude in Section 9.

2 Some Background Data of the AIDS Epidemic

It is now about 26 years since the first case of HIV was identified in 1981. Over these five lustra the disease has infected 65 million people worldwide (adults and children of all ages) and has taken the lives of more than 25 million (see a comprehensive up-to-date review in UNAIDS (2007)). [6]

Although all regions of the world have experienced its epidemic, AIDS is a phenomenon that affects most severely under-developed countries, [7] especially Sub-Saharan Africa (SSA). As of 2007, SSA is home for 22.5 millions of HIV infected people (that is, 68% of the globally infected population) and 1.6 millions of adult and child deaths due to AIDS occurr in SSA (that is, 78% of all AIDS deaths). Given that SSA hosts only 10% of the world population, it is the region with the largest share of HIV infected population (HIV prevalence rate), 5% in 2007, see the top panel in Figure 2. It is followed by far by the Caribbean, 1%, and Eastern Europe and Central Asia, 0.9%, while the global HIV prevalence rate is 0.8%, it is 0.4% in North America, and 0.3% in Western and Central Europe.

The epicenter of the AIDS epidemics is currently in Southern Africa with the highest HIV prevalence rates worldwide: Swaziland 25.9%, Botswana 24.1%, Lesotho 23.2%, Namibia 19.6%, South Africa 18.8%, Zimbabwe 18.1% and Mozambique 16.1%. Indeed, as much as one third of all people infected with HIV live in Southern Africa, and as much as one third of all AIDS deaths occur in Southern Africa. [8]

Although AIDS came as a demographic surprise, its major trends and medium-term outcomes have become discernible. In particular, the AIDS epidemics spreads smoothly and slowly, see, for example, the bottom panel of Figure 2 that depicts the time path of the number of adults and child deaths due to AIDS. A similar conclusion arises if we look at the time path of the HIV prevalence rate, globally and in SSA. Also, declines in national HIV prevalence are currently being observed in some Sub-Saharan African countries, but such trends are neither strong nor widespread. We observe that the flow of people newly infected has remained stationary at a pace of around 4 million per year (68% of the global) exceeding the number of deaths due to AIDS (which have been steadily rising from 2001 to 2006), and while this raises the stock of HIV infected, however, the HIV prevalence rates show no sizeable trend because of population growth.

In all, the epidemiological factsheet of AIDS is clear and far from anecdotist: the AIDS epidemic is a phenomenon of under-developed populations, concretely the Sub-Sahara; it builds up slowly over time; and it is not likely to vanish in the near future.

[6] This is half of the casualties of the Second World War (1937-45), 55-60 millions, and a similar number of deaths due to the biggest famine of all times, the Great Leap Forward famine in China (1958-1961), 30 millions. Also as a reference, in Europe alone, it is thought that over 20 million people died during the period 1347 to 1351 as a result of the Black Death.

[7] 85% of adults and children globally living with HIV in 2007 are in Africa, South-East Asia and Latin America

[8] In the 1980s and early 1990s the epidemic was experienced mostly in Eastern and Central Africa, where the HIV prevalence of women attending antenatal clinics were as high as 30%. Since the late 1990s the epidemic increased substantially in the Southern African countries with record high prevalence of 35.6% in Botswana, 23.6% in Lesotho, 16% in Malawi, 13.2% in Mozambique, 19.5% in Namibia, 19.9% in South Africa, 25.3% in Swaziland, 20% in Zambia, and 25.1% in Zimbabwe.

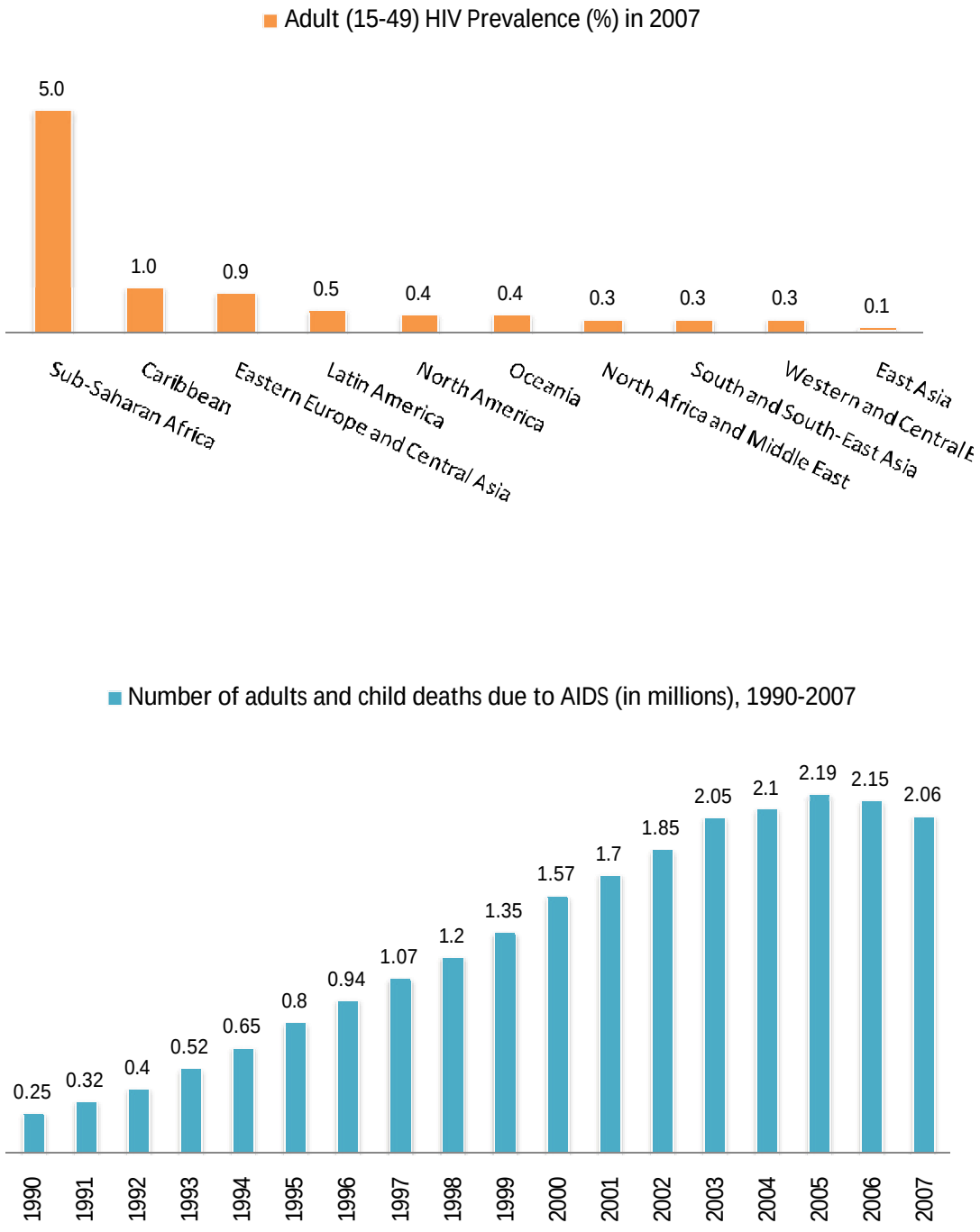

Figure 2: The AIDS epidemic is mostly Sub-Saharan, it builds up slowly, and it remains.

2.1 The Rate of Growth of the Population

The AIDS epidemic reduces the rate of growth of the population through higher mortality rates and lower fertility rates. According to he U.S. Census Bureau (see Stanecki (2004)) by 2010 five SSA countries are all expected to experience negative population growth due to AIDS: Botswana -2%, South Africa -1.4%, Swaziland -.4%, Mozambique -.2% and Lesotho -.2%. [9] The U.S. Census Bureau estimates that without AIDS these countries would have had a growth rate of 2% or greater. We can see this pattern in the top panel of Figure 3 that shows a cross-section of all African countries and associates a 1% rise of the share of population infected with HIV with 0.1% decreases of the population growth rate (Population Reference Bureau, 2005).

Mortality Rates. AIDS is currently the leading cause of death in SSA accounting for 20.4% of all deaths, see Jamison, Feachem, Makgoba, Bos, Baingana, Hofman, and Rogo (2006), and ranks as the number four cause of death globally. [10] The probability of dying for adults and adult HIV prevalence displays a positive, (and slightly curvilinear) relationship, see the report of Clark (2005) on the U.N. Wall Chart 2003. However, this is not the case for young children (mainly infected during pregnancy, at birth or through mother's milk), perhaps due to continued improvements in child mortality that offset the impact of AIDS.

Fertility Rates. The micro-evidence data suggests a negative effect of HIV infection on fertility. Zaba and Gregson (1998) find that lower fertility amongst HIV positive women causes a population attributable decline in total fertility, mainly due to foetal losses consequent to infection with HIV and co-infection with other sexually transmitted diseases, [11] of the order of .4% for each percentage point HIV prevalence in the general female population. More recently, Lewis, Ronsmans, Ezeh, and Gregson (2004), using DHS data for different SSA countries find that fertility among HIV-infected women was lower than that of HIV-uninfected women. [12] This fertility differential resulted in a population attributable decline in total fertility of .37% for each percentage point of HIV prevalence. Hunter, Isingo, Boerma, Urassa, Mwaluko, and Zaba (2003) they find, controlling for contraception, a substantial reduction (29%) in the fertility among HIV-infected women compared with uninfected women, using cohort data in rural Tanzania. They find the fertility reduction was most pronounced during the terminal stages of infection, but no clear association with duration of infection was observed. Also, higher HIV risk perceptions have been associated with lower fertility (see Yeatman (2007)), which has been interpreted as a lower demand for children in response to the epidemic, see Young (2005). The undercurrent view is held by Kalemli-Ozcan (2006), who suggests a potentially positive effect of increases in HIV risk on fertility rates.

[9] Overall, the epidemic has not led to a decrease in population in sub-Saharan Africa, due to relatively high fertility. Even accounting for AIDS-related mortality, United Nations projects the population of Sub-Saharan Africa to grow from 788 million in 2007 to 1.7 billion in 2050

[10] In SSA, AIDS is followed by far by malaria, 10.1%, and lower respiratory infections, 9.8%, as causes of death. Mathers and Loncar (2006) project AIDS will be the third cause of death worldwide in 2030.

[11] Besides the pathological sterility or HIV induced sterility, there is a wide range of possible determinants of fertility reduction: delayed marriage and onset of sexual relations, reduction in premarital sexual relations, non-marriage, increased divorce, increased widowhood, reduced remarriage, contraception, reduced desired family size, etc. (see Gregson, Zhuwau, Anderson and Chandiwana (1997)).

[12] The exception are those aged 15-19, in whom the selective pressure of sexual debut on pregnancy and HIV infection led to higher fertility rates among the HIV infected.

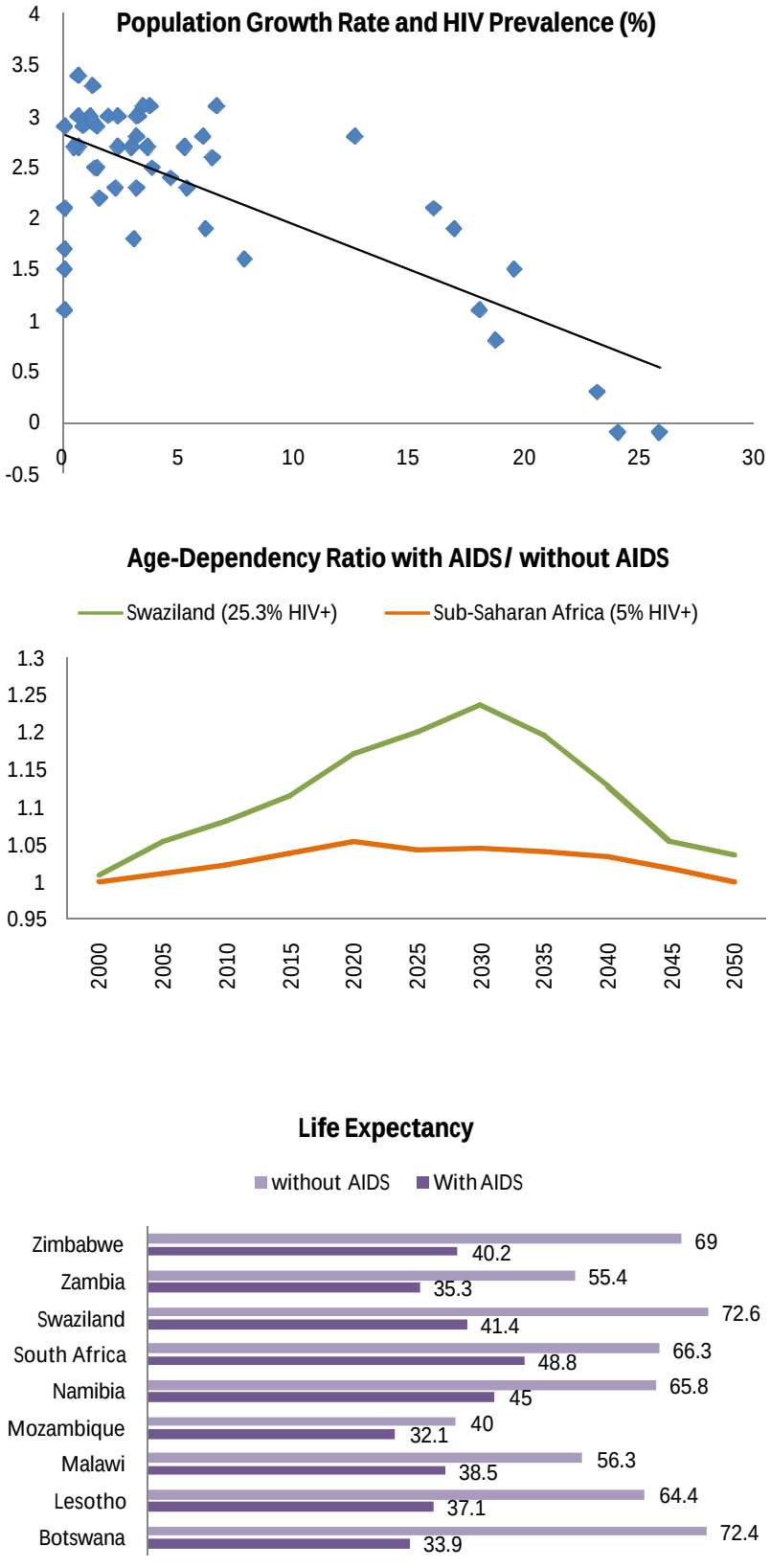

Figure 3: AIDS reduces population growth, raises dependency ratios and shortens life expectancy.

2.2 Age Distribution of the Population

The AIDS epidemic kills selectively, thinning the ranks of the most productive segment of the population in ages 15-49 years old. This shapes unambiguously the age composition of African societies and undermines the residual structure of families (since young adults are spouses and parents) raising the number of dependents (children and old adults) per worker by as much as 5% in SSA and 20-25% in highly infected countries such as Swaziland (see Jamison, Feachem, Makgoba, Bos, Baingana, Hofman, and Rogo (2006), Haacker (2004) and Greener (2004)).

While in developing African countries with low levels of HIV and AIDS, most deaths occur among the very young and very old, AIDS is reshaping the distribution of deaths by age and primarily strikes adults in their prime working-ages distorting the age structure of the population. [13] Because of the increasingly high AIDS-mortality in southern Africa, for example, people ages 20 to 49 accounted for almost three-fifths of all deaths in that region between 2000 and 2005, up from just one-fifth of all deaths between 1985 and 1990. This implies a raise in the dependency ratio, see the middle panel of Figure 3 in which the share of dependents, children and old adults, per worker raises above 20% in an scenario with AIDS for a highly infected country as Swaziland. [14]

2.3 Life Expectancy

The surge in AIDS deaths has also halted or reversed gains in life expectancy in many African countries. This substantial increases in adult mortality associated with AIDS reduces the average number of years a newborn can expect to live. This way, in the last 26, years countries like Botswana, Lesotho, Swaziland, Zambia, and Zimbabwe have also seen their life expectancies plunge to under 40, see the bottom panel in Figure 3. For example, in Lesotho, where an estimated 25% of all adults was living with HIV in 2006, life expectancy was nearly 60 years in 1995, but plummeted to 37.1 years by 2007, primarily because of AIDS-related mortality, while the UN projects Lesothos life expectancy would have improved to 64.4 had not been affected by the AIDS epidemics. Similar figures arise for the rest of Southern African countries. This implies a loss of as much as one third of individual's life time.

2.4 Labor Earnings

AIDS maims people. AIDS a slow killer, and before killing, especially the last two years of life, which is when AIDS develops, [15] AIDS shatters the labor efficiency of sick individuals. I compute this loss in individual labor efficiency by age, sex, marital status and HIV status using the Malawi

[13] The principal mode of HIV transmission in Malawi is heterosexual contact, which accounts for 90 percent of HIV infections in the country (UNAIDS (2007)). This is followed in importance by perinatal transmission (9 percent of all HIV infections). It is estimated that approximately 20 percent of babies born to HIV positive mothers will be infected around the time of birth. About one-half of children infected during the perinatal period will die before their fifth birthday.

[14] In addition, Appendix F I plot the age-pattern of the probability of dying and adult HIV prevalence (see Clark (2005)). For both sexes, as the HIV prevalence increases the probability of dying between ages 20 and 60 increases while the probability of dying at other ages remains little changed.

[15] The duration between HIV infection and the onset of AIDS varies but averages 9-10 years, and death typically ensues within 1-2 years of symptom onset.

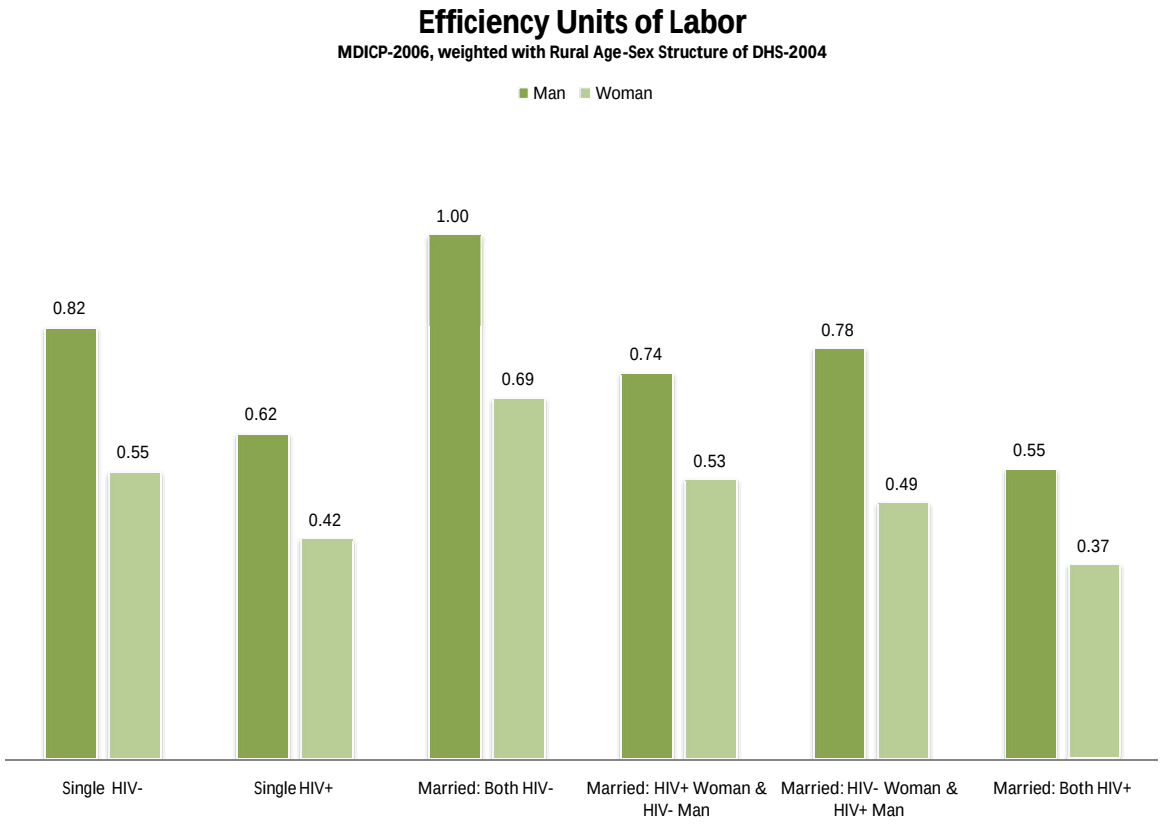

Figure 4: AIDS reduces labor efficiency units.

Diffusion and Ideational Change Project data set of 2006. The results are summarized in Figure 4 where I have normalized the efficiency units of the most efficient individual (an HIV- man married to an HIV- woman). [16]. The loss for men is larger than for women, one fourth against fifth. Looking at married couples, it is interesting to notice that the infection of either partner in a marriage drops the efficiency of the non-infected partner almost as much as if the odds had turned the other way round (so, married men depend a lot on the HIV status of their wives, and viceversa). That is, in a marriage, in terms of individual loss of efficiency, it does not really matter who gets infected, either the man or woman. The most dramatic drop in efficiency occurs when both partners are infected which almost halves the labor efficiency of both partners. Finally, on the aggregate I find that a society such as rural Malawi, with a mature AIDS epidemics with around 10.2% of HIV prevalence rate, losses as much as 2.7% of its earnings ability due to AIDS.

[16] A detailed description of this computation is in Section 6. I can not distinguish whether individuals are in the HIV stage without showing symptoms or have already developed AIDS from the data. One can imagine this loss is increasing and convex and parallel to the physical deterioration. My results, then, average the effects of the evolution of the disease.

3 The Model

3.1 Technology

I use the technology side in Hansen and Prescott (2002) theory of development. [17] This is a one-good model in which competitive firms decide the allocation of capital and labor across two sectors of production,

1. Malthus Sector

$$Y_m = A_m K_m^{\phi} N_m^{\alpha} L^{1-\phi-\alpha}$$

2. Solow Sector

$$Y_s = A_s K_s^{\theta} N_s^{1-\theta}$$

The Malthus sector uses three factors of production: capital, K_m, labor, N_m, and land, L, to produce output Y_m. The capital share of income in the Malthus sector is denoted by ϕ, the labor share by α, and the land share by $1-\phi-\alpha$. The total factor productivity of the Malthus sector grows exogenously at a rate γ_m, that is, $A_{mt} = A_{m0}\ \gamma_m^t$. The Solow sector uses the standard neoclassical production function with two factors of production: capital, K_s, and labor, N_s, to produce output Y_s. The capital share in the Solow sector is θ. The Solow sector will use capital more intensively setting $\theta \geq \phi$. The total factor productivity of the Malthus sector grows exogenously at a rate γ_s, that is, $A_{st} = A_{s0}\ \gamma_s^t$. Note that both technologies, Malthus and Solow, display constant returns to scale.

There are two key ingredients in this technology. Firstly, land is in fixed supply (normalized to one, $L = 1$), and hence, there are diminishing returns in capital and labor in the Malthus sector. Secondly, the Malthusian total factor productivity, A_m, grows slowlier than the total factor productivity of the neoclassical-industrial sector, A_s, that is, $\gamma_s > \gamma_m$.

Wealth in this economy is in the form of land and capital. Land is not reproducible and does not depreciate, and capital evolves over time following,

$$K_{t+1} = I_t - (1-\delta)\ K_t \tag{1}$$

where I_t is investement in capital, $K_t = K_{mt} + K_{st}$, and δ is the depreciation rate of capital.

Output from either sector can be consumed or invested in capital. The resource constraint for the economy is given by,

$$C_t + I_t = Y_{mt} + Y_{st} \tag{2}$$

Firms behavior is characterized by the following cost minimization problems:

[17] This technology has also been used, among others, in Parente and Prescott (2005) and Ngai (2004) to explain worldwide differences in income per capita, and explore the importance of technological barriers to growth.

1. Malthus Sector

$$c_m(w, r_K, r_L, A_m, Y_m) = \min_{K_m, N_m} \{r_K\ K_m + w\ N_m + r_L\ L\ :\ Y_m = A_m K_m^{\phi} N_m^{\alpha} L_m^{1-\phi-\alpha}, L = 1\}$$

2. Solow Sector

$$c_s(w, r_K, A_s, Y_s) = \min_{K_s, N_s} \{r_K\ K_s + w\ N_s\ :\ Y_s = A_s K_s^{\theta} N_s^{1-\theta}\}$$

where $c_m(.)$ and $c_s(.)$ are the Malthus sector and Solow sector cost functions respectively, and w is the wage, r_K is the interest rate on capital, and r_L is the interest rate on land.

Lemma 1. The Malthus sector is always operated. Lemma 1 in Hansen and Prescott (2002) For any set of factor prices, w and r_K, technology level, A_m, the marginal cost, MC_m, is always above the average variable cost, AVC_m,

$$\begin{aligned} MC_m(Y_m; w, r_K, A_m) &= \frac{1}{\phi+\alpha} A_m^{\frac{-1}{\phi+\alpha}} \left[\left(\frac{\phi}{\alpha}\right)^{\frac{\alpha}{\phi+\alpha}} + \left(\frac{\phi}{\alpha}\right)^{\frac{-\phi}{\phi+\alpha}} \right] w^{\frac{\alpha}{\phi+\alpha}}\ r_K^{\frac{\phi}{\phi+\alpha}}\ Y_m^{\frac{1-\phi-\alpha}{\phi+\alpha}} \\ &> (\phi+\alpha)\ MC_m(Y_m; w, r_K, A_m) = AVC_m(Y_m; w, r_K, A_m) \end{aligned}$$

That is, it is always profitable to operate the Malthus sector, $Y_{mt} > 0$ for all t. [18]

Lemma 2. Development is inevitable. For any given set of of factor prices, w and r_K, the Solow technology will start to operate when the Solow marginal cost of production is less than one (P=1),

$$1 \geq A_s^{-1} \left(\frac{w}{1-\theta}\right)^{1-\theta} \left(\frac{r_K}{\theta}\right)^{\theta}$$

where MC_s is evaluated at the Malthusian factor prices.

The results of Lemma 1 and Lemma 2 are illustrated in Figure 5 that depicts the marginal cost of production for both technologies. On the one hand, the marginal cost of production in the Malthus sector, MC_m, increases with output, because land is a fixed factor of production and diminishing returns to labor and capital apply. In addition, MC_m is convex if land is more intensively used than the other two factors, and concave otherwise, see Appendix G. We can also show that $MC_m(Y_m = 0) = 0$, and $MC_m(Y_m = \infty) = \infty$. On the other hand, the marginal cost of production in the Solow sector is strictly positive and constant. This implies that for any positive amount of output the Malthus sector will be always used to produce the first units of production because its marginal cost is lower than that of the Solow sector (Lemma 1). The possibilities of production change over time via increases in technology, capital and labor, and when the set of opportunities is large enough to produce output beyond the point where MC_m is above MC_s, the Solow sector starts to be operated (Lemma 2). This way, as output increases, the agricultural share of output decreases and converges to zero.

[18] Note that until we get to the point where $P(= MC) \geq$ *Average* $(Variable + Fixed)$ *Cost*, Malthus will be operating with negative profits, but still operating because the largest negative profits occure at $Y_m = 0$ due to the fixed factor of production, land, that has no other use out of the Malthus technology.

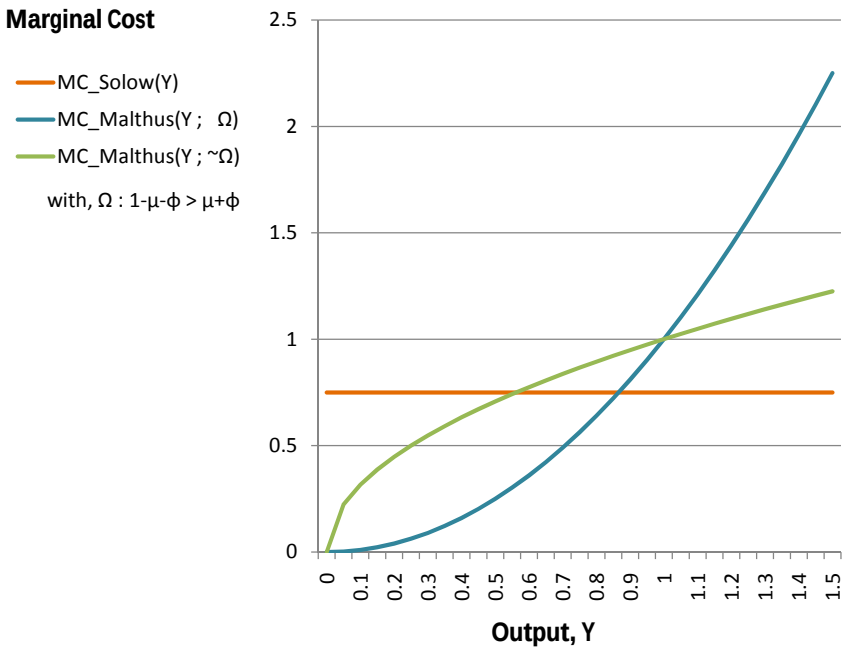

Figure 5: Marginal Cost of Production

Factor prices equate the marginal productivities across sectors,

$$w_t = (1-\theta)\, A_s\, K_s^{\theta}\, N_s^{-\theta} = \alpha\, A_m\, K_m^{\phi}\, N_m^{\alpha-1}\, L^{1-\phi-\alpha} \tag{3}$$

$$r_{Kt} = \theta\, A_s\, K_s^{\theta-1}\, N_s^{1-\theta} = \phi\, A_m\, K_m^{\phi-1}\, N_m^{\alpha}\, L^{1-\phi-\alpha} \tag{4}$$

$$r_{Lt} = (1-\phi-\alpha)\, A_m\, K_m^{\phi}\, N_m^{\alpha}\, L^{-\phi-\alpha} \tag{5}$$

An additional important result of this technology is that the capital-labor ratio is not a sufficient statistic to determine the input allocation across sectors, and hence, factor prices. To see this, given prices (3)-(5) and market clearing conditions, $K_t = K_{mt} + K_{st}$ and $N_t = N_{mt} + N_{st}$, the share of labor input in the malthus sector, $n_{mt} = \frac{N_{mt}}{N_t}$, is obtained by solving for n_m in [19]

$$G(n_{mt}; K_t, N_t) = \frac{1-\theta}{\alpha}\, \frac{A_{st}}{A_{mt}}\, K_t^{\theta-\phi}\, N_t^{1-\alpha-\theta}\, \psi^{-\phi}\, n_{mt}^{1-\alpha-\phi} - [1-(1-\psi)\, n_{mt}]^{\theta-\phi} = 0$$

By the mean value theorem we can show that there is a unique n_{mt} that solves $G(n_m; K, N) = 0$. Also, we can show that if $\frac{A_{St}}{A_{Mt}}\, K_t^{\theta-\phi}\, N_t^{1-\alpha-\theta}$ is increasing in t, n_m converges to zero.

This way, the time path of the agricultural share of output is a function of the aggregate level of the population, N_t, and the aggregate level of capital, K_t. These positive scale effects in terms of population and capital are present along the transition phase (from Malthus to Solow), first prominently at early stages of development, then dying out slowly as the Solow technology takes over the Malthus technology, and finally absent in the limiting industrial phase where the Solow technology is solely operated and the scale becomes irrelevant. This makes explicit the idea larger populations and capital can help, if not precipitate, economic development at the earliest stages of the process of industrialization, while they have minor

[19] See Appendix G for the analytical derivations of these results. See also Ngai (2004).

effects in already industrialized economies. Note the effect of the population is twofold: direct through N_t, and indirect through the determination of savings, K_{t+1}.

3.2 Demographics and Social Structure

I build an exogenous population process on the basis of a representative family (a continuum of identical families) that hosts individuals of three different age groups $i \in \{c, y, o\}$. These age groups respectively represent childhood (<15 years old), young adulthood (15-49 years old) and old age ($\geq$ 50 years old).

Formally, a family is a vector

$$\mu_t = (N_t^c, N_t^y, N_t^o)'$$

where N_t^i is the number of members of age group i.

To keep track of the evolution of the population over time I formulate a population matrix model that relates the age distribution of the population of each period to the preceding as follows,

$$\mu_{t+1} = \Gamma_t \, \mu_t \tag{6}$$

The time-varying population (projection) matrix or law of motion of the population, Γ_t, is composed by a mortality process, a fertility process and an ageing process as follows,

$$\Gamma_t = \underbrace{\begin{bmatrix} 1-\pi_t^c & 0 & 0 \\ \pi_t^c & 1-\pi_t^y & 0 \\ 0 & \pi_t^y & 1-\pi_t^o \end{bmatrix}}_{\text{Ageing Matrix}} \times \underbrace{\begin{bmatrix} \gamma_t^c & 0 & 0 \\ 0 & \gamma_t^y & 0 \\ 0 & 0 & \gamma_t^o \end{bmatrix}}_{\text{Mortality Matrix}} \times \underbrace{\begin{bmatrix} 1 & \phi_t^y & \phi_t^o \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}}_{\text{Fertility Matrix}} \tag{7}$$

where the series π_t^i denotes the aging rate at which individuals in age group i transit to the next age group between period t and $t+1$, the series γ_t^i are the survival rates (one minus mortality rates) at which individuals individuals in age group i survive between period t and $t+1$, This way, the first matrix in the right hand side of (7) is the ageing matrix, Π_t, the second is the mortality matrix, Γ_t^M, and the third is the fertility matrix, Φ_t.

The matrix ordering in the right hand side of (7) establishes the timing of events. Firstly, the young and old adults are subject to a fertility process by which they give birth at rate ϕ_t^y and ϕ_t^o. Then, all individuals in age group i are subject to a mortality process, Γ_t^M, by which they may die at rate $1-\gamma_t^i$. Finally, all members within the household are subject to an ageing process, Π_t, that carries individuals from childhood to young adulthood at rate π_t^c, from young adulthood to old age at rate π_t^c, and from old age to death at rate π_t^o. [20] It is perhaps useful to illustrate the population model with a life cycle graph isomorphic to the population projection matrix (7), see Figure 6.

[20] Also, individuals can not age from childhood to old age, or from young adulthood to death in a single step of the aging process and neither do they become younger over time.

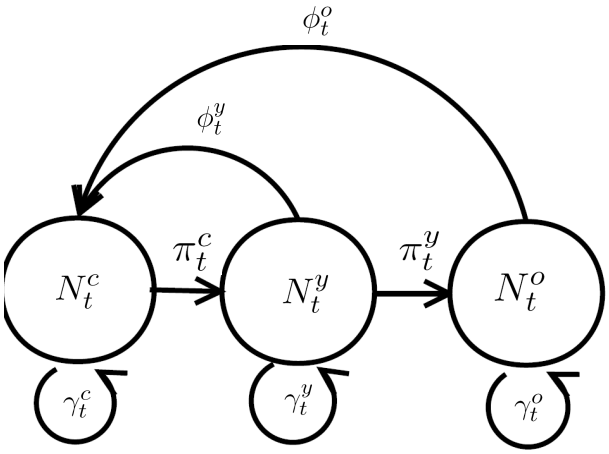

Figure 6: Life Cycle Graph

For instance, the amount of young adults in $t+1$, N_{t+1}^y, is given by the amount of children in t, N_t^c, that survived and aged, $\gamma_t^c\ \pi_t^c\ N_t^c$, plus the number of births (that survived and aged) born to young adults in t, $\phi_t^y\ \gamma_t^c\ \pi_t^c\ N_t^y$, plus the number of young adults that survived and did not age in t, $\gamma_t^y\ (1-\pi_t^y)\ N_t^y$, plus the number of births (that survived and aged) born to old adults in t, $\phi_t^o\ \gamma_t^c\ \pi_t^c\ N_t^o$ (see Appendix C.1).

This way, the population model (6) and (7) specifies a population process in which age intervals do not coincide with time (projection) intervals. That is, survivors may see time pass by without aging. This falls in the category of population models usually denoted as size-classified life-cycle models (see Keyfitz and Caswell (2005)). This specification allows me to consider shorter time periods than the age intervals associated with the three age groups distinctively affected by AIDS that I am modelling (a grouping for which most of the AIDS data is gathered), and it contrasts with the standard modelization of overlapping generations (OLG) where the age interval is equal to the time (projection) interval (that is, the mortality process is the ageing process),[21] see Blanchard (1985). [22]

Importantly, this specification of the population process captures the main channels through which AIDS distorts populations over time, in particular, the time path of the rate of growth of the population, the age dependency ratio and life expectancy. All these are population statistics that we can compute using time series of fertility rates, ϕ_t^i, mortality rates, γ_t^i, and, ageing rates, π_t^i, to iterate forward any initial distribution of the population μ_0 using (6) and (7):

1. Population Growth Rate. The total population is the sum of all members in each age-

[21] Indeed, the population matrix (7) is very different from the standard (Leslie matrix) used in age-classified models where the age interval does coincide with the projection interval (see, for instance, Ríos-Rull (2001)). In those models, age-specific fertilities appear in the first row of the population matrix and age-specific survival rates in the subdiagonal. However, in my model, fertilities appear in the subdiagonal while survival rates in the diagonal, because although agents may survive to next period they may remain in the age group.

[22] Besides the fact most of the AIDS data is gathered in those three age groups, I find my set up particularly useful over the standard OLGs for the study of the AIDS epidemic (a process that has build up smoothly and slowly during the last 25 years) because this way I do not have to cut off time data points (as it would be case if I used a standard OLG with age intervals larger than 1 year: for example, if I used five years intervals in a standard OLG, I would only have 5 time data points of the epidemic from 1981 to 2006), neither I have to keep track of the whole population, a computationally dantesque task and detail for which we actually have no data.

group, $N_t = \sum_i N_t^i$, then the rate of growth of the population is computed as $\frac{N_{t+1}-N_t}{N_t}$.

2. Age dependency ratios. The children dependency ratio, number of children per worker (young adults), $\frac{N_t^c}{N_t^y}$, and the old age dependency ratio, number of old adults per worker, $\frac{N_t^o}{N_t^y}$.

3. Life Expectancy. The expectancy of life (or time to death) is computed using the fundamental matrix of the ageing process times the mortality process, $N_t = I + \left[\Pi_t \times \Gamma_t^M\right] + \left[\Pi_t \times \Gamma_t^M\right]^2 + \left[\Pi_t \times \Gamma_t^M\right]^3 + \cdots = (I - \left[\Pi_t \times \Gamma_t^M\right])^{-1}$. The element $N_t(1,1)$ is the average duration (in years) an individual remains as a child if the mortality and ageing rates remain as they are in t forever. The element $N_t(2,1)$ is the average duration an individual is a young adult, and $N_t(3,1)$ represents the average duration an individual is an old adult. Then, the life expectancy (at the time of birth) in period t is the sum of the elements of the first column in N_t. This yields an expression for the life expectancy as a function of the mortality rates and the ageing rates (independently of the population structure). [23]

I will model the AIDS epidemic as a shock that changes the fertility rates from ϕ_t^i to $\phi_{A,t}^i$, and the mortality rates from $1-\gamma_t^i$ to $1-\gamma_{A,t}^i$ in a way that I specify below (see Section 6). This way, AIDS will reshape the law of motion of the population from Γ_t to $\Gamma_{A,t}$, generating an alternative population path, $\mu_{A,t}$, with subsequently distorted population growth rates, age dependency ratios and life expectancy.

To explore the consequences that the populations shocked by AIDS have on development, I cast the families problem sequentially in the next section.

3.3 Household Problem

This is an overlapping generations model where a representative family hosts three generations that represent childhood, young adulthood and old age. This family is a unitary household in the sense that there is total agreement on the decision making (that is, there are not strategic decisions across generations), in this case, granted to young adults. Moreover, families have perfect foresight of the exogenous population process given by (6) and (7).

Young adults solve,

$$\max_{c_t \geq 0, a_{t+1} \in A, s_{Kt} \in [0,1]} \sum_{t=0}^{\infty} \beta^t \; N_t^y \left[u^y \left(\frac{c_t}{\eta_{y,t}} \right) + \beta \; \gamma_t^y \left(\pi_t^y \; u^o \left(\frac{c_{t+1}}{\eta_{o,t+1}} \right) + (1 - \pi_t^y) \; u^y \left(\frac{c_{t+1}}{\eta_{y,t+1}} \right) \right) \right] \tag{8}$$

subject to the budget constraint,

$$c_t + a_{t+1} = w_t N_t^y + \left((1 + r_{Kt} - \delta) \, s_{Kt} + \frac{q_t + r_{Lt}}{q_{t-1}} (1 - s_{Kt}) \right) a_t$$

[23] The explicit analytical expression for the life expectancy is given in the Appendix C.2

Families maximize the sum of future utility discounted at a factor β. The instantaneous utility function of young adults is u^y. If young adults survive to next period, which happens at rate γ_t^y, and age, at rate π_t^y, they will value consumption with the utility function of old adults, u^o. Family consumption, c_t, is scaled by equivalences, $\eta_{y,t}$ for young adults and $\eta_{y,t}$ for old adults, that are functions of the family composition, $\mu_t = (N_t^c, N_t^y, N_t^o)'$.

The individual state of the family is its wealth, a_t, and its demographic composition, μ_t. The aggregate states of the economy are aggregate capital and the distribution of the population across age groups. Note that since there is a representative family, the family composition, μ_t, is the distribution of the population across age groups.

A family chooses how much to consume, c_t, how much to save, a_{t+1}, and the share of total wealth held in capital, s_{Kt}, and land, $1 - s_{Kt}$. The share s_{Kt} is chosen to equate the return on both assets, capital and land, at each period. This way, the portfolio decision is trivial and the only individual state is total wealth. The return on capital is $1 + r_{Kt} - \delta$ where δ is the depreciation rate, and the return on land is $\frac{q_t + r_{Lt}}{q_{t-1}}$ where q_t is the price of land in units of consumption.

Labor is inelastically supplied. Only young adults work and earn wages, w_t.

The household problem is characterized by the following conditions:

1. First order condition of s_k: A first order difference equation in q_t that equates the returns for capital and land,

$$(1 + r_{Kt} - \delta) = \frac{q_t + r_{Lt}}{q_{t-1}} \tag{9}$$

2. Firs order condition of a_{t+1}: The Euler equation for young agents,

$$\frac{\partial u^y(c_t)}{\partial c_t} = \beta\, \gamma_t^y\, (1 + r_{Kt+1} - \delta)\, \{\pi_t^y\, \frac{\partial u^o(c_{t+1})}{\partial c_{t+1}} + (1 - \pi_t^y)\, \frac{\partial u^y(c_{t+1})}{\partial c_{t+1}}\} \tag{10}$$

Thus, choosing a large enough T that ensures convergence to the Solow balanced growth path, the household problem is summarized by a system of $2 \times (T + 1)$ equations, (9)-(10), with $T + 1$ unknown prices q_t and $T + 1$ unknown allocations a_{t+1}.

4 Equilibrium

A Malthusian stationary equilibrium in which only the Malthus sector is operative, and a limiting Solowian stationary equilibrium, or balanced growth path, in which the share of output produced with Malthusian technology converges to zero, can be easily defined and solved. However, my central interest is the process of development and for this the evolution of the population and of savings have to be jointly determined along the transition from Malthus to Solow. To do so I will cast the equilibrium sequentially.

Equilibrium. Given the initial capital stock, K_0, the amount of land $L_0(= 1)$, the initial distribution of the population, μ_0, and, the sequences of the population law of motion,

$\{\Gamma_t\}_{t\geq 0}$, a competitive equilibrium in this economy consists of sequences of prices $\{q_t\}_{t\geq 0}$ and $\{w_t, r_{K,t}, r_{L,t}\}_{t\geq 0}$, sequences of firm allocations, $\{K_{mt}, K_{st}, N_{mt}, N_{st}, Y_{mt}, Y_{st}\}_{t\geq 0}$, sequences of household allocations, $\{c_t, a_{t+1}, s_{Kt}\}_{t\geq 0}$ such that the following are true:

1. Firms optimize: $w_t, r_{K,t}$ and $r_{L,t}$ equate marginal productivities across sectors, (3)-(5).

2. Households optimize: c_t, a_{t+1}, s_{Kt} solve the utility maximization problem, (8).

3. Goods market clears, and

$$K_{mt} + K_{st} = K_t$$
$$N_{mt} + N_{st} = N_t$$

4. Population evolves according to,

$$\mu_{t+1} = \Gamma_t \, \mu_t$$

5 Calibration

5.1 Technology and Preference Parameters

I calibrate the Malthus sector to a North-African country unaffected by the AIDS epidemic, Egypt, which is also a late starter as the SSA countries affected by AIDS. In the current version of the paper I have not obtained capital and labor shares for Egypt before the country took off. This way, I am currently using the capital and labor share associated with the Malthus sector for England 1750 (as in Hansen and Prescott (2002)).

The Malthusian TFP growth rate, γ_t^m, is calibrated such that income per capita growth is zero when only the Malthus sector is operated. Basically, population is limited by the available technology, so that the growth rate of population is proportional to the growth rate of technology, as assumed by Malthus (1798).

To calibrate the Solow sector I take South Africa, the most developed African country (the one most developed country in Africa) as the paradigm to which SSA converges. This way, I am giving Egypt a chance of doing well, that is, to reach modern-growth fast (by loering the standards of the modern-growth rate). [24] [25] The South Africa capital share is .29, and the South Africa investment share and capital output ratio yield a depreciation rate of .08 and a discount factor of .96. The South Africa TFP growth for the past 15 years averages 1.85.

[24] This is consistent with the finding of Parente and Prescott (2005) that countries that first achieved a certain level of income ($2000) later in history were able to double their income (to $4000) in a far shorter period than countries that achieved this level of income before.

[25] Disparities in income levels and convergence in growth rates with other regions of the world is not an issue I tackle here. In terms of relative convergence within its own regions, South Africa is the Germany of Africa. Malawi is the Spain of Africa. So imagine I am modelling the Spanish development path, I am going to target convergence modern-growth rate to Germany.

	Parameter	Value	Target
Malthus Sector			
	μ	.6	England 1750 labor share (Egypt in 1950)
	ψ	.1	England 1750 capital share (Egypt in 1950)
	A_{m0}	1.0	Normalization
Solow Sector			
	θ	.29	South Africa capital Share
	γ^s	1.85	South Africa TFP growth
	δ	.08	South Africa Depreciation
	β	.97	South Africa K/Y
	A_{s0}	.45	Matches agricultural share of Egypt in 1990

Note: Malthus refers to pre-take off data respectively for each country.

Table 1: Calibration

I will use logarithmic utility function [26]. In the current version of the paper I assume that $u^y = u^o$, and $\eta_{y,t} = \eta_{o,t} = \eta_t$.

I use OECD equivalence scales,

$$\eta_t = .5\ N_t^c + N_t^y + .7\ N_t^o \tag{11}$$

5.2 Population Path without AIDS

I incorporate exogenously the population paths of Egypt in the model, $\mu_t = (N_t^c, N_t^y, N_t^o)$. The population path of Egypt, is taken for U.N. Population Division with the actual pre-2006 population groups plus post-2006 U.N. population projections. The properties of this path are depicted in Figure in terms of the population growth rate and age structure.

6 Specification of the AIDS Shock

The current version of the paper identifies the effect of AIDS directly on the population growth rate and on the dependency ratio using data from the Population Reference Bureau and projections from United Nations Population Division. In addition I compute the loss due to AIDS in the efficiency units of labor using the 2006 Malawi Diffusion and Ideational Change Project data set. I am currently working on the identifying AIDS alters fertility rates and mortality rates by age groups (with the data depicted in Appendix F) in order to use fully the population model posed in (6) and (7) to derive how AIDS changes the population growth rate, the population structure and life expectancy. The latter identification has the advantage

[26] Increasing the intertemporal elasticity of substitution makes growth faster. However this does not change much my results since the spirit of my exercise is the comparison between development paths where the utility function is the same.

that incorporates life expectancy into the analysis and provides a structured decomposition of the effects of AIDS on development, that is, it allows me to isolate the effects of the growth rates, population structure and life expectancy on development. [27] I do not expect my latter identification to change substantially my findings in Section 7 which currently use the direct identification strategy that explain next.

6.1 The Population Growth Rate

AIDS reduces population growth. I incorporate this reduction by directly reducing the population growth rate of the Egypt population path using the cross-sectional relationship between the growth rate and the HIV prevalence rate that I depicted in the top panel of Figure 3 (data from Population Reference Bureau for 2005),

$$\gamma^i_{A,t} = \gamma^i_t - .08\ HIV^i_t$$

where HIV^i_t is the HIV prevalence of an infected country i. The projections of the HIV prevalence rates, HIV^i_t, of Lesotho and Malawi with which I will infect Egypt are in the Appendix D.

6.2 Age Dependency Ratio

AIDS increases the dependency ratio, that is, the share of dependents, $N^c_t + N^o_t$, per worker, N^y_t. I modify the Egypt AIDS-free dependency ratio, D_t, to an 'infected' dependency ratio, $D_{A,t}$, using the U.N. AIDS versus No-AIDS scenario projections for these dependency ratios, **dependency ratio**

$$D_{A,t} = D_t + \nu_t\ D_t\ \frac{HIV^i_t}{HIV^{Swaziland}_t} \geq \frac{N^c_t + N^o_t}{N^y_t} = D_t$$

where ν_t is the ratio of the AIDS versus No-AIDS scenario dependency ratio for Swaziland according to the U.N, which I depicted for Sub-Saharan Africa and Swaziland in the center panel of Figure 3. The projections of the HIV prevalence rates, HIV^i_t, of Lesotho and Malawi with which I will infect Egypt are are in the Appendix D.

6.3 Efficiency Units of Labor

Here I quantify the aggregate loss in labor efficiency, a combination of size and quality of hours, due to AIDS using the MDCIP-2006 weighted with the age-sex structure of the rural sample in the Malawi Demographic and Health Survey 2004.

On the one hand, in the data people differ by individual characteristics such as (age, sex, marital status, health status, number of children...) and by aggregate characteristics

[27] This way, this version is silent about the effect of AIDS on the expectancy of life which I will incorporate shortly. Note, that in order to incorporate the effect of life expectancy, I need to identify the AIDS shocks via mortality rates as I have formulated in Section 3.2. Then, I can compute the life expectancy as formulated in Appendix C.2 and evaluate how changes in mortality, and in turn, life expectancy alter the development path. This will be available soon, along with the subsequent decomposition of effects: growth rates versus age structure versus life expectancy versus efficiency units of labor.

(infrastructures, region...) that can help to determine individual's j labor earnings, $w_t^j\ h_t^j$, where w_t^j are wages and h_t^j the labor supply. That is, not only people differ in the amount of hours that they work (size) but also these hours do not worth the same for every individual. One would expect a sick individual to produce less than a healthy individual, and then, be rewarded accordingly), a quality that is reflected in the wages. On the other hand, in my model a set of identical young adults supply labor inelastically, N_t^y, to earn identical wages, w_t. In order for my model to inherit the properties of the data regarding the loss in labor earnings, I define labor earnings as, $w_t\ \epsilon_t$, where ϵ_t is the sum of all individual's labor efficiency grouped by own HIV status, spouse HIV status (includes marital status), age and sex,

$$\epsilon_t = \sum_g N_t^g\ \epsilon_t^g \tag{12}$$

where N_t^g is the number of individuals in group g, and ϵ_t^g is the labor efficiency of one individual in group g. [28]

The data counterpart of labor earnings I am interested are all sources of income that use labor as the main input. I will use the reported 'earnings' in Kachawa (cash) and the reported agricultural production in Kachawa of individuals in the MDICP-2006. To learn the variation of earnings across individuals I will apply a statistical Heckman selection model [29] to my definition of labor earnings. I specify labor earnings as depending on own HIV status and controlling for HIV status of the spouse, sex, number of children, age, age squared, education, education squared as variables that determine labor earnings, and, spouse earnings, age and education as the exclusion restrictions that determine the decision to work. [30]. The results of this exercise for the individual labor earnings have been discussed in Section 2.4 and summarized in Figure 4. [31]

Using (12), the results reported Figure 4, and the demographic structure of rural Malawi, I obtain an aggregate loss of labor efficiency of 2.7%. [32] Given that rural Malawi in MDCIP has an HIV prevalence of 10.2%, and assuming linearity in this relationship, this means that for every percent point of HIV prevalence, aggregate labor efficiency reduces by 0.27%, and I

[28] This follows the spirit of the real business cycle literature in which the aggregate labor input is the sum of individual hours quality-adjusted by wages, see Kydland and Prescott (1993) and Hansen (1993).

[29] This statistical procedure allows me to correct for sample bias. One can imagine that sick individuals do not go to work, but that does not mean that their earnings would be zero had they gone to work.

[30] Since earnings depend on the amount of hours worked I also include in the selection equation all variables that determine earnings conditional on working.

[31] The output of the selection model is,

$Earnings$	C	HIV	HIV^S	Sex	Age	Age^2	$Children$	$Educ$	$Educ^2$
	10.12^*	$-.44^*$	$-.32^{**}$	$-.22^*$	$.03^*$	$-.0005^*$	$.05^*$	$.14^*$	$-.006$
	(.36)	(.21)	(.18)	(.09)	(.02)	(.00)	(.02)	(.05)	(.01)

Own HIV status and all controls, except for education squared, turn out to be significant.

[32] Previous studies have focused mainly on the effects of HIV/AIDS on the labor supply (and absenteeism) at the micro level. Exploring the progression of the disease of tea estate workers in Western Kenya between 1997 and 2001, Fox, Rosen, MacLeod, Wasunna, Bii, Foglia, and Simon (2004) find in a nonrepresentative survey that during the last two years of their lives AIDS adults earn about 16% less than before developing AIDS and have about 40 days more leave days. Similarly, Cohen (2002) reports that AIDS workers of fisher factories in Lake Malawi work 55 days less per year than healthy individuals. Thirumurthy, Zivin, and Goldstein (2005) find a positive impact of AIDS treatment on labor supply. Also, Dorward, Mwale, and Tuseo (2006) provide evidence of HIV/AIDS leading to a fall in unskilled wages in Rural Malawi with Integrated Household Survey Data 1997/1998.

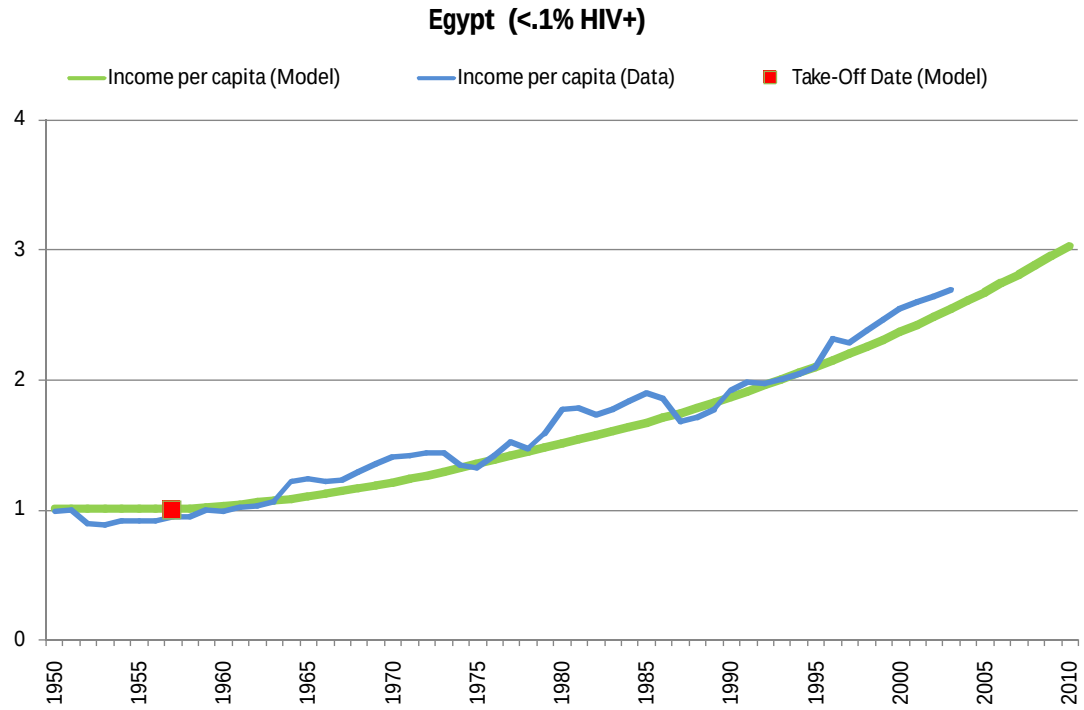

Figure 7: Income Per Capita, Model and Data.

specify this AIDS shock as follows,

$$\epsilon^i_{A,t} = \alpha_t \; N^y_t \qquad \text{with } \alpha_t = 1 - .027 \; \frac{HIV^i_t}{HIV^{Malawi}_{2006}}$$

7 Findings

7.1 Development Path without AIDS

In Figure 7 I depict the income per capita path of Egypt, data (blue line) and model (green line). The growth take off date ensures that the agricultural share of output in the data is equal to model in 1990.

7.2 Alternative Population Path with AIDS

Figure 8 depicts the data population path of Egypt using the pre-2006 data plus the post-2006 U.N. projections (blue line). When I introduce Lesotho's AIDS I find the population growth

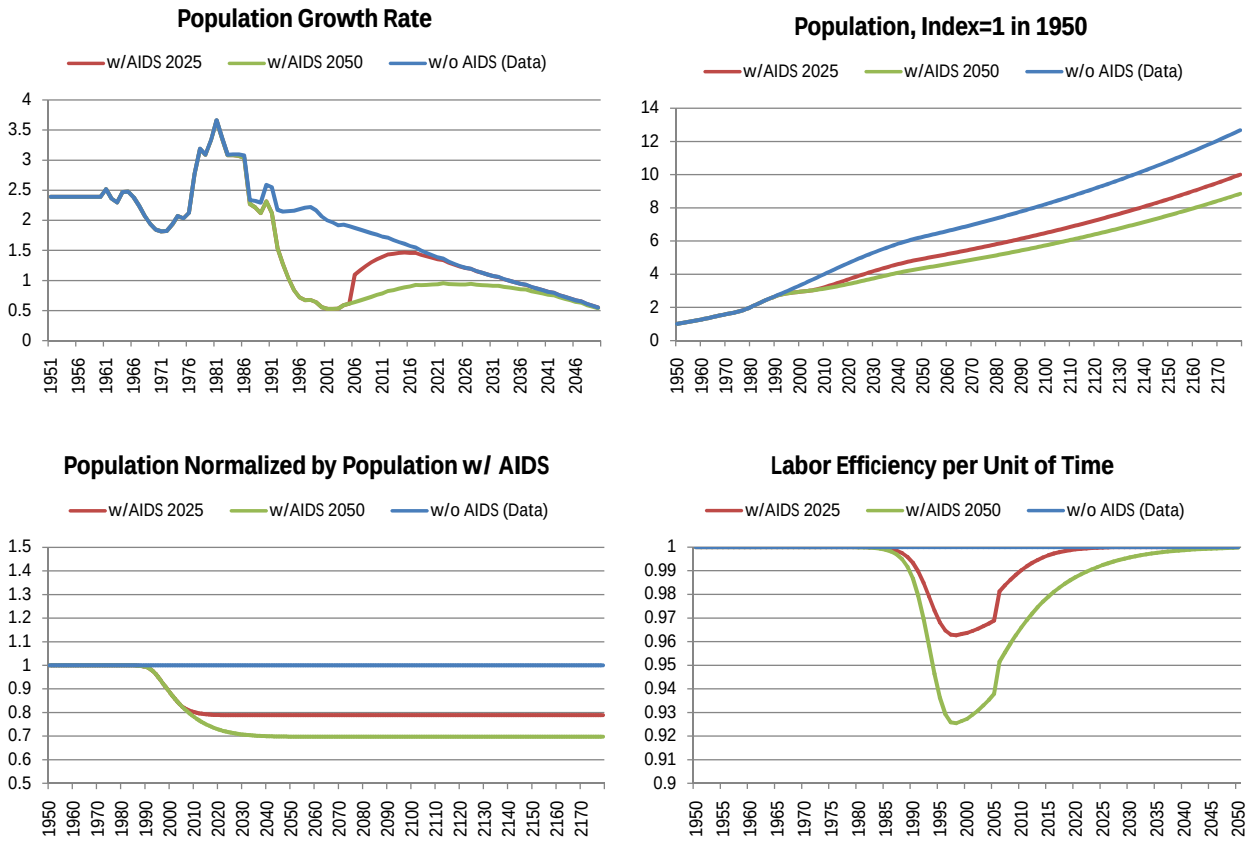

Figure 8: Population Path: Infecting Egypt's Population in 1981 with Lesotho's AIDS

rate of Egypt (green line in top-left panel of Figure 8) when AIDS is assumed to vanish in 2050 falls to a value of 1.5% below the data at the peak of the epidemics in 2003. In all the population of Egypt with AIDS (top-right panel of Figure 8, and detrended with respect to the no-AIDS scenario in the bottom-left panel) is around 30% less of the actual population without AIDS. The bottom-right panel shows the loss in labor efficiency units of Egypt had it been infected with Lesotho's AIDS (green line). I find a maximum loss of 7% at the peak of the epidemics.

The consequences for economic development of incorporating this alternative population path with AIDS are discussed next.

7.3 Alternative Development Path with AIDS

Here I surprise Egypt in 1981 with Lesotho's AIDS. That is, I incorporate into the modelled Egypt in 1981 the population path with AIDS described above in the previous subsection.

Egypt in 1981 was 22% agricultural in terms of output, that is, relatively advanced in the process of industrialization.

I find that output per capita in the AIDS scenario (green line in the top-left panel in Figure 9) falls below the no-AIDS scenario (blue line). This is perhaps better observed if we normalize the AIDS path with the no-AIDS path (top-right panel in Figure 9). Output per capita falls by about 9% at the peak of the epidemics when the economy has AIDS with respect to the no-AIDS scenario. The process of industrialization is delayed by as much as 45 years, see the bottom-left panel in Figure 9 that depicts the agricultural share without AIDS minus the agricultural share with AIDS. These results represent a combination of the specification of the AIDS shock and the development stage in which Egypt was when I have infected it with AIDS.

I have hit by surprise Egypt in 1981 with the perfect foresight of an AIDS population path that will reduce its population slowly over time (reaching a minimum around 25 years after I hit them with the AIDS shock) and increase the age-dependency ratio. The fact that AIDS is a slow process, means that agents have time to slow down their accumulation of capital close to their optimal level when the the peak of the expansion arrives. Reducing population, we are also reducing savings, in particular with the presence of a distorted age structure that raises the consumption income ratio to strain further capital accumulation. Fewer workers and fewer savings reduce aggregate output further than the reduction associated with the population (which decreases less than the labor force). Further, this economy is in the process of industrialization, and since aggregate population and aggregate savings determine the speed of the industrialization, if reductions in both capital and labor due to AIDS are not offset by improvements in efficiency, AIDS slows down the transition from agriculture to industry. In this case, 45 years, because the economy is already relatively advanced in the process of industrialization and its levels of capital and efficiency are already large.

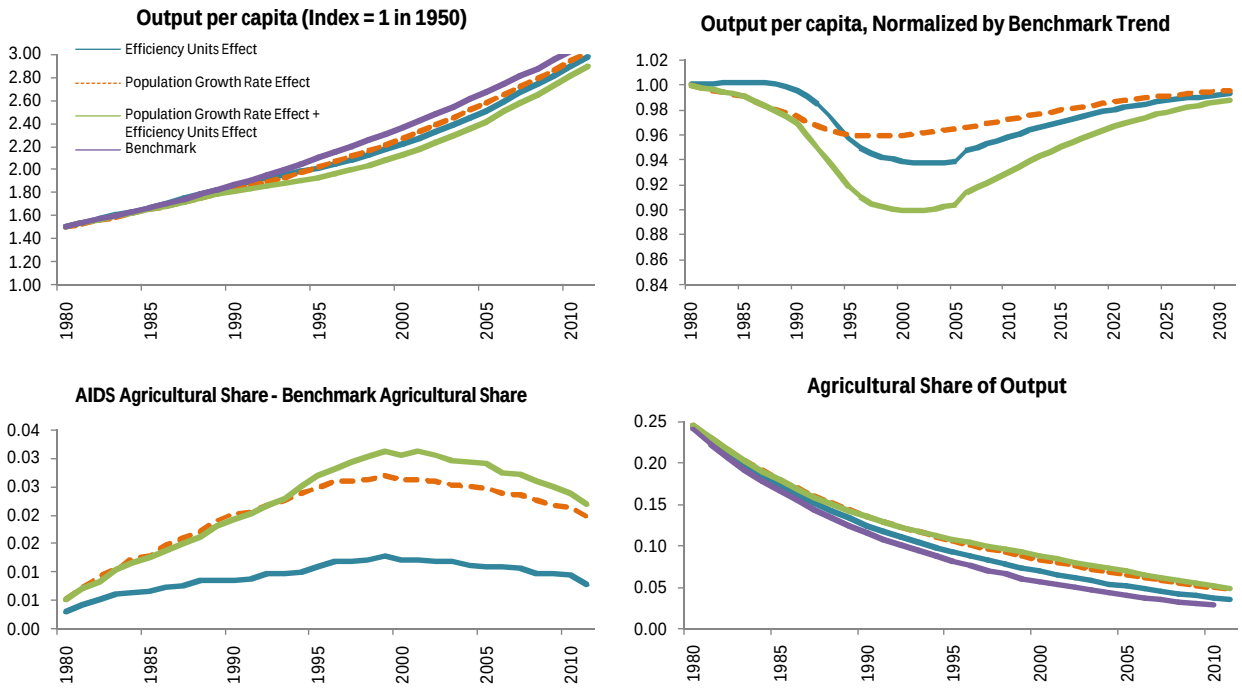

Figure 9: Development Path: Infecting Egypt's Population in 1981 with Lesotho's AIDS

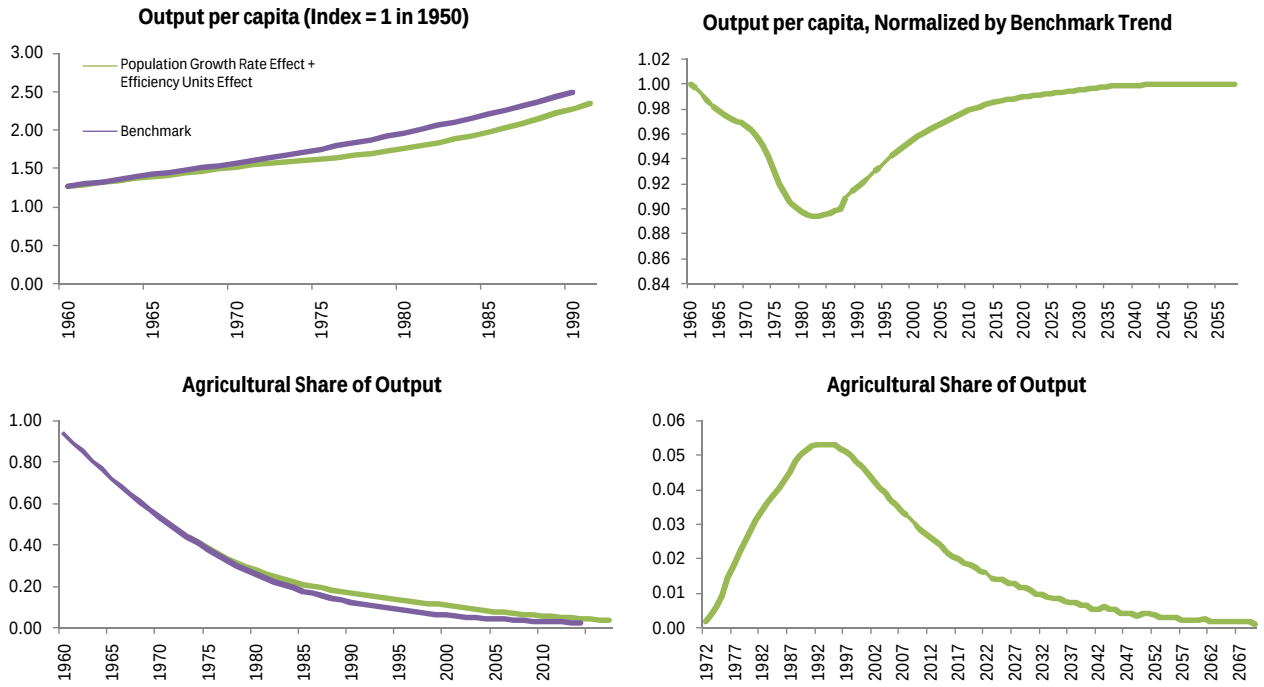

Figure 10: Development Path: Infecting Egypt's Population in 1972 with Lesotho's AIDS

In Figure 10, I show the development path that results from infecting Egypt in 1972 with Lesotho's AIDS in 1981. Egytp was 43% agricultural in 1972, as Lesotho was in 1981 when Lesotho got hit by the epidemics. This is, I think of Egypt in 1972 as Lesotho in 1981. In this case output per capita falls close to 11% at the peak of the epidemic (see top-right panel in Figure 10). The industrialization process gets delayed by about 60 years with maximum difference with respect to no-AIDS scenario of 5% more of agricultural share at the peak of the epidemics (see bottom-right panel in Figure 10). In the previous example Egypt was more advanced in the industrialization process with a 22% agricultural share of output and the adverse effects of AIDS were less severe. This reflects the fact that the effect of AIDS on development does depend on the stage of development, because the less developed countries are, the less efficiency and savings have to offset loses in population.

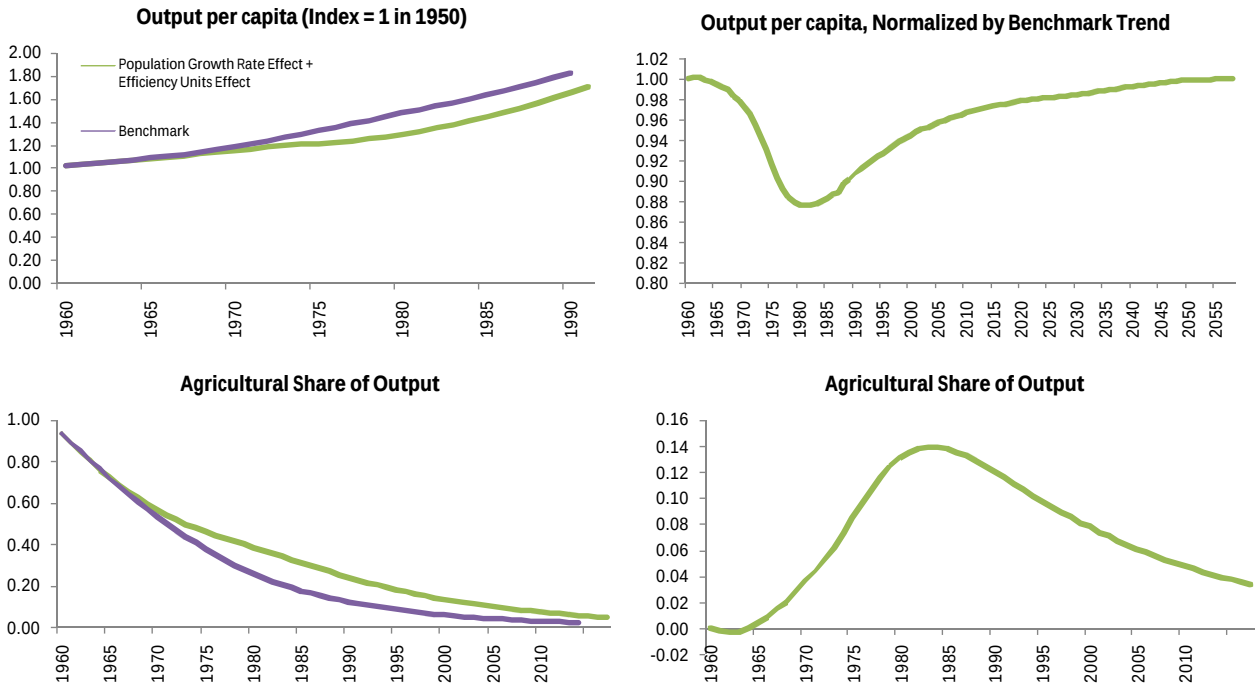

Figure 11: Development Path: Infecting Egypt's Population in 1964 with Lesotho's AIDS

In Figure 11, I show the development path that results from infecting Egypt in 1964 with Malawi's AIDS in 1981. Egypt was 72% agricultural in 1964, as Malawi was in 1981 when Malawi got hit by the epidemics. This is, I think of Egypt in 1964 as Malawi in 1981. In this case output per capita falls close to 12% at the peak of the epidemic (see top-right panel in Figure 10). The industrialization process gets delayed by as much as a century with maximum difference with respect to no-AIDS scenario of 13% more of agricultural share at the peak of the epidemics (see bottom-right panel in Figure 11). This emphasizes the role of the development stage in the understanding of the effects of AIDS on development.

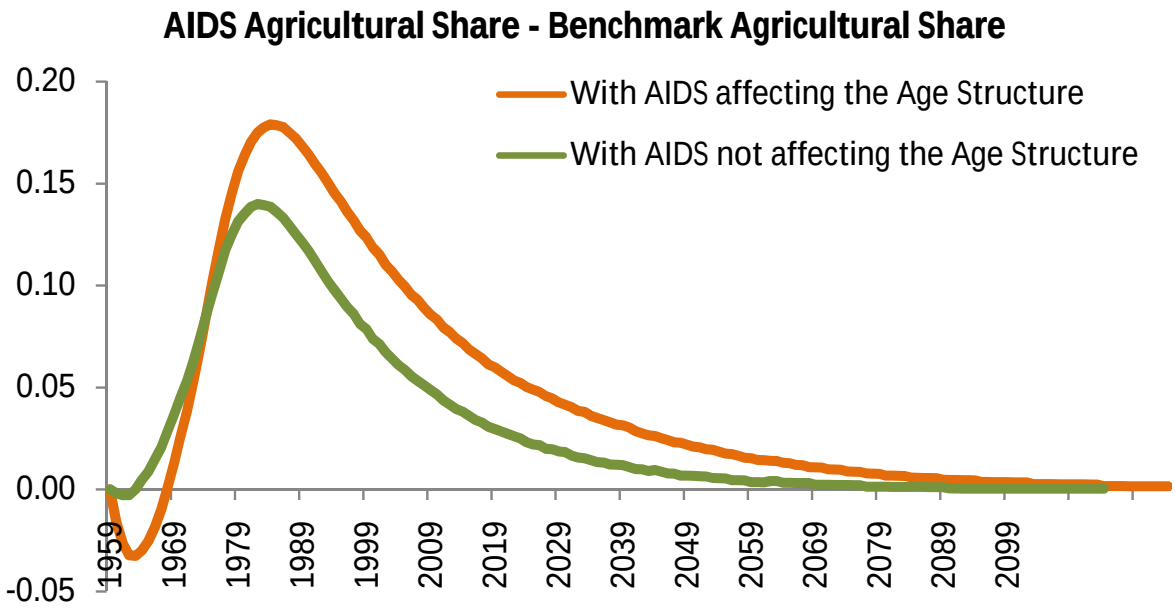

Figure 12: Impact of the Age Structure: Industrialization Delay

In Figure 12, I show how the change in the age composition due to AIDS (raising the consumption to income ratio) extends the adverse effects that AIDS has on development. Figure 12 shows the effects on the process of industrialization of incorporating the AIDS epidemic to Egypt in 1959 when I do not allow AIDS to affect the dependency ratio (green line) and when I do allow AIDS to affect the dependency ratio (orange). We observe that the effects of AIDS on the age structure of the population extend the delay of the process of industrialization by about 32%.

8 Misspecification of the AIDS shock: The Black Death

Previous work on the effect of the AIDS epidemic on development has built on the idea of that AIDS has some similarities with the Black Death. The Black death is a external disturbance that swaps population rapidly and does not discriminate, killing individuals in all age groups. In this section I exemplify the effects of a such a demographic disturbance posing a shock that kills overnight (by surprise) half of the population and that does not make distinction by ages. Therefore, here I simplify my previous analysis and shut down the age structure in the model assuming a representative agent rather than a representative family.

The effect of the Black Death on an economy that has completed their process of development, hence, an economy that is along the Solow balanced growth path is plotted in Figure 13. Decreasing the population by half at one point in time (top-left panel in Figure 13), and by surprise, that is, not allowing the agents to modulate their investment, raises income per capita (top-right panel in Figure 13). There is a sudden excess of available resources, capital, for the survivors. Importantly, the effect of the Black Death on the process of industrialization is negligible (see bottom-right panel in Figure 13). The process of industrialization stands through the level of technology, aggregate capital and population. It is the case then, that in an already developed economy there is enough technology and capital (even though agents start to run it down to adjust for the new optimum) to offset a Black Death population shock.

However, the effect the Black Death on an economy that is at the earliest stages of development is sizeable, as depicted in the bottom-right panel in Figure 14. In this case, the economy has not had time to build up their stock of capital nor the level of technology is large, hence, the population remains as key determinant for industrialization. Small populations with not too much capital (nor technology) find more attractive the Malthusian sector. In this case, reducing the population with a Black Death shock not only to reverts the process of industrialization but also brings the economy back to a pre-industrial Malthus regime. That is, running down the population, and in turn capital, in an economy that took off recently is able to generate process of de-industrialization or de-urbanization.

The effect that the developmental stage matters for the understanding of demographic shocks is illustrated in figure 15, where the delays in the process of industrialization raise unambiguously with the agricultural share that the economy has when hit by the external demographic disturbance.

Therefore, setting aside the possible discussion on the specification of the AIDS shock as a Black Death shock, one aspect that I think previous studies are missing is the effect that the AIDS shock (even when interpreted as the Black Death) has on the process of industrialization, a dimension of economic development that the Solow model, invariably used in the literature that has studied AIDS, does not capture. [33]

[33] This Black Death experiment I have carried in this section is a numerical exercise without any ambition of calibration. However, one can imagine that calibrating this economy to continental Europe may, partly, help us to understand delays in the process of industrialization of continental Europe with respect to England since the bubonic episodes in continental Europe are known to have preceded and have been more virulent than those in England. Perhaps, it may also be interesting to study the consequences of the Spanish flue that has the sudden aspect of the Black Death (the Spanish flue killed 25 million people in 25 weeks, 1918-1919) while it has the age structure aspect of the AIDS epidemic (the Spanish flue affected mostly young adults, not children or individuals in old age).

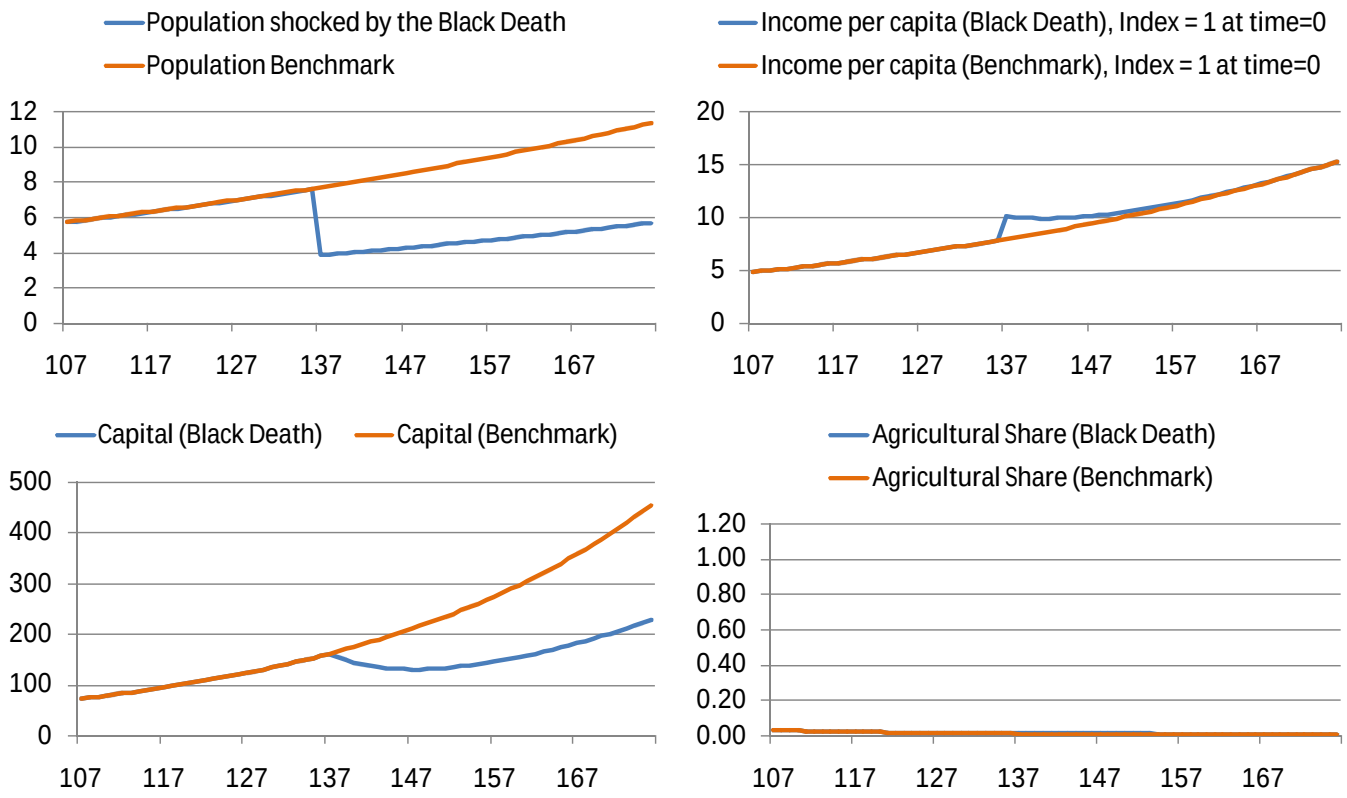

Figure 13: The Black Death at Late Stages of Development

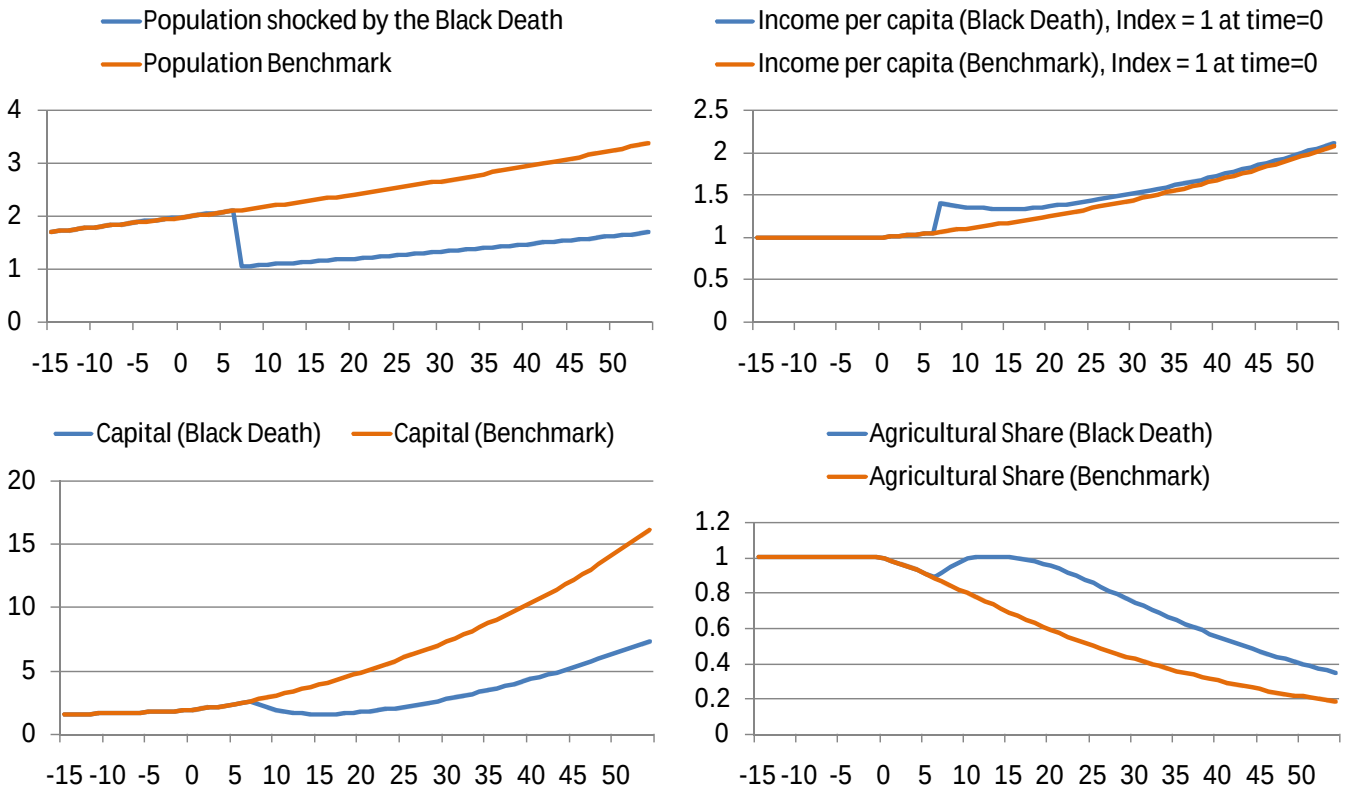

Figure 14: The Black Death at Early Stages of Development

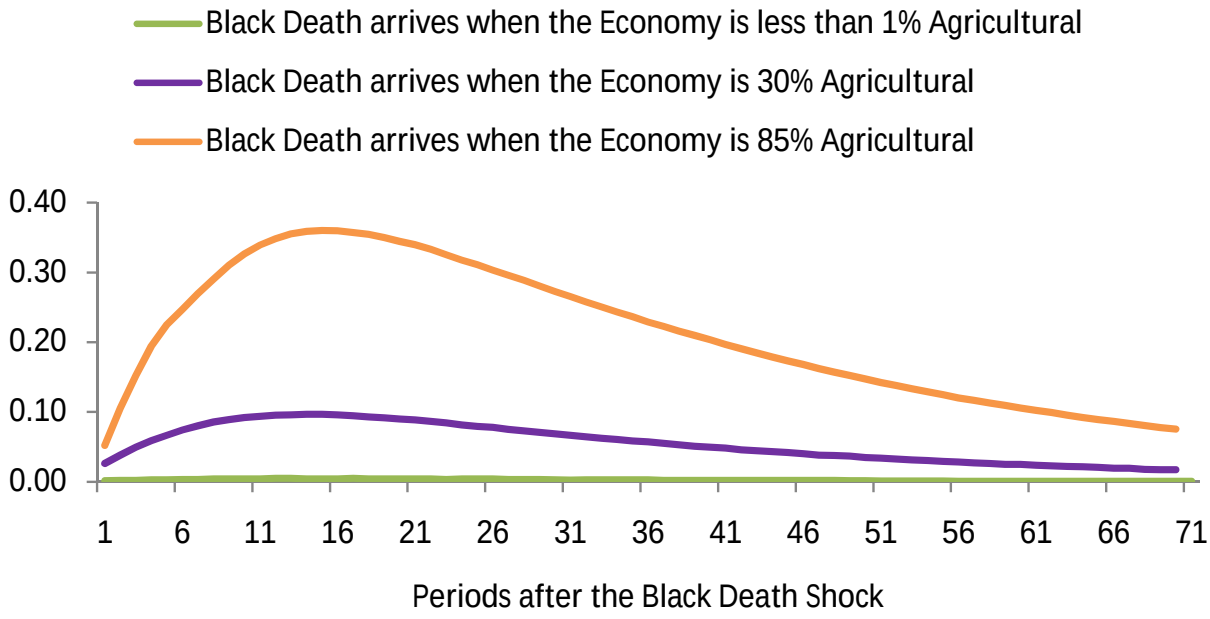

Figure 15: Industrialization Delay: The Black Death Agricultural Share minus Benchmark Agricultural Share

9 Conclusion

In this paper I have investigated the effects the AIDS epidemic has for the process of economic development in terms of income per capita and industrialization. I have incorporated in a standard theory of development an exogenous population process that keeps track of the main effects of the AIDS epidemic on the population growth, age structure and life expectancy over time. I have also computed the aggregate loss in labor efficiency due to AIDS using rural Malawian data (MDICP-06). I have found that when I infect a country unaffected by AIDS with AIDS, that is, when I surprise a healthy country with the population path generated by AIDS, the aggregate effects of AIDS on economic development are much worse than we think. In the context of a model that replicates the early development stage in which the Sub-Sahara is immersed, I have found AIDS (i) delays industrialization for almost a century, and (ii) reduces income per capita by 12% when the prevalence rate is at its highest. Moreover, the uneven impact of AIDS on the age composition of the population extends the adverse effects of AIDS on development by 32%.

I conclude that populations reshaped by the AIDS epidemic (reduced populations with more dependents per worker) hold development up. Hence, if we do not succeed in promoting better health in poor countries, and in Sub-Saharan Africa this means primarily getting rid of AIDS, we should not expect much success from other policies that try to fix the problem of persistent poverty.

One important next step seems to be the exploration of the quantitative impact of AIDS-related reproductive health policies proposed to palliate the effect AIDS has on populations (see, for instance, the policies set forth by the U.S. President's Emergency Plan for AIDS Relief). [34] These polices manifest the complex demographic reality of AIDS and are, to a large extend, tailored to the affected population groups: support families that foster or care for AIDS orphans; increase the prevention of mother-to-child HIV transmission (at birth and breastfeeding); reduce infection of adults thorough counselling on the use of condoms, circumcisions or treatment of genital soars; and treat infected adults with antiretroviral drugs that extend their lifetimes. This suggests that if we want to evaluate how much AIDS-related policies accelerate (or not) the process of industrialization or increase (or not) the income per capita of SSA countries, we need richer models of family structure that capture the heterogenous impact AIDS has on individuals and their families. Once a useful set of policies is found, the question of whether the countries affected by AIDS can implement these polices by themselves or not is still open.

[34] `http://www.pepfar.gov`.

References

Acemoglu, D., and S. Johnson (2007): "Disease and Development The Effect of Life Expectancy on Economic Growth," Working Paper.

Aghion, P., and S. Durlauf (eds.) (2005): *Handbook of Economic Growth*Elsevier Science Publications.

Aghion, P., and P. Howitt (1992): "A Model of Growth through Creative Destruction," 60(2), 323–52.

Becker, G., K. Murphy, and R. Tamura (1990): "Human Capital, Fertility, and Economic Growth," *Journal of Political Economy*, 98, 12–37.

Bell, C., S. Devarajan, and H. Gersbach (2003): "The long-run economic costs of AIDS: With an application to South Africa," World Bank Policy Research Wrling Paper n3152.

Blanchard, O. J. (1985): "Debt, Deficits, and Finite Horizons," *Journal of Political Economy*, 93(2), 223–247.

Bloom, D., D. Canning, and J. Sevilla (2002): *The Demographic Dividend: A New Perspective on the Economic Consequences of Population Change*. RAND Corporation.

Bloom, D. E., and A. Mahal (1997): "Does the AIDS epidemic threaten economic growth," *Journal of Econometrics*, 77, 105–127.

Boldrin, M., L. E. Jones, and A. Khan (2007): "Three Equations Generating an Industrial Revolution?," Working Paper.

Bonnel, R. (2000): "HIV/AIDS: Does it increase or decrease growth in Africa?," The World Bank.

Boserup, E. (1981): *Population and Technological Change: A Study of Long-Term Trends*. Chicago: University of Chicago Press.

Clark, S. (2005): "Understanding Perceptions of HIV Risks Among Married Couples in Malawi: Why do Wives Worry More?," Working Paper.

Cohen, D. (2002): "Human Capital and the HIV Epidemic in sub-Saharan Africa," ILO Programme on HIV/AIDS and the World of Work, Geneva.

Cuddington, J. (1993): "Modelling the Macro-Economic Effect of AIDS with an application to Tanzania," *The World Bank Economic Review*, 7(2).

Cuddington, J., T. Hancock, and A. Rogers (1994): "A Dynamic Aggregative Model of the AIDS Epidemic with Possible Policy Interventions," *Journal of Policy Modelling*, 16(5).

Doepke, M. (2004): "Accounting for fertility decline during the transition to growth," *Journal of Economic Growth*, 9, 347–383.

Dorward, A., I. Mwale, and R. Tuseo (2006): "Labor Market and Wage Impacts of HIV/AIDS in Rural Malawi," *Review of Agricultural Economics*, 28(3), 429.

Fernández-Villaverde, J. (2001): "Was Malthus Right? Economic Growth and Population Dynamics," Working Paper.

Fox, M., S. Rosen, W. MacLeod, M. Wasunna, M. Bii, G. Foglia, and J. Simon (2004): "The Impact of HIV/AIDS on Labour Productivity in Kenya," *Tropical Medicine and International Health*, 9(3), 318–325,.

Gallup, J. L., and J. D. Sachs (2001): "The Economic Burden of Malaria," *American Journal of Tropical Medicine and Hygiene*, 64(1), 85–96.

Galor, O., and D. Weil (2000): "Population, Technology, and Growth: From Malthusian Stagnation to the Demographic Transition and Beyond," *American Economic Review*, 90, 806–828.

Gollin, D., S. L. Parente, and R. Rogerson (2002): "The Role of Agriculture in Development," *American Economic Review*, 92(2), 160–164.

——— (2004): "Farm Work, Home Work, and International Productivity Differences," *Review of Economic Dynamics*, 7(4), 827–850.

Greener, R. (2004): *The macroeconomics of HIV/AIDS*chap. 5 The Impact of HIV/AIDS on Poverty and Inequality. International Monetary Fund.

Greenwood, J., and A. Seshadri (2002): "The U.S. Demographic Transition," *American Economic Review*, 92, 153–159, AEA Papers and Proceedings.

Haacker, M. (2004): "The macroeconomics of HIV/AIDS," International Monetary Fond.

Hansen, G. D. (1993): "The Cyclical and Secular Behaviour of the Labor Input: Comparing Efficiency Units and Hours Worked," 8(1), 71–80.

Hansen, G. D., and E. C. Prescott (2002): "Malthus to Solow," *American Economic Review*, 92(4), 1205–1217.

Hunter, S., R. Isingo, J. Boerma, M. Urassa, G. Mwaluko, and B. Zaba (2003): "The Association Between HIV and Fertility in a Cohort Study in Rural Tanzania," *Journal of Biosocial Science*, 35(2), 189–199.

Jamison, D. T., R. G. Feachem, M. W. Makgoba, E. R. Bos, F. K. Baingana, K. J. Hofman, and K. O. Rogo (eds.) (2006): *Disease and Mortality in Sub-Saharan Africa*. The International Bank for Reconstruction and Development/The World Bank.

Jayachandran, S., and A. Lleras-Muney (2007): "Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines," Working Paper.

Jones, C. I. (2001): "Was an Industrial Revolution Inevitable? Economic Growth Over the Very Long Run," *Advances in Macroeconomics*, 1(2).

Kalemli-Ozcan, S. (2002): "Does the Mortality Decline Promote Economic Growth," *Journal of Economic Growth*, 7(4), 311–349.

——— (2006): "AIDS, Reversal of the Demographic Transition and Economic Development: Evidence from Africa," NBER Working Papers 12181.

Kambou, G., S. Devarajan, and M. Over (1992): "The Economic Impact of AIDS in an African Country: Simulations with a General Equilibrium Model of Cameroon," *Journal of African Economies*, 1(1).

Kelley, A. C. (1988): "Economic Consequences of Population Change in the Thirld World," *Journal of Economic Literature*, 27, 1685–1728.

Keyfitz, N., and H. Caswell (eds.) (2005): *Applied Mathematical Demography*. Springer.

Kremer, M. (1993): "Population Growth and Technological Change: One Million B.C. to 1990," *Quarterly Journal of Economics*, 108, 681–716.

Kuznets, S. (1960): *Demographic and Economic Change in Developed Countries*chap. Population Change and Aggregate Output. Princeton University Press.

Kuznets, S. (1965): *Economic Growth and Structure*. New York.

Kydland, F. E., and E. C. Prescott (1993): "Cyclical Movements of the Labor Input and Its Implicit Real Price," *Federal Reserve Bank of Cleveland Economic Review*, 29, 12–23.

Lee, R. D. (1983): ""Economic Consequences of Population Size, Structure and Growth," *Newsletter of the IUSSP*, 17, 43–59.

——— (2003): "The Demographic Transition: Three Centuries of Fundamental Change," *Journal of Economic Perspectives*, 17(4), 167–190.

Lewis, J., C. Ronsmans, A. Ezeh, and S. Gregson (2004): "The Population Impact of HIV on Fertility in sub-Saharan Africa," *AIDS*, 18(2), 35–43.

Livi-Bacci, M. (1992): *A Concise History of World Population*. Blackwell.

——— (2001): *Seismic Shifts: The Economic Impact of Demographic Change*chap. 3 Demographic Shocks: The View from History. Conference Series 46, Federal Reserve Bank of Boston.

Lorentzen, P., J. McMillan, and R. Wacziarg (2005): "Death and Development," NBER Working Paper Number 1620.

Lucas, R. E. (2002): *Lectures on Economic Growth*chap. 5 The industrial revolution: Past and Future. Harvard University Press.

Mathers, C., and D. Loncar (2006): "Projections of Global Mortality and Burden of Disease from 2002 to 2030," *PLos Medicine*, 3, 11:e442.

Ngai, L. R. (2004): "Barriers and the Transition to Modern Growth," *Journal of Monetary Economics*, 51(7), 1353–1383.

North, D. C. (1981): *Structure and Change in Economic History*. New York: Norton.

Over, M. (1992): "The macroeconomic impact of AIDS in Sub-Saharan Africa," The World Bank.

Papageorgiou, C., and P. Stoytcheva (2007): "What Do We Know About the Impact of AIDS on Cross-Country Income So Far?," IMF Working Paper.

Parente, S. L., and E. C. Prescott (2005): "A Unified Theory of the Evolution of International Income Levels," in *Handbook of Economic Growth*, ed. by P. Aghion, and S. Durlauf, vol. 1, chap. 21, pp. 1371–1416. Elsevier.

Ríos-Rull, J.-V. (2001): "Population Changes and Capital Accumulation: The Aging of the Baby Boom," *Advances in Macroeconomics, The B.E. Journals in Macroeconomics*, 1(7).

Simon, J. (1977): *The Economics of Population Growth*. Princeton: Princeton University Press.

Soares, R. R. (2005): "Mortality Reductions, Educational Attainment and Fertility Choice," *The American Economic Review*, 95(3), 580–601.

Stanecki, K. A. (2004): "The AIDS Pandemic in the 21st Century," U.S. Census Bureau, International Population Reports, WP/02-2.

Thirumurthy, H., J. Zivin, and M. Goldstein (2005): "The Economic Impact of AIDS Treatment: Labor Supply in Western Kenya," NBER Working Paper Number 11871.

UNAIDS (2006): *Report on the Global AIDS Epidemics 2006*.

——— (2007): *2007 AIDS Epidemic Update*.

United-Nations (2006): *World Population Prospects: The 2006 Revision Population Database*.

Werker, E., A. Ahuja, and B. Wendell (2006): "Male circumcision and AIDS: The macroeconomic impact of health crisis," Working Paper, Harvard School of Business.

Williamson, J. G. (2001): *Seismic Shifts: The Economic Impact of Demographic Change*chap. 8 Demographic Shocks and Global Factor Flows. Conference Series 46, Federal Reserve Bank of Boston.

Young, A. (2005): "The gift of the dying: The tragedy of AIDS and the welfare of future African Generations," *Quarterly Journal of Economics*, 120(2), 423–466.

Zaba, B., and S. Gregson (1998): "Measuring the Impact of HIV on Fertility in Africa," *AIDS*, 12(1), 41–50.

Zaba, B., A. Whiteside, and J. Boerma (2004): "Demographic and socieoecomoic impact of AIDS: taking stock of the empirical evidence," *AIDS*, 18(2), 1–7.

A A Brief Timeline of HIV/AIDS

While there are different views on the origins of HIV/AIDS (recent evidence traces the first known case of AIDS to a man who died in the Democratic Republic of Congo in 1959), there exists a consensus in that the first significant number of cases of HIV were identified in June 1981 in U.S.A. among gay men. Within few months, the Center for Disease Control (CDC) stated that the identified immune system disorder was caused by an infection, and AIDS was defined for the first time in 1982. That year, the CDC identified the groups at risk were gay men, injecting drug users, people of Haitian origin and haemophiliacs. By the end of 1982, AIDS was detected on five continents, and, in 1984, cases of AIDS passed on by heterosexual intercourse are identified in Africa. HIV was identified as the cause of AIDS in 1983 by Luc Montagnier of the Pasteur Institute in Paris and later by Robert Gallo of the National Cancer Institute in Washington.

B Data: MDICP, UN and WHO

B.1 Malawi Diffusion and Ideational Change Project

I use intensively the Malawi Diffusion and Ideational Change Project (MDCIP) panel data conducted by the Population Studies Center at UPENN available at `http://www.malawi.pop.upenn.edu`. This survey is ideal for my purposes because it tracks the same individual and household over time, a feature that is essential for the estimation of the transition matrices that represent how an individual evolves over different demographic states.

The most relevant elements of the MDICP for my purposes are: household rosters, including basic demographic information on all members usually residing in the sample households; marriage and partnership histories; cash income, labor market participation and small business activity of the households.

B.1.1 Sampling Strategy, Selection and Attrition

From `http://www.malawi.pop.upenn.edu`: "The sampling strategy was not designed to be representative of the national population of rural Malawi, although the sample characteristics closely match the characteristics of the rural population of the Malawi Demographic and Health Survey. The target sample for the first MDICP wave was 500 ever married women age 15-49 in each district, plus their husbands. The third survey wave added a sample of approximately 400 adolescents age 15-29 in each district."

B.2 Population Projections from United Nations and the World Health Organization

B.2.1 Sources and Method of Projection from United Nations

The future population of each country is projected starting with an estimated population for 1 July 2005. The 2005 estimate is derived from the most recent population data available for each country, obtained usually from a population census or a population register. In cases where recent data on the components of population growth are not available, estimated demographic trends are projections based on the most recent available data. To project the population until 2050, the United Nations Population Division uses assumptions regarding future trends in fertility, mortality and international migration. Because future trends cannot be known with certainty, a number of projection variants are produced.

The 2006 Revision includes eight projection variants and three AIDS scenarios. The eight variants are: low; medium; high; constant-fertility; instant-replacement-fertility; constant-mortality; no change (constant-fertility and constant-mortality); and zero-migration. The three AIDS scenarios named No-AIDS, high-AIDS and AIDS-vaccine. These scenarios are variations of the medium variant and differ from each other and from the medium variant in terms of the path mortality follows because they each incorporate different assumptions regarding the course of the HIV/AIDS epidemic. See United-Nations (2006).

Importantly, that AIDS scenarios considered by the United Nations World Population Prospects incorporate the effects of AIDS on mortality, but not on fertility.

B.2.2 Mortality rates

For Egypt:

1. Time series of age-specific number of deaths and population are taken from World Health Organization Mortality Database and the years for which the data are available are: 1955-1967, 1970 - 1980, 1987, 1991-92, 2000. And time series of total population and population by age-groups, Infant mortality rates and number of births (all in 5-year increment) are taken from United Nations Population Database.
2. Number of deaths and population are then calculated for 3 age groups, namely, children, young and old generations, where the statistics are computed by summing observations with ages below 15, between 15 and 49, and above 50 respectively.
3. Intermediate values of 5-year interval series from UN are estimated by linear interpolation to obtain the full series of 1950 - 2050.
4. Ratio of total population to population by age group and the ratio of number of infant deaths to total population are calculated.
5. The ratio between number of deaths and total population are computed for each age group for available data point. By observation, series for young and old generations are roughly constant over time, so missing values for these 2 series are filled in by assuming values of last available year in the data set. Data are then projected annually to 2050 by assuming the ratio of number of deaths to total population stay constant from 2000 till 2050. While for series of age¡15, missing values before year 2000 are filled in by linear interpolation. The projection of 2001-2050 is taken from the ratio of number of infant deaths to total population (as calculated from (4)).
6. Time series of mortality rates of each age group are then computed by multiplication of the ratio between numbers of deaths (of each age group) to total population (from (5)) with the ratio of total population to population per age group (from (4)).

B.2.3 Fertility rates

Fertility rates are conventionally defined with respect to the female population. To make the model consistent with the data I define the fertility rates ϕ_t^i of age group i as the fraction of births in age group i to the total population in age group i. Time series of births from 1950 - 2050 are obtained from the United Nations World Prospects Database for every five years. Intermediate values are obtained by linear interpolation.

C Ageing Rates and Life Expectancy

C.1 Calibration of the Ageing Rates

To obtain the series of ageing rates, π_t^c, π_t^y and π_t^o, solve for the the system of $3 \times T$ equations and three unknowns $3 \times T$ in (6)-(7), explicitly,

$$N_{t+1}^c = \gamma_t^c \left(1-\pi_t^c\right) N_t^c + \phi_t^y \gamma_t^c \left(1-\pi_t^c\right) N_t^y + \phi_t^o \gamma_t^c \left(1-\pi_t^c\right) N_t^o \quad (13)$$

$$N_{t+1}^y = \gamma_t^c \pi_t^c N_t^c + \phi_t^y \gamma_t^c \pi_t^c N_t^y + \gamma_t^y \left(1-\pi_t^y\right) N_t^y + \phi_t^o \gamma_t^c \pi_t^c N_t^o \quad (14)$$

$$N_{t+1}^o = \gamma_t^y \pi_t^y N_t^y + \gamma_t^o \left(1-\pi_t^o\right) N_t^o \quad (15)$$

Given time series data for fertility rates, ϕ_t^i, mortality rates, γ_t^i, and the population by age groups, N_t^i, I can solve for π_t^c in (13). Then, I use π_t^c to solve for π_t^y in in (14). Then, I use π_t^c and π_t^y to solve for π_t^o in in (15).

The amount of children in $t+1$, N_{t+1}^c, is given by the amount of children in t, N_t^c, that survived and did not age, $\gamma_t^c \left(1-\pi_t^c\right) N_t^c$, plus the number of births (that survived and did not age) born by young adults, $\phi_t^y \gamma_t^c \left(1-\pi_t^c\right) N_t^y$, plus the number of births (that survived and did not age) born by old adults, $\phi_t^o \gamma_t^c \left(1-\pi_t^c\right) N_t^o$.

The amount of young adults in $t+1$, N_{t+1}^y, is given by the amount of children in t, N_t^c, that survived and aged, $\gamma_t^c \pi_t^c N_t^c$, plus the number of births (that survived and aged) born by young adults, $\phi_t^y \gamma_t^c \pi_t^c N_t^y$, plus the number of young adults that survived and did not age $\gamma_t^y \left(1-\pi_t^y\right) N_t^y$, plus the number of births (that survived and aged) born by old adults, $\phi_t^o \gamma_t^c \pi_t^c N_t^o$.

The amount of old adults in $t+1$, N_{t+1}^o, is given by the number of young adults that survived and aged $\gamma_t^y \pi_t^y N_t^y$, plus the number of old adults that survived and did not age, $\gamma_t^o \left(1-\pi_t^o\right) N_t^o$.

Note that I do not allow individuals to shrink, or to grow two age groups in a single time interval.

C.2 Formula of the Life Expectancy

The fundamental matrix of $\Pi_t \times \Gamma_t^M$ is,

$$N_t = I + \left[\Pi_t \times \Gamma_t^M\right] + \left[\Pi_t \times \Gamma_t^M\right]^2 + \left[\Pi_t \times \Gamma_t^M\right]^3 + \cdots = \left(I - \left[\Pi_t \times \Gamma_t^M\right]\right)^{-1}$$

Then, the life expectancy, LE_t, is computed as,

$$\begin{aligned} LE_t &= \sum_j N_t(j,1) \\ &= \frac{1-\gamma_t^o-\gamma_t^y+\gamma_t^o\gamma_t^y-\gamma_t^o\gamma_t^y\pi_t^o+\gamma_t^y\pi_t^y-\gamma_t^o\gamma_t^y\pi_t^y+\gamma_t^o\gamma_t^y\pi_t^o\pi_t^y}{\left(1-\gamma_t^o\left(1-\pi_t^o\right)\right)\left(1-\gamma_t^c-\gamma_t^y+\gamma_t^c\gamma_t^y+\gamma_t^c\pi_t^c-\gamma_t^c\gamma_t^y\pi_t^c+\gamma_t^y\pi_t^y-\gamma_t^c\gamma_t^y\pi_t^y+\gamma_t^c\gamma_t^y\pi_t^y\pi_t^c\right)} \end{aligned}$$

D Projection of the HIV Prevalence Rate

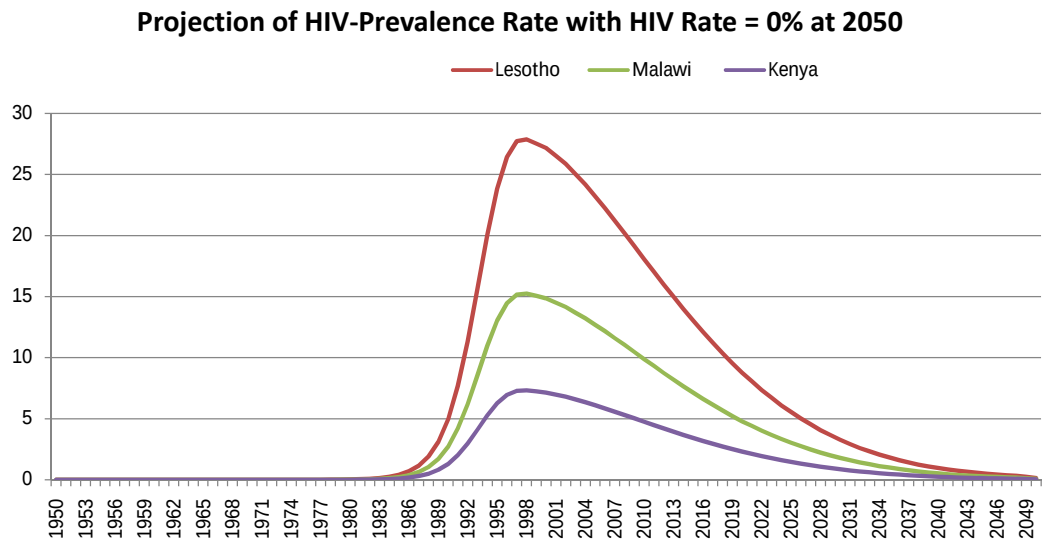

Figure 16: Projection of HIV Prevalence Rate

These projections are computed using the Estimation and Projection Package (EPP, 2007) from surveillance data. This is an epidemiological model that fits available HIV rates and projects them controlling for the rate of growth of the population, the proportion of new risk population entrants, the start year of the epidemic and a behavior change parameter. As a first step, I have used the HIV projection of Botswana that EPP2007 provides and adjusted it to the HIV rate of Lesotho, Malawi and Kenya. I have assumed that the epidemics ends in 2050, what epidemiologists consider a reasonable lower bound. I am also extending the analysis to different scenarios, a more optimistic in which the epidemics vanishes in 2025 and a more pessimistic one in which the epidemics vanishes in 2100.

This alternative thought experiment goes as follows:

1. Remove post-1990 AIDS from populations of countries with mature AIDS epidemics.
2. Construct an AIDS-free population path (1950 onwards) for these countries with post-1990 populations for which we have removed AIDS.
3. Use this AIDS-free population path to calibrate the model economy to these countries before 1990 and project the post-1990 development path (income per capita and agricultural share) of these economies had AIDS not existed.
4. Compare the projected post-1990 development path in the no-AIDS scenario with the data (AIDS scenario).

E Alternative Counterfactual: Removing AIDS from Infected Countries

Note this thought experiment is the reverse to infecting a healthy country as we have done with Egypt in the body of the paper. The main difference between the counterfactual in the paper and this alternative thought experiments strands in the calibration. While for the first counterfactual experiment, infecting Egypt with HIV, we can use data available up to today for our calibration, in the latter counterfactual, we can only use pre-AIDS data to do so. This is why the first counterfactual exercise is preferred.

F Identification of the AIDS shock through Mortality Rates

Here I depict the data I am using to identify the effect of AIDS on mortality. This is the median U.N. projected sex-age-specific probability of dying 2000-2005 by HIV prevalence group for 35 countries in Africa with HIV Prevalence Estimates of 1.0 percent or greater.

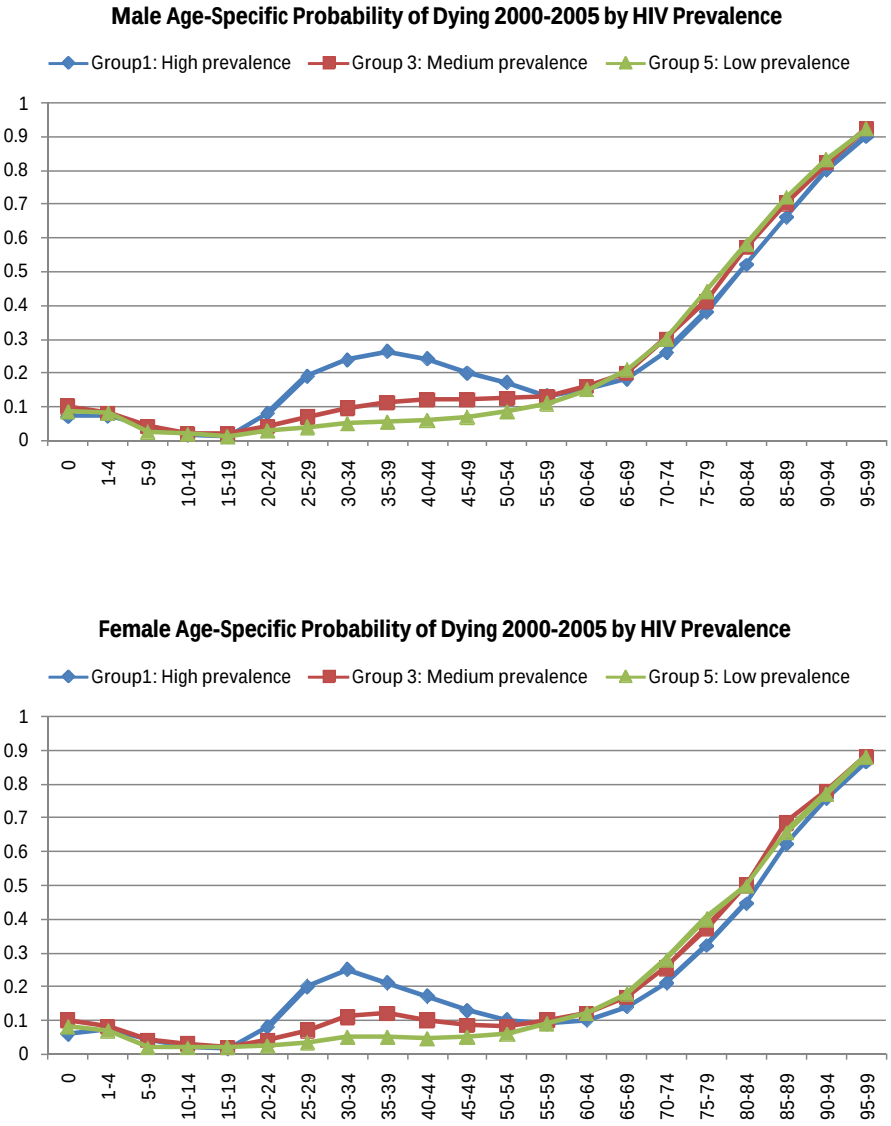

Figure 18: Sex-Age Specific Probability of Dying by HIV Prevalence Group

G Appendix: Some Analytical Derivations of the Development Theory

G.1 Marginal Cost of Production: Solow Technology

$$c_s(w, r_K, Y_s) = \min_{K_s, N_s} \{r_K \; K_s + w \; N_s : Y_s = A_s K_s^{\theta} N_s^{1-\theta}\}$$

Substitute in the objective function the N_s isolated from the technological constraint,

$$c_s(w, r_K, Y_s) = \min_{K_s} \{r_K \; K_s + w \; \left(Y_s A_s^{-1} K_s^{-\theta}\right)^{\frac{1}{1-\theta}}\}$$

FOC (K_s)

$$r_K = w \frac{\theta}{1-\theta} K_s^{\frac{-1}{1-\theta}} Y_s^{\frac{1}{1-\theta}} A_s \frac{-1}{1-\theta}$$

$$K_s = A_s^{-1} \; \left(\frac{w}{1-\theta}\frac{\theta}{r_K}\right)^{1-\theta} \; Y_s$$

Then,

$$N_s = A_s^{-1} \; \left(\frac{w}{1-\theta}\frac{\theta}{r_K}\right)^{-\theta} \; Y_s$$

Then

$$\begin{aligned}
c_s(w, r_K, Y_s) &= r_K \; A_s^{-1} \; \left(\frac{w}{1-\theta}\frac{\theta}{r_K}\right)^{1-\theta} \; Y_s + w \; A_s^{-1} \; \left(\frac{w}{1-\theta}\frac{\theta}{r_K}\right)^{-\theta} \; Y_s \\
&= A_s^{-1} \; w^{1-\theta} \; r_K^{\theta} \left[\left(\frac{\theta}{1-\theta}\right)^{1-\theta} + \left(\frac{\theta}{1-\theta}\right)^{-\theta}\right] \; Y_s \\
&= A_s^{-1} \; w^{1-\theta} \; r_K^{\theta} \left[(1-\theta)^{-(1-\theta)} + (\theta)^{-\theta}\right] \; Y_s \\
&= A_s^{-1} \; \left(\frac{w}{1-\theta}\right)^{1-\theta} \; \left(\frac{r_K}{\theta}\right)^{\theta} Y_s
\end{aligned}$$

G.2 Marginal Cost of Production: Malthus Technology

$$c_m(w, r_K, r_L, Y_m) = \min_{K_m, N_m} \{r_K\ K_m + w\ N_m + r_L\ L_m : Y_m = A_m K_m^{\theta} N_m^{1-\theta}, L_m = 1\}$$

Substitute in N_m isolated from the technological constraint,

$$c_m(w, r_K, r_L, Y_m) = \min_{K_m} \{r_K\ K_m + w\ \left(Y_m A_m^{-1} K_m^{-\phi}\right)^{\frac{1}{\mu}}\} + r_L$$

FOC (K_m)

$$r_K = w \frac{\phi}{\mu} K_m^{\frac{-(\phi+\mu)}{\mu}} Y_m^{\frac{1}{\mu}} A_m \frac{-1}{\mu}$$

$$K_m = A_m^{\frac{-1}{\phi+\mu}}\ \left(\frac{w}{\mu}\frac{\phi}{r_K}\right)^{\frac{\mu}{\phi+\mu}}\ Y_m^{\frac{1}{\phi+\mu}}$$

Then,

$$N_m = A_m^{\frac{-1}{\phi+\mu}}\ \left(\frac{w}{\mu}\frac{\phi}{r_K}\right)^{\frac{-\phi}{\phi+\mu}}\ Y_m^{\frac{1}{\phi+\mu}}$$

Then

$$\begin{aligned} c_m(w, r_K, r_L, Y_m) &= r_K\ A_m^{\frac{-1}{\phi+\mu}}\ \left(\frac{w}{\mu}\frac{\phi}{r_K}\right)^{\frac{\mu}{\phi+\mu}}\ Y_m^{\frac{1}{\phi+\mu}} + w\ A_m^{\frac{-1}{\phi+\mu}}\ \left(\frac{w}{\mu}\frac{\phi}{r_K}\right)^{\frac{-\phi}{\phi+\mu}}\ Y_m^{\frac{1}{\phi+\mu}} + r_L \\ &= A_m^{\frac{-1}{\phi+\mu}}\ \left[\left(\frac{\phi}{\mu}\right)^{\frac{\mu}{\phi+\mu}} + \left(\frac{\phi}{\mu}\right)^{\frac{-\phi}{\phi+\mu}}\right]\ w^{\frac{\mu}{\phi+\mu}}\ r_K^{\frac{\phi}{\phi+\mu}}\ Y_m^{\frac{1}{\phi+\mu}} + r_L \end{aligned}$$

G.3 Allocation Across Sectors

Inputs markets must clear, and wage rates and interest rates must be equated across sectors. This yields the following 4 equations and 4 unknowns K_s, K_m, N_s and N_m:

$$\begin{aligned} K_s + K_m &= K && (16)\\ N_s + N_m &= N && (17)\\ (1-\theta)\, A_s\, K_s^{\theta}\, N_s^{-\theta} &= \mu\, A_m\, K_m^{\phi}\, N_m^{\mu-1} && (18)\\ \theta\, A_s\, K_s^{\theta-1}\, N_s^{1-\theta} &= \phi\, A_m\, K_m^{\phi-1}\, N_m^{\mu} && (19) \end{aligned}$$

Divide (18) by (19),

$$\psi \frac{K_s}{N_s} = \frac{K_m}{N_m}$$

and define $k_m = \frac{K_m}{K}$, $k_s = \frac{K_s}{K}$, $n_m = \frac{N_m}{N}$, and $n_s = \frac{N_s}{N}$. Then

$$\begin{aligned} k_s + k_m &= 1\\ n_s + n_m &= 1\\ \psi \frac{k_s}{n_s} &= \frac{k_m}{n_m}\\ \psi k_s &= \frac{n_s}{n_m}(1-k_s)\\ \psi k_s &= \frac{n_s}{n_m} - \frac{n_s}{n_m} k_s\\ \left(\psi + \frac{n_s}{n_m}\right) k_s &= \frac{n_s}{n_m}\\ k_s &= \frac{n_s}{n_m\left(\psi + \frac{n_s}{n_m}\right)}\\ k_s &= \frac{1-n_m}{n_m\left(\psi + \frac{1-n_m}{n_m}\right)}\\ k_s &= \frac{1-n_m}{n_m \frac{\psi n_m + 1 - n_m}{n_m}}\\ k_s &= \frac{1-n_m}{\psi n_m + 1 - n_m}\\ k_s &= \frac{1-n_m}{1-(1-\psi)\, n_m} \end{aligned}$$

Plug the last one into the wages (18) ,

$$\begin{aligned}
(1-\theta)\, A_s\, K_s^{\theta}\, N_s^{-\theta} &= \mu\, A_m\, K_m^{\phi}\, N_m^{\mu-1} \\
(1-\theta)\, A_s\, k_s^{\theta}\, K^{\theta}\, n_s^{-\theta}\, N^{-\theta} &= \mu\, A_m\, k_m^{\phi}\, K^{\phi}\, n_m^{\mu-1}\, N^{\mu-1} \\
(1-\theta)\, A_s\, k_s^{\theta}\, K^{\theta-\phi}\, n_s^{-\theta}\, N^{1-\mu-\theta} &= \mu\, A_m\, k_m^{\phi}\, n_m^{\mu-1} \\
(1-\theta)\, A_s \left(\frac{1-n_m}{1-(1-\psi)\, n_m}\right)^{\theta} (1-n_m)^{-\theta}\, K^{\theta-\phi}\, N^{1-\mu-\theta} &= \mu\, A_m \left(1-\frac{1-n_m}{1-(1-\psi)\, n_m}\right)^{\phi} n_m^{\mu-1} \\
(1-\theta)\, A_s \left(\frac{1-n_m}{1-(1-\psi)\, n_m}\right)^{\theta} (1-n_m)^{-\theta}\, K^{\theta-\phi}\, N^{1-\mu-\theta} &= \mu\, A_m \left(\frac{1-(1-\psi)\, n_m-1+n_m}{1-(1-\psi)\, n_m}\right)^{\phi} n_m^{\mu-1} \\
(1-\theta)\, A_s \left(\frac{1}{1-(1-\psi)\, n_m}\right)^{\theta} K^{\theta-\phi}\, N^{1-\mu-\theta} &= \mu\, A_m \left(\frac{\psi\, n_m}{1-(1-\psi)\, n_m}\right)^{\phi} n_m^{\mu-1}
\end{aligned}$$

that is,

$$G(n_{mt}; K_t, N_t) = \frac{1-\theta}{\mu}\, \frac{A_{st}}{A_{mt}}\, K_t^{\theta-\phi}\, N_t^{1-\mu-\theta}\, \psi^{-\phi}\, n_{mt}^{1-\mu-\psi} - [1-(1-\psi)\, n_{mt}]^{\theta-\phi} = 0 \tag{20}$$

This is one equation and one unknown n_m, G determines the fraction n_m in terms of K, N, A_s, A_m, and importantly, in terms of the population growth rate and the population level. What

By feasibility we know that n_m is bounded between 0 and 1, $n_m \in [0, 1)$. We also find that for any triple $(\frac{A_{st}}{A_{mt}}, K_t, N_t)$, $G(0; \frac{A_{st}}{A_{mt}}, K_t, N_t) < 0$ and $G(1; \frac{A_{st}}{A_{mt}}, K, N) > 0$:

$$\begin{aligned}
G(0; \frac{A_{st}}{A_{mt}} K_t, N_t) &= -1^{\theta-\phi} < 0 \\
G(1; \frac{A_{st}}{A_{mt}} K_t, N_t) &= \frac{1-\theta}{\mu}\, \frac{A_s}{A_m}\, K^{\theta-\phi}\, N^{1-\mu-\theta}\, \psi^{-\phi} - \psi^{\theta-\phi} > 0 \\
&= 1 - \frac{\mu}{1-\theta}\, \frac{A_m}{A_s}\, K^{-(\theta-\phi)}\, N^{-(1-\mu-\theta)}\, \psi^{\theta} > 0 \\
&= 1 - \left(\frac{\phi}{\theta}\right)^{\theta} \left(\frac{\mu}{1-\theta}\right)^{1-\theta} \frac{A_m}{A_s}\, K^{-(\theta-\phi)}\, N^{-(1-\mu-\theta)} > 0 \\
&= 1 - MC_{Y_s}\, (w_m SS, r_{KMSS}) > 0
\end{aligned}$$

and $G(1; \frac{A_{st}}{A_{mt}} K, N) > 0$ because both sectors are operated. There is a unique n_m that solve $G(n_m; \frac{A_{st}}{A_{mt}} K, N) = 0$.