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# THE GIFT OF THE DYING: THE TRAGEDY OF AIDS AND THE WELFARE OF FUTURE AFRICAN GENERATIONS*

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This paper simulates the impact of the AIDS epidemic on future living standards in South Africa. I emphasize two competing effects. On the one hand, the epidemic is likely to have a detrimental impact on the human capital accumulation of orphaned children. On the other hand, widespread community infection lowers fertility, both directly, through a reduction in the willingness to engage in unprotected sexual activity, and indirectly, by increasing the scarcity of labor and the value of a woman's time. I find that even with the most pessimistic assumptions concerning reductions in educational attainment, the fertility effect dominates. The AIDS epidemic, on net, enhances the future per capita consumption possibilities of the South African economy.

## I. INTRODUCTION

In coming years, the AIDS epidemic will visit upon the peoples of southern Africa a plague of immeasurable horror. The growth of HIV infection among African adults during the 1990s far exceeded most forecasts, with infection rates among pregnant women in, for example, South Africa reaching 25 percent by the year 2000 (Figure I).While adults with HIV typically remain asymptomatic for eight or nine years, they develop AIDS soon thereafter and quickly succumb to the torturous infections and cancers which overwhelm their degraded immune systems. Thus, existing infection rates, in and of themselves, presage the immi-

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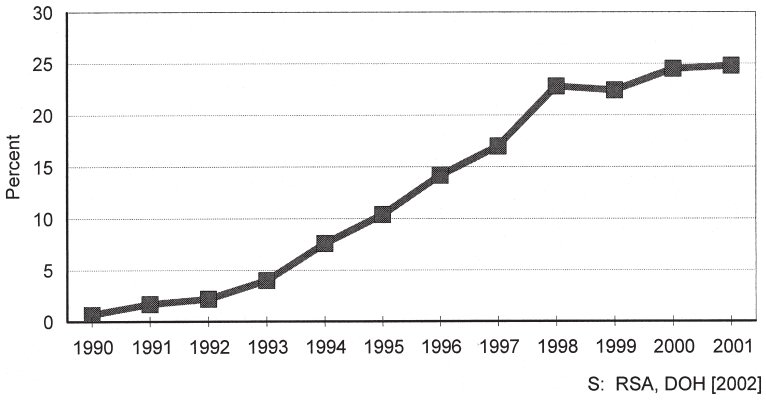



FIGURE I
HIV Prevalence—South Africa (seroprevalence tests of pregnant women)

nent death of at least five million South African adults in the coming decade, without consideration of the millions among future cohorts who will be infected at birth or as young adults.

In considering the economic consequences of the AIDS epidemic, one is drawn to historical examples of similar demographic catastrophes, perhaps the most well researched of which is the Black Death in Britain in the late fourteenth century. Reaching Britain in 1348, and recurring in a series of waves, the Black Death is estimated to have reduced Britain's population to about half of its preplague level by 1377 [Hatcher 1977]. As in the case of AIDS today, contemporary accounts of the human suffering brought about by the plague are depressing in the extreme. Economic data, however, paint a surprisingly different picture. With a declining labor force, real wages rose rapidly during the plague years, and then *remained high* throughout the fifteenth century, as Britain's population stagnated [Steffen 1901; Phelps-Brown and Hopkins 1956]. While early scholars (e.g., Saltmarsh [1941] and Hatcher [1977]) argued that the fifteenth century was a period of continued high mortality, more recent work (e.g., Poos [1991] and Goldberg [1992]) has emphasized the positive role played by the high postplague wages in increasing female labor market participation and lowering fertility, generating a self-reinforcing cycle of high incomes and labor scarcity that lasted for more than a century. Regardless of the precise mechanism, it is clear that the Black Death, in a purely economic sense, was a boon to the generations which survived and succeeded it, who, for

a sustained period of time, experienced living standards which were not seen again until the late nineteenth century.[1]

In this paper I try to calculate the impact of the AIDS epidemic on future living standards in South Africa. Drawing inspiration from the recent historical work on the Black Death, I do so in the context of a Beckerian model which endogenizes participation, fertility, and education decisions with behavioral equations estimated off of household survey data. In simulating the future evolution of the South African economy, I emphasize two competing effects. On the one hand, the epidemic is likely to have a detrimental impact on the accumulation of human capital on the part of orphaned children. On the other hand, widespread community infection lowers fertility, both directly, through a reduction in the willingness to engage in unprotected sexual activity, and indirectly, by increasing the scarcity of labor and the value of women's time. I find that even with extremely pessimistic assumptions concerning reductions in educational attainment, the fertility effect dominates. The AIDS epidemic enhances the future per capita consumption possibilities of the South African economy, in effect endowing it with additional resources. These resources can be used to care for the afflicted and provide higher living standards to future generations.[2]

The paper proceeds as follows: Section II details the model, a simple Beckerian household model embedded in a Solovian constant-savings macroeconomic framework. Section III explains the sources of data, while Section IV estimates the normal household behavioral coefficients and the impact of community HIV infection on fertility. Section V calibrates the macroeconomy and lays out my assumptions concerning the evolution of the epidemic and its impact on household behavior. Section VI then simulates the economy under a variety of scenarios, showing the importance of the fertility and education assumptions and the role Beckerian behavior plays in amplifying and extending the model's dynamics. Section VII presents empirical evidence on recent trends in the South African economy which indicate a dramatic reduction in fertility with minimal reductions in school enrollment, sup-

1. See Steffen's [1901] Tafel II and Phelps-Brown and Hopkin's [1956] Figure 3.

2. Most studies (e.g., Over [1992], Arndt and Lewis [2000], and Bell, Devarajan, and Gersbach [2003]) estimate that AIDS will have a negative impact on future living standards, but do not consider the fertility effects emphasized in this paper. Bloom and Mahal [1995] are an exception to the general pessimism, finding, in a cross-sectional regression, no relation between the incidence of AIDS and the growth of GDP per capita and, incidentally, also noting the parallels between the AIDS epidemic and the Black Death.

porting the most optimistic of my simulations. Section VIII concludes.

## II. Model: Becker and Solow

I model household behavior in the Beckerian tradition, assuming that couples, endowed with human capital $E_m$ (male) and $E_f$ (female) and financial resources $Y$, derive utility from the quantity of children $n$, the "quality" of children $q$ (as measured by their human capital), individual leisure $l_m$ and $l_f$, and material consumption $C_m$. In a one-period framework, each couple maximizes

$$U(n,q,l_m,l_f,C_m), \tag{1}$$

subject to the nonlinear budget constraint

$$l_m W_m + l_f W_f + n t_f W_f + n q P_q + P_c C_m \leq Y + T^*(W_m + W_f), \tag{2}$$

where the $W_i$ are the returns to labor market participation (functions of $E_i$), $P_c$ and $P_q$ the financial costs of consumption and quality, $T$ the individual endowment of time, and $t_f$ the time cost of fertility, which I assume is borne principally by women. I will use household surveys to estimate a simple utility-consistent system of demand and then use it to predict behavior.

The strictures of data severely limit what can be estimated. It is difficult to measure variation across households in the cost of education, so I shall assume, perforce, that the cost of education is proportional to the cost of material consumption ($P_q = a_q P_c$). In third-world household surveys, data on assets are generally unavailable and, in any case, are endogenously determined by individual productivities. Consequently, I ignore income effects. The only remaining plausibly exogenous variation across households then lies in each couple's educational endowments, $E_m$ and $E_f$. In principal, this allows the estimation of a maximum of two price effects in each demand equation. In practice, however, spousal educational attainment is unavailable for many individuals (who are either divorced, widowed, or as yet unmarried) and, even when available, moves quite closely with own education. For children, mothers are identified much more frequently than fathers. These considerations lead me to specify each component of household demand as a function of only one relative price:

$$\begin{aligned} \text{Male Labor Supply} &= LM(W_m/P_c) \\ \text{Female Labor Supply} &= LF(W_f/P_c) \\ \text{Fertility} &= F(W_f/P_c) \\ \text{Children's Education} &= Q(W_f/P_c). \end{aligned} \tag{3}$$

As I will show further below, this simplistic system performs extraordinarily well in predicting out of sample household behavior.

The Hicksian composite commodity theorem states that the expenditure function and underlying preferences can be reexpressed as functions of the aggregates of products with common costs [Deaton and Muellbauer 1980]. Despite the nonlinearity of the budget constraint, this theorem can be applied to the problem above. Thus, the only requirement for consistency with utility maximization is that the matrix of compensated substitution terms for the negative of male labor supply ($-h_m = l_m - T$), the negative of female labor supply ($-h_f = nt_f + l_f - T$), and overall financial expenditures ($C = nqP_q + P_cC_m$),

$$\frac{\partial \mathbf{X}}{\partial \mathbf{P}'} + \frac{\partial \mathbf{X}}{\partial Y}\mathbf{X}', \tag{4}$$

$$\text{where } \mathbf{X}' = (-h_m, -h_f, C) \quad \text{and} \quad \mathbf{P}' = (W_m, W_f, P_c),$$

be a symmetric negative semidefinite matrix.[3] In the context of the demand system (3), this reduces to requiring that $LM'$ and $LF'$ be nonnegative. Given this, one can integrate to derive the money metric indirect utility function,

$$V(Y, w_m, w_f) = Y + \int_0^{w_m} LM(x)dx + \int_0^{w_f} LF(x)dx, \tag{5}$$

$$\text{where } w_i = W_i/P_c,$$

and associated household utility function

$$U(C, h_m, h_f) = C - \int_0^{h_m} w_m(x)dx - \int_0^{h_f} w_f(x)dx, \tag{6}$$

$$\text{where } w_m(\,) = LM(\,)^{-1} \quad \text{and} \quad w_f(\,) = LF(\,)^{-1}.$$

Household utility is simply given by total consumption expendi-

3. This is a straightforward application of Theorem 8 in Epstein [1981].

tures minus the disutility of labor, i.e., the area under the individual labor supply curves.

The preceding one-period model can be extended to a multiperiod lifetime setting by, in the standard macro-theorist's fashion, assuming time-separable utility, so that (3) and (6) represent the period-by-period household demands and flow of utility. Aggregating (6) across all individuals and dividing by the total population, one sees that average utility per capita is given by consumption per capita minus the disutility of labor per capita. If labor supply is of the isoelastic form $h_i = \gamma w_i^{\varepsilon_i}$, per capita utility is then given by

$$u = y\left[(1 - s) - \left(\frac{\varepsilon_m}{1 + \varepsilon_m}\right)\theta_{L_m} - \left(\frac{\varepsilon_f}{1 + \varepsilon_f}\right)\theta_{L_f}\right], \tag{7}$$

where $y$ is output per capita, $s$ the savings rate, and $\theta_i$ the share of each factor in total income. For constant savings rates and factor shares, this justifies the popular fixation on output per capita as a measure of welfare.

To close the model, one must specify the evolution of the macroeconomy and, in particular, the capital stock. Here, any number of frameworks could be used to motivate the savings rate, e.g., overlapping generations with or without actuarially fair market interest rates, intergenerational insurance, or accidental bequests. I heroically sweep these issues aside and, following Solow, assuming that the savings rate, by some combination of private and public sector behavior, remains fixed. This allows me to focus on the mechanisms emphasized in this paper, e.g., fertility and education, and their impact on overall future consumption possibilities. The element left unexamined, the evolution of the savings rate, will determine the ultimate allocation of these consumption possibilities across generations.[4]

4. Thus, one can see the presentation as part of a Ramsey problem in which the planner maximizes the discounted flow of per capita utility by changing national savings and investment through the usual tax and expenditure mechanisms, but with no direct control over household decisions. For appropriate choices of the discount parameter and elasticity of intertemporal substitution, the planner will, in the absence of the epidemic, choose the same initial and steady state savings rate, with some (hopefully minimal) dynamics in between. Taking this as the benchmark, one can then evaluate whether the epidemic, overall, increases or decreases future per capita consumption possibilities. The planner's choice of a savings rate during the epidemic then determines the distribution of these consumption possibilities across generations. Since my emphasis is on the impact of the epidemic, and not the appropriate distributional choice across future generations, I do not pursue this further.

## III. Data and Estimation Strategy

I estimate the model using the microdata files of the South African 1995 October Household Survey (OHS) and the 1998 Demographic and Health Survey (DHS).[5] The OHS provides a variety of personal and behavioral information on each household member (e.g., age, sex, education, labor force participation, income, fertility, etc.), as well as recording recent deaths in the household. Most of the data reported in this survey are consistent with other sources,[6] but the reported retrospective fertility histories are very low (i.e., appear to involve incomplete reporting) and cannot match the historical population distribution. Thus, I use the DHS, whose primary focus is fertility, to estimate that aspect of behavior.[7]

Estimation proceeds in a simple two-step process: I first use the data of the October Household Survey to estimate incomes as a function of age, sex, and education, and then use the predicted relative incomes by educational attainment as the independent variable in the household behavioral equations. Exogenous variation in individual education levels then allows me to identify the price elasticity in each demand equation. I focus only on the behavior of individuals 25 and over, whose education is taken as completed. In the data, however, both fertility and labor supply begin as early as age twelve (albeit, at extremely low levels). The retrospective fertility histories in the Demographic and Health Survey allow me to identify the behavior of fertility at earlier ages, under the assumption that each woman, when young, knew what her final educational attainment would be. For participation, I have only a single cross section, so I simply extend the quadratic age profile estimated off of adults 25 and over.

I estimate both fertility and hours of work using a Poisson

5. The OHS and DHS data sets can be ordered through the web sites www.statssa.gov.za and www.measuredhs.com, respectively. The construction of variables from the raw data is explained in the Appendix at the end of this paper.

6. For example, predicted employee earnings (estimated off of interval income data) are just 12 percent below national accounts compensation of employees, while the reported mortality rates provide a good match to long-term (1970 to 1996) intercensal cohort survival rates.

7. Official South African data on births, based upon voluntary registration, are woefully inaccurate. Births can be registered years after the fact. Thus, 238,000 births were registered in 1991 as occurring in that year, but by the year 2000 the total number of births registered for 1991 had risen to 680,000 [RSA, SSA 2001b]. Yet, the 1996 census found about 960,000 five year olds (microdata files). The primary incentives for birth registration are medical insurance requirements and access to social welfare benefits, and these do not appear to be sufficient to ensure timely or comprehensive coverage. Official mortality data face similar problems.

count model, where the probability of observing a count value of $y_i$ is given by

$$P(Y_i = y_i) = \frac{e^{-\lambda_i}\lambda_i^{y_i}}{y_i!} \quad \text{where } \ln \lambda_i = \mathbf{B}'\mathbf{x}_i. \tag{8}$$

As the expected count is given by $\lambda_i$, with a quadratic in age and the ln relative wage as regressors, this produces isoelastic demand and supply functions. The Poisson, while extremely easy to estimate, has the undesirable feature that the variance of the process is equal to its mean. There are also additional potential error correlations posed by sample design (clustering) or the use of panel data (in the fertility equation). However, as shown by Gourieroux, Monfort, and Trognon [1984], provided that the mean of $Y$ conditional on $x_i$ is equal to $\lambda_i$, maximum likelihood estimates based upon the Poisson distribution are consistent, whatever the process generating the variance of $Y$ may be. Consequently, I use pseudo-maximum likelihood methods, estimating the coefficients using the maximum likelihood Poisson model, but calculating robust standard errors allowing for an arbitrary variance process between observations within survey primary sampling units and across multiple observations for individuals, as well as adjusting for the two-step procedures involved in using parameter estimates from the income equation.[8] This allows me to simplify the presentation of the results. As a sensitivity test, I have also estimated the various equations using constant, linear

8. Let $\mathbf{B_1}$ and $\mathbf{B_2}$ be the vectors of parameters estimated in the first and second step, respectively, across sample sizes $n_1$ and $n_2$. Further, let $N$, the union of the two samples, be divided into $M$ independent clusters $G_1, G_2, \ldots, G_M$, and let $l_{ik}$ denote the log likelihood of observation $k$ in equation $i$. Then, accounting for the two-step procedure and arbitrary correlation within clusters, asymptotically

$$n_1^{1/2}(\hat{\mathbf{B}}_1 - \mathbf{B}_1) \sim N(0, n_1\mathbf{H}_{11}^{-1}\mathbf{g}_{11}\mathbf{H}_{11}^{-1}) \quad \text{and} \quad n_2^{1/2}(\hat{\mathbf{B}}_2 - \mathbf{B}_2) \sim N(0, n_2\mathbf{ABA}'),$$

where

$$\mathbf{H}_{ij} = \sum_{t=1}^{n_i} \frac{\partial^2 l_{it}}{\partial \mathbf{B}_i \partial \mathbf{B}_j'}, \quad \mathbf{g}_{ij} = \sum_{m=1}^{M} \left( \sum_{k \in G_m} \frac{\partial l_{ik}}{\partial \mathbf{B}_i} \right) \left( \sum_{k \in G_m} \frac{\partial l_{ik}}{\partial \mathbf{B}_j'} \right),$$

$$\mathbf{A} = [\mathbf{H}_{22}^{-1} - \mathbf{H}_{22}^{-1}\mathbf{H}_{21}\mathbf{H}_{11}^{-1}] \quad \text{and} \quad \mathbf{B} = \begin{bmatrix} \mathbf{g}_{22} & \mathbf{g}_{21} \\ \mathbf{g}_{12} & \mathbf{g}_{11} \end{bmatrix}.$$

The proof is a simple extension of the standard proof of the asymptotic distribution of the one- or two-step MLE using a first-order Taylor expansion (e.g., Davidson and MacKinnon [1993] and Murphy and Topel [1985]) with the empirical Hessian estimator of the information matrix (which in my sample usually produces substantially larger standard errors than the outer-product-of-the-gradient estimator, which is known to understate standard errors in finite samples [Davidson and MacKinnon 1993, p. 477]).

and generalized variance-mean ratio negative binomial, as well as generalized event count [Winkelmann and Zimmermann 1991], cross-section models, and, where applicable, random effects Poisson and negative binomial panel data models [Hausman, Hall, and Griliches 1984].[9] The results, in almost every case, are very similar, and are reported in footnotes.

I depart from the Poisson model in the determination of children's educational attainment, where I used an ordered probit. Less educated women tend to have more children, who are in turn given relatively less human capital. In this sense, the variance of educational outcomes is quite important, as a mean preserving spread in initial educational attainment will lower average educational levels and welfare in the next generation. Furthermore, bad outcomes tend to be perpetuated from one generation to another. This becomes relevant in assessing the impact of the AIDS epidemic, where I will assume that the education of orphaned children is permanently interrupted at the time of their parents' death. I use an ordered probit model, dividing the population into a state space of fourteen educational categories based upon individual years of education,[10] to keep track of the variance of educational attainment and allow poor outcomes to have disproportionate dynamic effects.

## IV. Estimates

Table I presents the estimation of the Beckerian elements of the model. I begin, in columns (1) and (2), by predicting the ln of before- and after-tax hourly wages as a function of quadratics in

9. With the conditional mean of $Y_i$ given by $E[Y_i|\mathbf{x_i}] = \exp(\mathbf{B}'\mathbf{x_i})$, in the Poisson model the variance-mean ratio is 1. In the constant and linear variance-mean ratio negative binomial models, it is given by $1 + a$ and $1 + aE[Y_i|\mathbf{x_i}]$, respectively. In the generalized event count model it is given by $1 + aE[Y_i|\mathbf{x_i}]^k$, while in the generalized variance-mean negative binomial model it is given by $1 + \exp(\mathbf{B_2'z_i})E[Y_i|\mathbf{x_i}]$ and I use the same set of regressors to determine the variance-mean relationship ($\mathbf{z}_i = \mathbf{x}_i$). The Poisson and constant and linear variance-mean binomial models are special cases of the generalized event count model, and the generalized negative binomial model subsumes the Poisson and linear variance-mean negative binomial as a special case or limiting distribution. The random effects panel data Poisson and negative binomial models have linear and constant variance-mean ratios (respectively), and take into account each panel member's average realized value of the dependent variable. As is usually the case, the various non-Poisson models do better in predicting the distribution of the dependent variable (e.g., the number of zeros). However, as my emphasis will be on the mean, which is fairly insensitive to the choice of distribution, I opt for the simple Poisson formulation, with robust standard errors.

10. As detailed in the Appendix.

TABLE I
ESTIMATION OF THE BECKERIAN ELEMENTS OF THE MODEL

| | (1) Before-tax in wages per hour | (2) After-tax in wages per hour | (3) Male hours of work | (4) Female hours of work | (5) Fertility | (6) Children's completed education |
|---|---|---|---|---|---|---|
| Model | Interval regression | Interval regression | Poisson | Poisson | Poisson | Ordered probit |
| Age | .0820 | .0666 | .1737 | .2125 | .5134 | .0354 |
| | (.0028) | (.0028) | (.0037) | (.0057) | (.0081) | (.0081) |
| $Age^2$ | −.0008 | −.0006 | −.0021 | −.0026 | −.0096 | −.0006 |
| | (.0000) | (.0000) | (.0000) | (.0001) | (.0002) | (.0001) |
| $E$ | .0379 | .0341 | | | | |
| | (.0044) | (.0042) | | | | |
| $E^2$ | .0082 | .0070 | | | | |
| | (.0003) | (.0003) | | | | |
| Sex | −.2190 | −.1768 | | | | |
| | (.0095) | (.0094) | | | | |
| Wage index | | | .1710 | .4399 | −.3538 | 1.1382 |
| | | | (.0078) | (.0134) | (.0140) | (.0378) |
| Year of birth | | | | | −.0031 | .0292 |
| | | | | | (.0013) | (.0016) |
| HIV | | | | | −1.633 | |
| | | | | | (.1896) | |
| Constant | 2.932 | 3.225 | −.0791 | −1.600 | −1.847 | |
| | (.0571) | (.0559) | (.0781) | (.1195) | (2.596) | |
| N | 26289 | 25967 | 27253 | 33238 | 171206 | 10135 |
| Pseudo $R^2$ | .1975 | .1810 | .1620 | .1495 | .0551 | .0549 |

Interval regression—the data are interval coded, so estimation involves maximizing the likelihood that the dependent variable (i.e., the predicted value plus the normally distributed error term) falls within the interval brackets. Wage index = $B_E E_i + B_{E2} E_i^2$, with $B$'s from column (2). $E_i$ equals own education in columns (3)–(5) and mother's education in column (6). Age in column (6) equals mother's age at time of child's birth. HIV is the historical antenatal clinic HIV seroprevalence rate for the woman's quinqennial age group at that time of her life. The sample in column (5) is made up of a panel of 7276 individuals. The construction of the remaining variables and sample selection procedures are explained in the Appendix. All standard errors are adjusted for clustering (on enumeration areas or, for (5), on panel individuals) and the two-step procedure.

age and education and a dummy variable for sex.[11] The return to education in South Africa is quite high, with a 234 percent increase in ln after-tax incomes associated with a movement from 0

11. These estimates do not adjust for selectivity bias (see Killingsworth and Heckman [1986]). I have run more complicated (joint maximum likelihood) systems, augmenting the wage equations with probit selection equations, where the probability of working is determined by the variables in the wage equation plus marital status, children per adult in the household, and the total number of adults in the household (all interacted with sex, i.e., a separate participation equation by sex). First, the point estimate of the correlation between the error terms in the participation and wage equations is negative, which goes against the standard presumption in this literature (this holds even when I allow sex-based covariance

to 16 years of education and a 92 percent premium to tertiary education alone. There is also a great deal of variation in educational attainment within the OHS household sample, with 22 percent of persons over age 25 having two years of education or less and 24 percent having twelve years or more. The extraordinary dispersion in educational outcomes and average earnings by educational group present in South Africa identify the price effects in the demand equations of the model.[12] I use the coefficients on education in column (2) to construct an index of relative lifetime wages, wage index, which is used as an independent variable in the remaining columns of the table.[13]

Columns (3) and (4) of Table I estimate the labor supply response of males and females to a change in the value of their time. As is frequently the case in the labor literature, I find that

between the errors in the participation equations and the joint wage equation, or when I estimate the entire wage/participation model separately by sex). Second, and more importantly, while the age and sex profiles are somewhat flattened, the educational profile of incomes, which is used to identify the price effects in all the subsequent equations, is virtually unchanged. Consequently, to keep things as simple as possible, I ignore selection issues and estimate relative labor incomes using data on workers alone, as reported above. I should note that the data are interval coded (hence the pseudo $R^2$) and that I restrict the sample to employees, i.e., exclude the self-employed.

In estimating separate equations for before- and after-tax hourly incomes, my approach produces an unrealistic linear budget constraint by worker characteristic and, also, implies negative effective tax rates for the least educated young and old age groups. I have run a more realistic model, in which I take into account the nonlinearities in the tax code, work opportunities, and supply decisions (due to fixed costs, fatigue effects, etc.) by modeling participation as a probit zero-one decision subject to a lump sum before- and after-tax income. I calculate after-tax income in two ways, using the tax code and then with a separate regression (mirroring the hourly estimates of Table I). First, I find that the predicted labor supply response to a change in the after-tax wage is virtually the same whether I use the tax code or the after-tax regression; i.e., the after-tax regressions are a close approximation to the actual tax code. Second, the coefficient estimates of the probit model predict that, at current levels, a 1 percent rise in wages will increase the supply of effective male and female labor in the South African sample by .18 and .44 percent, respectively. This is quite close to the Poisson estimates presented in Table I. Since the Poisson constant elasticity functional form allows for easy interpretation of the coefficients and calculation of the disutility of labor, and the linear before- and after-tax budget constraint avoids the complexity of calculating the tax burden at each level of income during the simulations, I opt for the simplified formulation presented above.

12. The reader might be concerned that differences in incomes and economic and social opportunities between racial groups in South Africa bias the model's estimates of behavioral responses to income. In an unpublished appendix, available from the author, I show that while race is a significant determinant of incomes and behavior in South Africa, the inclusion of racial dummies has virtually no impact on the model's estimate of the overall household response to changes in wage income. To keep things as concise as possible, in this paper I focus on the simple model presented above.

13. The before-tax equation is used later in the paper to calculate the effective labor supply, which determines output and wages in general equilibrium.

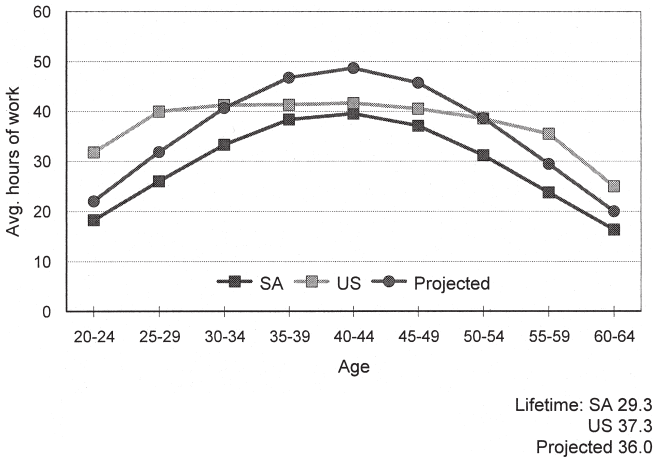



FIGURE II
Male Hours of Work

female labor supply is more elastic than men's. With every 1 percent increase in wages, male labor supply rises .17 percent, and female labor supply rises .44 percent.[14] A better sense of the implications of the coefficient estimates is provided by Figures II and III, where I graph the average hours of work by age group in the prime working years in South Africa and the United States, and then predict the behavior of the United States population using the coefficient estimates of the model.[15] Average male

14. Alternative distributional assumptions yield mostly similar results. The linear and generalized variance-mean ratio negative binomial models produce male/female wage coefficients of .17/.42 and .19/.45, respectively, while the generalized event count model provides estimates of .22/.49. Only the estimates of the constant variance-mean ratio negative binomial model (.44/.77) differ substantially. However, this model is a restricted version of the generalized event count model and is strongly rejected in the data.

15. The SA data are the predicted OHS population values. The U. S. data are the 2000 Consumer Expenditure Survey [BLS 2002] population values. To project U. S. behavior, I first use the microdata of the CES to estimate after-tax wages in the United States with the same functional form as column (2) of Table I. I then predict the behavior of the CES sample using the coefficient estimates of columns (3) and (4) of Table I, applying a purchasing power exchange rate of 1.74, based upon Penn World Table 6.1 [Heston, Summers, and Aten 2002], to convert the year 2000 U. S. dollar wages to 1995 South African Rand. Table I uses the education slope of the wage equation to estimate the supply price response, ignoring the age profile of wages, which is subsumed in the quadratic age term. To predict U. S. behavior, allowing for the slightly different U. S. age profile of incomes, I reinterpret the age-specific constant terms to exclude the age profile of wages, which is then captured in the wage supply response term. My choice of CES income variables, samples, and weights is described in the Appendix.

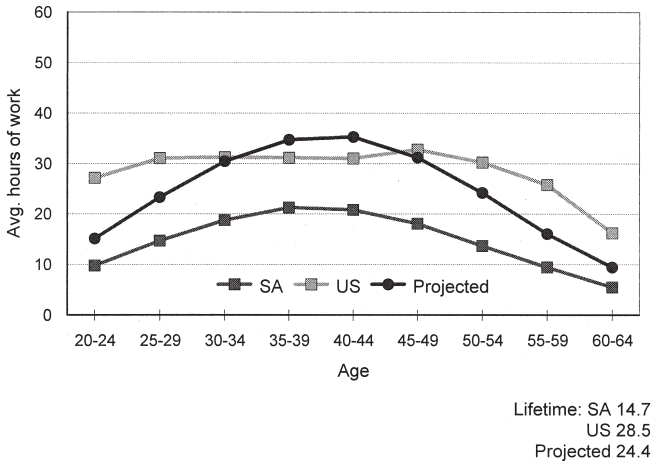



FIGURE III
Female Hours of Work

hours of work between the ages of 20 and 65 are 29.3 in South Africa and 37.3 in the United States. Using U. S. wage values, the model predicts that the United States male population should work an average of 36.0 hours. The model produces a profile of labor supply that is somewhat more hump-shaped than that present in the United States, reflecting patterns seen in South Africa. Part-time employment opportunities may be better rewarded in a more service-oriented economy, and this structural, demand consideration, might explain the higher relative participation of younger and older age groups, who have lower intrinsic labor supply, in the U. S. economy.[16] The female labor supply equation produces a similar pattern, predicting a substantial rise in participation, but not quite attaining the levels or shape seen in the U. S. economy. While not perfect, the model does not produce obviously embarrassing results (e.g., predicting 100 hours of work at U. S. income levels).

Column (5) of Table I uses the retrospective fertility histories of the Demographic and Health Survey to calculate the response

16. Among workers aged 29 or less or 55 or more in the South African economy, 90 percent worked 35 hours or more. The comparable statistic for the United States CES sample is 67 percent.

of fertility to a change in the price of a women's time.[17] I include a time trend to account for trends in wages and the price of education. The time series aspect of the DHS data also allows me to measure the impact of community HIV infection on fertility. As shown in the table, I find that the historical HIV infection rate for each woman's quinqennial age group, as recorded in the maternity clinic seroprevalence surveys cited in the Introduction,[18] has a strong negative effect on predicted fertility.[19] Figure IV graphs year 1995 and 2000 fertility rates in South Africa and the United States, respectively, and then projects U. S. fertility using the coefficients of the model.[20] The model comes close to matching both the life-cycle pattern and the overall level of U. S. fertility.

I estimate the parental demand for quality in the upbringing of their children, in column (6) of Table I, by running an ordered probit of the educational attainment of adults aged 25 or more, whose education is completed, on their year of birth (to capture

17. The reader might worry that there is a bit of a logical inconsistency in using a count model to predict births since, as the time interval gets shorter, more than one birth event becomes biologically impossible. However, for the time frame of Table I, i.e., one year, multiple birth events may occur. The frequency of multiple pregnancies in one birth event (twins, etc.) in the South African DHS by age group is almost identical to that reported in U. S. data (except for women over 40 where, perhaps due to fertility drugs, the U. S. numbers are much higher than those reported in South Africa). However, two-thirds of reported multiple birth events within a twelve-month period in the South African data do not involve multiple pregnancies, but rather represent children born nine to twelve months apart. In a high fertility environment, back-to-back pregnancies are not that rare. I should note that the predicted Poisson probabilities of more than two births in a given year are negligible.

18. Thus, for a woman aged 26 at the time of the 1998 DHS, the HIV variable is the infection rate among 25–29 year olds in the year 1997, when the probit examines her fertility history at age 25, the infection rate among 20–24 year olds in 1996, when the probit examines her fertility at age 24, and so on.

19. The three negative binomial cross-section models all give exactly the same coefficient estimates as column (5) of the table, as each likelihood is maximized at the limiting Poisson distribution. The generalized event count cross-section model yields coefficients of $-.35$, $-.003$, and $-1.59$ on the wage index, year of birth, and the HIV seroprevalence rate, which are virtually identical to the Poisson estimates reported in the table. Similarly, the random effects Poisson and negative binomial panel models both yield coefficients of $-.35$, $-.003$, and $-1.65$. Absent the time trend and seroprevalence rates, the coefficient on wage index is $-.38$.

As the impact of HIV on fertility plays an important role in the subsequent analysis, it is worth reporting some additional sensitivity checks. Since HIV infection rates may be proxying for a nonlinear time trend, I tried including higher order polynomials in time. With a quadratic, the coefficient on HIV falls to $-.71$, but with cubic or quarter time trends it rises to $-2.93$ and $-2.75$, respectively (in each case, remaining highly significant). Thus, a coefficient of $-1.63$ is not, for various formulations of the time trend, an extreme possibility.

20. I fix the time trend at 1995, which is what I use in the later simulations of the South African economy. The actual U. S. data are from CDC [2002], while the S. A. and U. S. projected values are the predicted OHS and CES sample behaviors, as described in the footnote earlier above.

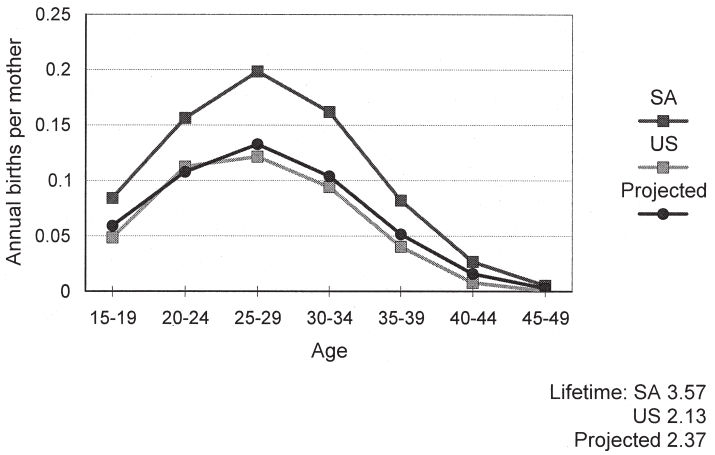



FIGURE IV
Fertility by Age Group

trends in wages and prices) and the wage index and the age at the time of birth of their mothers. Maternal income has a strong effect on realized education. As mother's educational attainment varies from 0 to 16 years, the average educational attainment of a child born in 1970 (aged 25 in 1995) to a 30 year old mother varies from 7.9 to 14.6 years.[21] While persons aged 25 and over living with their mothers constitute a somewhat rarified sample in the developed worked, raising issues of sample selection, this is not a problem in South Africa, where I am able to identify the mother of about one-sixth of all individuals over 25 and one-third of individuals aged 25 to 40. In an unpublished appendix, available upon request, I show that my fertility and education estimates, drawn off of subsamples of the data sets, when applied in a retrospective manner to the OHS female population aged 62 to 99 to predict their surviving children aged 25 to 50, produce an adult population distribution which closely matches the actual size and educational attainment of the 1995 OHS population aged

21. The same equation run on the wage index of fathers yields a coefficient of 1.06, with average children's attainment going from 8.1 to 14.3 years as their father's attainment varies from 0 to 16 years. I opt for mother's attainment as it is available twice as often as father's attainment. I include a quadratic in mother's age at the time of birth for consistency with the other household demand equations, but its practical impact is not large. For a woman with 12 years of education, children's education attainment ranges from a minimum of 12.1 to a maximum of 12.6 years as the mother's age at the time of birth varies.

25 to 50.[22] Projection to U. S. income levels is somewhat less meaningful in this case, given international variation in both public support of education and the human capital interpretation of a year of education. For what it is worth, individuals aged 25 had an average educational attainment of 9.5 years in South Africa in 1995 and 13.3 years in the United States in 2000. The model's prediction for the 2000 25 year old U. S. cohort is 13.4 years.[23]

To summarize, the enormous variation in educational attainment and mean incomes by educational category in South Africa allows me to meaningfully estimate the impact of predictable differences in lifetime income on household decisions. The model matches historical fertility and educational distributions in South Africa and, when projected substantially out of sample, produces patterns of behavior that are not altogether inconsistent with those of the U. S. economy.[24] In the following section I calibrate the macroeconomy to match historical South African data and lay

22. The same Appendix shows that the non-AIDS mortality estimates (briefly described in the Appendix to this paper) are consistent with intercensal mortality rates.

23. This prediction is based upon the one-fifth of the U. S. CES 25-year old sample for which I can identify mother's education. I set the cohort trend at 1970, matching the value for the 25-year old 1995 South African cohort which I use in the simulations which follow, and adjust 2000 U. S. wages to the 1975 birth year using the 1975–2000 .9 percent per annum growth in real, composition adjusted, wages implied by the BLS estimates of the growth of nominal labor compensation minus the growth of effective labor input and the GDP personal consumption deflator (data from http:/www.bls.gov/web/prod3.supp.toc.htm and Council of Economic Advisers [2003]). Without adjustment for the trend in U. S. wages, the predicted value is 13.9 years of education. As noted further below, real wage growth in South Africa during the 1970–1995 period is close to zero.

24. In the interest of science, I should note that this should not be interpreted as evidence that the simple demand system I posit, with no cross-price effects, is an accurate description of reality. Since spousal education varies quite closely with own education, the responses reported in Table I really represent the response to a proportional increase in all of the labor prices faced by the household. I confirm this when I estimate cross-price effects, e.g., in the participation and fertility equations, using spousal education, in the fraction of cases where it is available, and find that the sum of the coefficients on both prices is virtually identical to the own price effects reported in Table I. The problem is that the estimates are not naturally consistent, e.g., partner's education has no effect on male labor supply but has a strong effect on female labor supply. To be consistent with utility maximization, the demand system including cross-price effects would have to be severely restricted, reducing to something close to what I report in Table I.

In the table above I have estimated the response to changes in the overall lifetime labor prices faced by households. Since spousal education moves closely with own education, and since I shall be exploring simulations in which the overall level of wages moves up and down for decades at a time, the coefficients reported in Table I arguably provide a good approximation of likely behavior, even if their interpretation as pure, contemporaneous, own price effects is something of a fiction.

out my assumptions concerning the evolution and behavioral impact of the AIDS epidemic.

## V. Calibration

I calibrate the macroeconomy using standard aggregate data. I initialize the capital stock using the Federal Reserve Bank of South Africa's estimate of the 1995 capital stock and set the depreciation rate at .06, the reported ratio of capital consumption to capital for that year. I then fix the Solovian savings rate at .175, the average real investment to GDP ratio in the period 1946–1995, and set the labor share at .62, the average ratio of compensation of employees[25] to value added during the same period. I assume a Cobb-Douglas production function in capital and effective labor:

$$Y = AK^{\alpha}EL^{1-\alpha}, \quad \text{where} \quad EL = \sum_i W_i L_i. \tag{9}$$

Effective labor is given by the sum of labor supplies differentiated by sex, age, and education, with fixed relative weights given by my estimates, in column (1) of Table I, of before-tax wages in 1995. I assume a closed economy, with the evolution of aggregate factor returns determined by the unit elastic production function and the domestic supply of capital and labor.[26] To focus on the role played by changes in factor supplies, I assume zero future total factor productivity growth.[27]

Turning to the HIV epidemic, the South African maternity clinic seroprevalence data presented at the beginning of this paper suggest that overall infection rates had begun to plateau by the year 2000. Across age groups, infection rates currently peak

25. Adjusted, using census data, for the implicit labor income of the self-employed.

26. In the unpublished appendix I consider the case of perfect capital mobility. This framework excludes most Beckerian effects, as the wage per unit of effective labor is fixed at its initial value. Consequently, the results are similar to the "No Becker" scenario further below where I exclude any endogenous response of household behavior to movements in the wage.

27. Historical estimates of total factor productivity growth are hampered by the poor quality of South African census data (discussed below), whose coverage is inconsistent and where changes in the questionnaire design have induced large changes in measured participation. For what it is worth, real remuneration per worker grows .3 percent per annum between 1970–1995 (http://www.reservebank.co.za), which, after adjustment for rising educational attainment, is suggestive of close to zero growth in effective wages per worker and, by implication, total factor productivity.

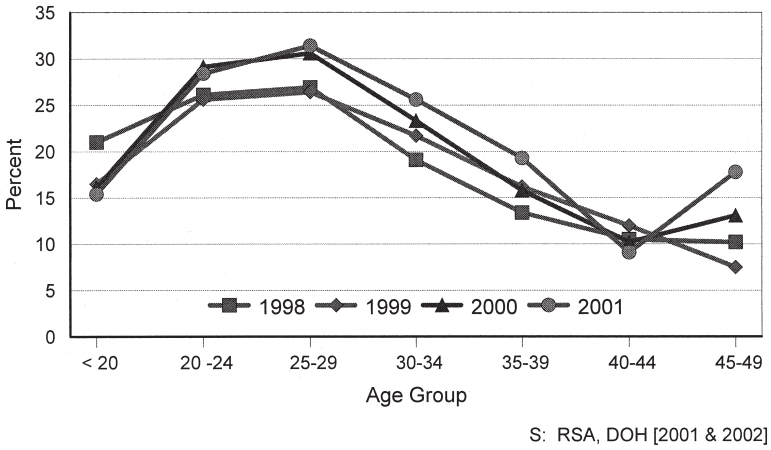



FIGURE V
HIV Prevalence by Age Group

at ages 25–29 (Figure V), and decline thereafter. I interpret the current infection rates of persons over 30 not as random risk, but rather as reflecting the infection of women who, were they 25–29 in 2000, would form part of the current cohort of infected women. It seems reasonable to assume that there is a segment of the population whose interaction with other individuals inevitably, but perhaps unknowingly, leads to infection and that, given the extraordinarily high infection rates already present in the population, the size of this group is given by the maximal infection rate of the 25–29 age group.[28] Consequently, I assume that new cohorts will experience life-cycle infection profiles similar to the upward sloping part of the 2000 age cross section. I assume that, with medical breakthroughs or behavioral change, post-2000 cohort infection rates decline, sinusoidally, to zero over 50 years.[29]

28. In this, I follow the WHO-UNAIDS modeling recommendations [UNAIDS Reference Group 2002]. The UNAIDS Group suggests using aggregate infection rates to estimate the overall "at risk" group and communicability parameters, using an ad hoc procedure to initialize the epidemic. As I need historical age-specific infection rates, I opt for simply using the existing seroprevalence data, with the "at risk" proportion set by the maximal age-specific infection rate in 2000 and the life-cycle pattern of infection given by the upward sloping cross section.

29. Specifically, I begin by smoothing the historical age specific seroprevalence data, running the ln of the infection to noninfection rate (a logit means model) on polynomials in age and year, weighting the observations to account for the heteroskedasticity induced by differing sample sizes. I then use these estimates of cohort age-specific infection rates and UNAIDS estimates of HIV sur-

I also assume that male and female infection rates are evenly matched.[30] Where available, prevalence data indicate fairly equal infection rates across educational groups,[31] which I shall assume as well.

Regarding mortality, adult individuals infected with HIV typically experience a short period of flu-like symptoms, after which they remain, superficially, asymptomatic, until their immune system collapses and succumbs to opportunistic infections and cancers.[32] I use the WHO-UNAIDS [UNAIDS Reference Group 2002] recommendations on adult survival times absent retroviral therapy (Figure VI), which suggest a median survival after infection of nine years.[33] While mother-to-child transmission can be reduced through antiretroviral therapy, cesarean delivery, and avoidance of breastfeeding, these are all costly and currently about one-third of children born of HIV-positive mothers in African countries, and South Africa in particular, are

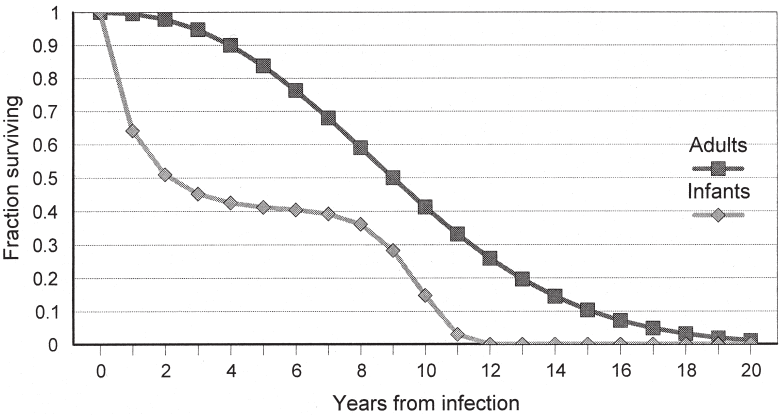
vival rates (discussed below) to calculate the cumulative cohort infection for each of the age groups in the cross section in the year 2000. This produces a cross-sectional function $S(\text{age})$ which happens to peak at age 25. I then assume that the life-cycle cumulative infection pattern for future cohorts will be $S^t(\text{age}) = S(\text{age}) * \sin[(\text{pi}/2) * (2050 - t)/50]$, where $t \geq 2001$ is the year in which the cohort begins sexual activity (assumed to start at age 12) and $S^t(\text{age}) = S^t(25)$ for all ages $> 25$. The last cohort to be infected is that born in 2037, which becomes sexually active in 2049. For cohorts aged 12–24 in 2000, I assume that their life-cycle pattern is given by $S(\text{age})$ up to $S(25)$, while for cohorts over 25 in 2000 I assume that their cumulative infection rate rises by 10 percent of the gap between their current cumulative infection rate and $S(25)$ each year up to age 39.

30. Knowledge of infection rates among men is fairly limited, as the best data on infection rates come from antenatal clinic attendees. However, studies of African couples consistently find that about 30 to 40 percent of male partners of HIV-infected females and 30 to 40 percent of female partners of HIV-infected males are themselves HIV negative [Carpenter, Kamali, Ruberantwari, et al. 1999; Gray, Wawer, Serwadda, et al. 1998; Hira, Nkowane, Kamanga, et al. 1990; Serwadda, Gray, Wawer, et al. 1995]. This suggests relatively equal infection rates once the virus spreads to the general community. The Nelson Mandela/HSRC 2002 Household Survey [HSRC 2002], the only large sample community survey of South Africa, found higher infection rates among women aged 15–24, but no statistically significant difference between males and females aged 25 and above.

31. See HSRC [2002], RSA DOH Eastern Cape [2000] and RSA DOH Free State [n.d.]. Each source shows variation across educational groups, but there is no consistent pattern by educational attainment or across the surveys.

32. http://www.avert.org/hivstages.htm (11/3/2002).

33. The UNAIDS Group estimates that retroviral therapy would add about three years to median survival times. In November 2003 the South African government announced it would be distributing free retroviral drugs, but as of March 2004 only 1500 people had received such support, the government had only begun to ask for bids from pharmaceutical companies, and the health minister was extolling the benefits of consuming olive oil, lemon juice, and garlic [Goering 2004].




S: UNAIDS [2002].

FIGURE VI
Cumulative Survival Rates

infected,[34] a proportion that I assume will remain constant for the foreseeable future. Pediatric AIDS progresses horribly rapidly, with one-third of HIV infected South African children dying from the virus within one year of birth [Bobat, Coovadia, Moodley et al. 1999]. I use the recommended children's mortality profile of the UNAIDS Group (Figure VI), which predicts that all infected children die by age twelve.

Turning to Beckerian behavior, as HIV-infected individuals remain superficially healthy for long periods of time, I assume that adult economic participation and children's education proceeds normally until the final year of each adult's life.[35] To incorporate the impact of orphanhood, I assume that the education of the surviving children of adults who die of AIDS is interrupted at the moment of their parents' (assumed joint) death. Thus, children born of parents with HIV will get minimal education, as their parents generally die before they reach nine years of age,

34. http://www.ama-assn.org/special/hiv/newsline/briefing/mother.htm (11/3/2002), http://www.avert.org/motherchild.htm (11/3/2002), Unicef [2002] and Bobat, Coovadia, Moodley, et al. [1999].

35. Morgan, Malamba, Mayanja, et al. [2000] report a median survival time from the development of AIDS to death in Uganda of 9.3 months. Thus, the epidemiological pattern is one of a prolonged period of apparent health, followed by extremely rapid deterioration and death (see also http://www.avert.org/hivstages.htm, 11/3/2002).

while children whose parents contract HIV after their birth will terminate their education if and when their parents die before they reach 25 years of age.[36] This almost certainly overstates the impact of the HIV epidemic. Empirical estimates of the impact of orphanhood on children's educational outcomes in Africa range from nil [Lloyd and Blanc 1996] to a 5 to 10 percent decline in enrollment rates after the death of each parent [Case, Paxson, and Ableidinger 2004; Evans and Miguel 2004].[37] In contrast, my approach results in dual orphaned children born after the year 2005, whose parents die early in their life, acquiring only one-fifth of the years of education of nonorphaned children.[38] Neverthe-

36. To incorporate this effect, I use the OHS to estimate an additional ordered probit educational equation, representing attainment at ages 6–24 as a function of a quadratic term in age and the mother's wage index (interacted with the quadratic in age). Individuals who reach age 25 without their parents dying from AIDS are endowed with their full human capital, as determined by column (6) in Table I earlier.

37. Lloyd and Blanc [1996], using cross-section data for seven countries, conclude that family networks make up for the loss of a parent, as they find that orphanhood generally leads to a slight decline in enrollment, but produces both positive and negative effects (depending on the country) on completed attainment, with almost all results being statistically insignificant (their Table 8). Evans and Miguel [2004], using panel data for Kenya, report a 2.6 to 3.7 percent decline in school participation postparent death in samples with 85 to 92 percent participation rates, but the total effect rises to about an 8 percent decline when leads and lags on parental death are included (their Figure 1 and Tables 1 and 4). Case, Paxson, and Ableidinger using cross-sectional data for ten African countries, report from 3.8 to 4.9 percent declines with the death of a single parent and between a 12.1 to 15.2 percent decline with the death of both parents in a sample with a mean enrollment rate of 66 percent (their Table 3 and p. 21). Case, Paxson, and Ableidinger show an increased effect with the death of both parents, but Evans and Miguel do not find a negative interaction.

For my part, running a probit of current school attendance in the OHS data on a quadratic in age, district dummies, and the survival status of each parent for individuals 6 to 24, I find that the death of both parents reduces the average annual attendance rate from .77 to .70 (no significant negative interaction). South African data on attendance are not synonymous with achievement, as there is frequent repetition of grades (see Strauss [1999]). As an alternative measure, I run an ordered probit of educational attainment on age, district dummies, and the survival status of each parent for individuals 15–19 and 20–24 in the OHS, finding that the death of both parents reduces educational attainment an average of .6 years (from 8.6 with dual survival) for the 15–19 age group and .9 years (from 9.9 with dual survival) for the 20–24 age group. Both procedures suggest that about 10 percent of cumulated years of education are lost with the death of both parents.

38. Children born prior to 2005 do somewhat better, with, for example, the 1995 orphaned birth cohort getting half of the years of education of nonorphaned children. Early on, as the epidemic is still spreading, the average parent becomes infected at an older age so their children have the opportunity to acquire more human capital.

As noted in an earlier footnote, there is considerable discordance in the HIV status of couples, so that educational losses are likely to be more spread out than my assumption of dual orphaning allows. This is difficult to model, however, as spouses sero-convert over time (Carpenter, Kamali, Ruberantwari, et al. [1999], Hira, Nkowane, and Kamanga, et al. [1990], and Serwadda, Gray, Wawer, et al.

less, I take this extreme assumption as a baseline, a maximal bound on the conceivable losses of human capital due to the epidemic.[39] As an alternative, I also consider the possibility that orphanhood has no impact, whatsoever, on the realized education of children. Reality lies somewhere between these extremes, although the empirical literature on orphanhood and recent trends in South African enrollment (see Section VII) suggest that the path of the epidemic may end up being closer to the no-impact scenario.

With regard to fertility, HIV is well-known to be a sexually transmitted disease and, consequently, community-wide infection should lower the demand for unprotected sexual activity and, by extension, children. This is confirmed in Table I's estimates, earlier, of the impact of age-specific community infection rates on fertility.[40] I use these estimates to modify fertility, with diminishing force as the epidemic wanes and community infection rates decline. The decline of fertility associated with the HIV epidemic plays an important role in the simulations, raising the capital-labor ratio faced by future cohorts and offsetting the reductions in human capital brought about by premature parental death. Although recent South African demographic data support my esti-

[1995] find between 5 and 25 percent sero-conversion among healthy spouses within a year), so that the final degree of dual versus single orphaning is difficult to predict. I opt for assuming that all AIDS-induced adult deaths produce immediate dual orphans.

39. One might argue that, aside from the issue of orphanhood, the epidemic will reduce the demand for human capital as the expected return from educational investments is reduced by the shortened expected lifetimes of the current youth. This view, however, is rejected by Section VII's data on continued school enrollment. In that sense, the data are supportive of this paper's rather narrow view of education, at least at the levels seen in Africa, as being primarily a parental consumption good, without substantial regard for economic return.

40. These estimates, perforce, did not distinguish between behavioral or physiological responses. A growing body of field research (e.g., Gray, Wawer, Serwadda, et al. [1998], Carpenter, Nakiyingi, Ruberantwari, et al. [1997], and Hunter, Isingo, Boerma, et al. [2003]) indicates that HIV may reduce the fertility of women who are otherwise mostly asymptomatic and unaware of their infectious status by about 50 percent (explanations focus on amenorrhea and early term miscarriages). This physiological response, however, is not large enough to explain the estimates of Table I, which suggest that, at a 100 percent population infection rate, fertility would be about 20 percent of normal. One can interpret this difference as representing an external effect, i.e., the impact of community infection on individual behavior. I have explored various simulations in which I parse Table I's estimates into physiological and behavioral effects, and found that the aggregate results are quite similar to the simple specification of a common behavioral response. To keep the analysis simple, I focus on that interpretation in this paper. An unpublished appendix, available from the author, reviews the literature on the physiological impact of HIV on fertility and presents the alternative simulations.

mates of the impact of HIV on fertility (see Section VII), I will also consider simulations in which HIV has no impact on fertility, highlighting the important role played by this variable.

Finally, I should note that in all scenarios I shall assume a constant savings rate. The assumption of a constant savings rate allows me to focus on other factors I believe are critical, such as the change in fertility and the possible loss of educational capital. Further, while the South African investment to GDP ratio has fluctuated, it shows no trend, either in the historical past or during the first decade and a half of the epidemic (see Section VII below). Finally, future movements in the savings rate are best thought of as allocations across generations in the consumption possibilities engendered by the epidemic. I take as my baseline simulation of the positive evolution of the economy with HIV the assumption, consistent with current empirical trends and convenient for exposition, that, whatever the private determinants of savings, government policies will adjust so as to distribute these consumption possibilities intergenerationally on the basis of a constant savings rate. However, putting aside that particular savings path, I will ultimately show that the epidemic allows all future surviving generations to enjoy higher living standards, while still leaving ample resources to support the infirm. This is the central point of the paper.

## VI. Simulation

In the pages that follow I simulate the evolution of the South African economy under a variety of circumstances. I consider five scenarios: (1) "No HIV"—the path taken absent the epidemic; (2) "HIV"—the economy with the HIV epidemic; (3) "No Becker"—the economy with the epidemic, but with no endogenous response to changes in wages, i.e., all education, fertility, and participation decisions by educational class kept at their values along the No HIV path;[41] (4) "No Fertility"—the No-Becker scenario, but with the added dimension that HIV does not have the negative effect on fertility estimated earlier in Section IV; and (5) "Full Education"—the HIV path, but without the assumption that children's education is interrupted at the time of their parents' death. The

41. This simulation still contains Beckerian effects in that changes in the distribution of educational attainment lead to changes in average behavior and the behavior by educational class responds to the value of the real wage along the No HIV path.

TABLE II
SUMMARY OF THE SIMULATIONS

| | |
|---|---|
| No HIV | Household behavior (participation, fertility, and final educational attainment of newborn children) from 1995 on determined by current wages using equations of Table I (with time trends fixed at 1995 values). 1995 population initialized using OHS adult distribution (age $\geq$ 25), with number and ultimate educational attainment of preexisting youth cohorts projected from historical household equations (including pre-1995 time trends).* Capital stock initialized using national accounts data and evolving in accordance with a fixed savings rate. Wages determined in general equilibrium by capital and labor supply. Mortality given by pre-AIDS mortality estimates, discussed in the Appendix. |
| HIV | Same as No HIV, except that fertility is lowered to include the impact of current HIV infection rates on community fertility (as estimated in Table I), while education, including the ultimate attainment of preexisting 1995 youth cohorts, is modified under the assumption that human capital accumulation ceases at the time of parental deaths due to AIDS. HIV infection rates decline to zero over 50 years, as summarized in footnote 29 earlier. HIV survival rates given by Figure VI, with AIDS and non-AIDS mortality assumed to be independent. |
| No Becker | Same as HIV, except that wages present along the No HIV path are used as the input in determining household behavior, i.e., no endogenous response to realized wages. |
| No Fertility | Same as No Becker, except that HIV does not influence community fertility. |
| Full Education | Same as HIV, except that orphans do not suffer loss of educational capital (attainment equals the demand of parents at time of children's birth). |

The number and educational attainment of individuals less than 25 in 1995 has to be projected because the OHS underenumerates youth and because the ultimate educational attainment of preexisting youth cohorts is unknown and needs to be forecasted (including adjustment for their eventual orphaning in the HIV scenarios).

components and assumptions that enter into the simulations are summarized in Table II. Before delving into economic calculations, it is worth recalling the human cost of the epidemic which, as shown in Figure VII, could cumulatively claim 18 millions lives (in the baseline HIV scenario). What follows is a statement about economic conditions and the welfare of survivors, not about the desirability of the wholesale slaughter of innocents.

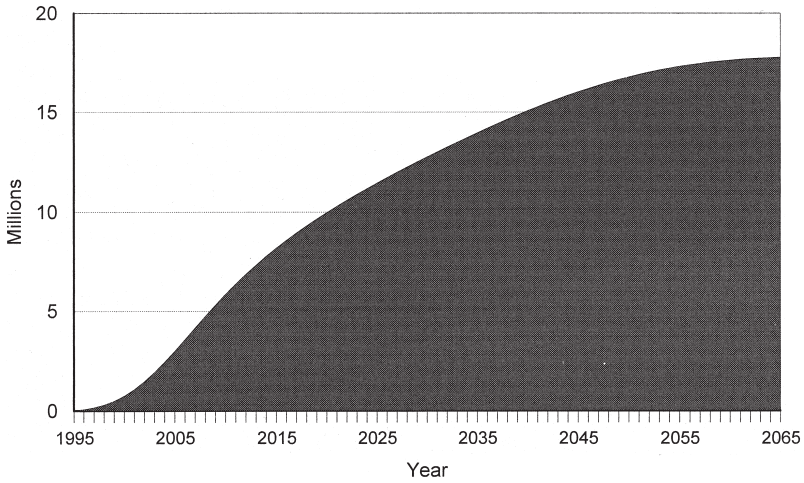



FIGURE VII
Cumulative AIDS Deaths

Figure VIII depicts the evolution of the wage per unit of effective labor. Absent the HIV epidemic, the wage initially declines, as better educated young cohorts put pressure on the ratio of capital to effective workers, but eventually begins to climb, as

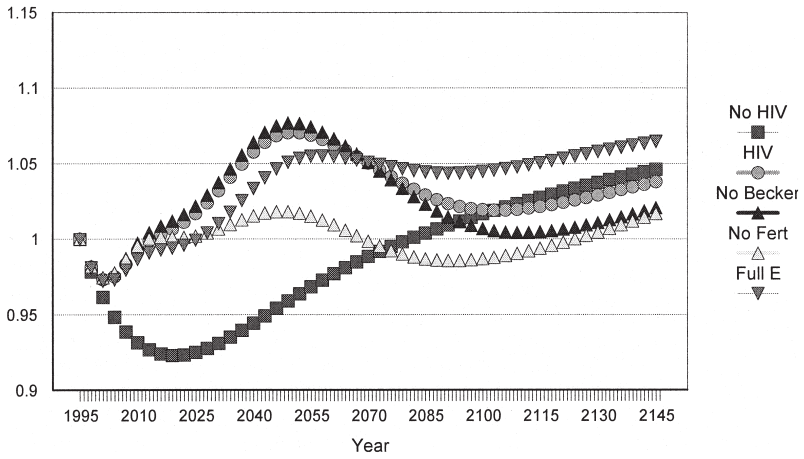



FIGURE VIII
Wage per Unit of Effective Labor (1995 = 1)

higher levels of education lead to lower fertility and population growth. This pattern is reversed by the epidemic, as high mortality in the first decades of the twenty-first century drives up the wage, which then comes back down as the epidemic wanes and the labor force is built back up. In the baseline HIV scenario, the wage rises to 11 percent above the No HIV path by 2040. Although most of the mortality associated with the epidemic is resolved by 2040 (Figure VII), it takes some 60 years for the wage to return to the No HIV transition path, as the reduced fertility brought about by the changed population structure and higher wages sustains a higher capital-labor ratio. In the No Becker scenario, the failure of fertility to respond to temporarily higher wages results in a more rapid decline in the wage, which ultimately falls below the levels achieved along the No HIV path. When, in addition, the HIV epidemic has no impact on fertility, the wage falls well below the No HIV transition path. Finally, in the Full Education scenario, the high wages brought about by the epidemic are completely self-sustaining. High wages lead to lower fertility and better educated children, which in turn sustain higher wages.

The impact of the epidemic on the accumulation of human capital is summarized in Figure IX, which depicts average educational attainment by cohort birth year. In the HIV, No Becker, and No Fertility scenarios, I assume that the orphaning of chil-

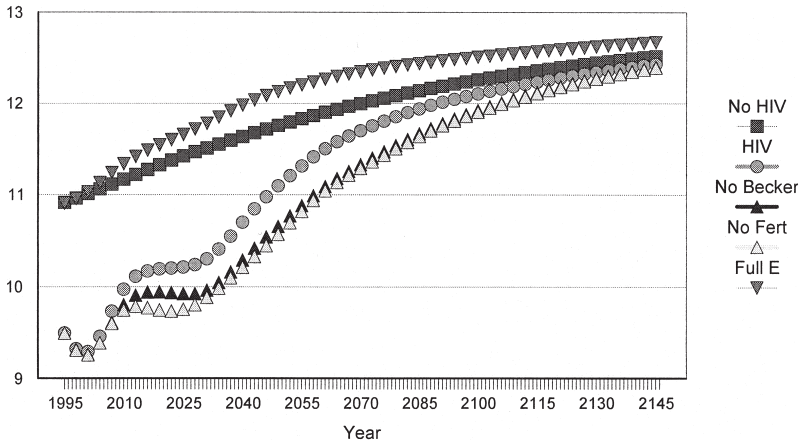



FIGURE IX
Educational Attainment by Birth Year

dren leads to an immediate and permanent interruption of their education. As shown in the figure, this lowers the educational attainment of the 1995 cohort by about one and a half years and substantially reduces the human capital of cohorts for the next 100 years.[42] Uneducated individuals have higher fertility and less educated children, so the losses from orphaning early in the epidemic persist for generations. The positive effect of higher wages on the demand for quality in children and the lower fertility of HIV-infected cohorts work against this effect. Consequently, the reductions in educational attainment are smaller in the HIV scenario than they are in the No Becker or No Fertility scenarios. The loss of human capital explains why the wage along the No Becker and No Fertility paths, shown earlier in Figure VIII, falls below the original, No HIV, transition. Lower educational attainment, for a given real wage per effective worker, leads to higher average community fertility, which drives the real wage down, perpetuating lower levels of education and further high fertility. In contrast to the above, in the Full Education scenario I assume that the death of parents does not interrupt their children's education. In this case, the rise in wage (Figure VIII), leads to higher demand for children's education, which in turn supports continued lower fertility, sustaining both wages and the level of education attainment.

Figure X graphs the GDP per capita realized along the various epidemic paths relative to the levels sustained along the No HIV transition to the steady state.[43] In the baseline HIV simulation, the initial rise in the wage per effective worker more than

42. About one-fourth of all HIV-free (i.e., surviving) children born in 1995 are orphaned before completing their education, with the ratio falling to 10 and 5 percent for the cohorts born in 2020 and 2045, respectively. The education acquired by orphaned children depends upon how long their parents survive. Children born in 1995 who are eventually orphaned get about one-half of the years of education of nonorphaned children, as the epidemic in 1995 was still spreading and much of the adult population was as yet uninfected. Orphaned children born after the year 2005, when the epidemic is established and on-going, get only about one-fifth of the education of nonorphaned children.

43. Absent the epidemic, GDP per capita rises .8 percent per annum for the first century, in the gradual transition to a steady state 3.5 times greater than its value in 1995. As the real wage per effective worker rises only 10 percent in the transition to the steady state, almost all of this growth is driven by the accumulation of labor, and not capital. The transition is driven by increases in educational attainment, as better educated cohorts raise better and better educated offspring. Higher levels of education increase effective labor per capita directly, through productivity, and indirectly, by raising age-specific participation rates and lowering fertility, which in turn shifts the population structure in favor of age groups with higher participation. The steady state involves an average adult educational attainment of 13.0 years, well above the cross-sectional average of 8.5 in 1995.

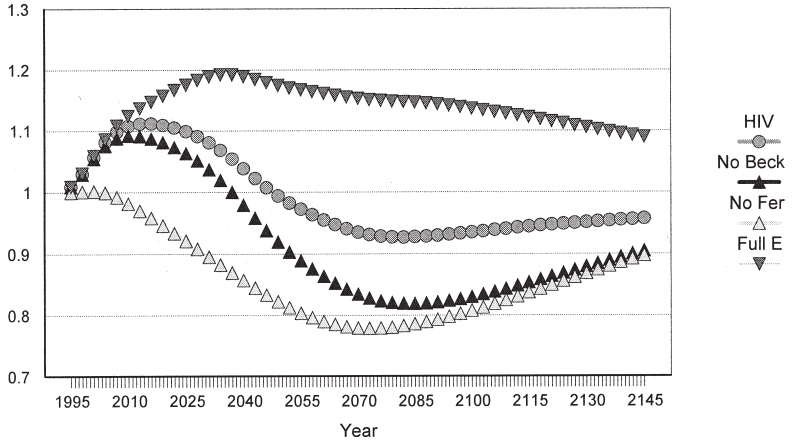



FIGURE X
GDP per Capita (relative to No HIV)

offsets the losses in cohort educational attainment, so that output per capita remains, for some 50 years, above the No HIV path. Eventually, however, the reduced educational attainment becomes dominant, and output per capita falls slightly below the No HIV path. In the No Becker scenario, neither participation, fertility, nor children's education respond to the high temporary wages, so output per capita rises by less during the wage boom and then falls considerably below the No HIV path, as the economy is burdened by large uneducated cohorts. Conditions are worse in the No Fertility case, where, despite the wage boom, rising dependency ratios, brought about by adult mortality combined with continued fertility, drag the economy down from the very beginning. In this case, high fertility in an uneducated population ultimately leads to an output per capita about three-quarters of what would have been experienced absent the epidemic. At the other extreme, in the Full Education scenario, with no adverse effects on children's education, high wages and low fertility lead to higher output per capita everywhere along the path. Although this scenario seems decidedly Panglossian, it is not far removed from recent developments in the South African economy where, as discussed in the next section, the epidemic, at least in its early stages, appears to have been associated with falling fertility and sustained human and physical capital accumulation.

TABLE III
ALTERNATIVE CONSUMPTION POSSIBILITIES

| | Percentage increase in living standards in perpetuity | Retroviral expenditures per AIDS patient (2004 US$) |
|---|---|---|
| Full Education | 65.6 | $11000 |
| HIV | 5.6 | $ 8800 |
| No Becker | 4.3 | $ 7000 |
| No Fertility | −3.0 | NA |

NA = not applicable, as it is not possible to support a retroviral program while maintaining the living standards of the No-HIV path.

Figure X provides some insight into the influence of the HIV epidemic on future living standards under a fixed savings rate. Rather than consider different intergenerational allocations driven by different savings scenarios, one might broadly summarize the impact of the epidemic on future consumption possibilities by answering the following question: allowing the savings rate to vary as necessary, what percentage increase, relative to the No HIV path, in future living standards (taken as per capita consumption minus the disutility of labor supply)[44] is possible under the different scenarios?[45] The answer to this question is given in the first column of Table III. Starting with the Full Education scenario, one sees that a 6.6 percent increase in living standards in perpetuity is possible. The early booms of the baseline HIV and No Becker scenarios are followed in later years by a relative decline in GDP per capita (Figure X), as the reduction in human capital takes its toll. Nevertheless, with an appropriate increase in savings early on these two scenarios allow for 5.6 and 4.3 percent increases in perpetuity, respectively, relative to the No HIV world. Finally, in the case of the No Fert scenario, output per capita begins to decline immediately, and there is no boom that can be invested. In this case, an immediate and permanent

44. As noted in Section II, this is equal to the flow of per capita utility absent the HIV epidemic. One should hesitate to use this term for the epidemic, as it does not take into account disutility from mortality risk and the modification of behavior (e.g., reduction in fertility) brought about by the presence of this risk. While one could use assumptions and modeling structure to calculate "compensations" for these factors, I think it more reasonable to simply talk about living standards. For generations that live after the epidemic, this is equivalent to utility, given the demand functions estimated in this paper.

45. This calculation is done subject to the transversality condition that in the new steady state the capital-labor ratio permanently sustains the new level of consumption.

3 percent reduction in living standards allows the economy to distribute the severe trough of later years evenly across all generations.

While the preceding calculations focus on the welfare of the living, one might also consider the support of the dying. In the second column of Table III, I calculate the maximum annual expenditure on retroviral therapy for AIDS patients possible consistent with providing all living individuals, everywhere along the path, with the alternative future No HIV living standards. In this calculation, I follow UNAIDS [2002] and assume that retroviral drugs will extend the life of terminal AIDS patients by three years; i.e., each patient receives three years of treatment. I assume that these persons do not contribute in any way to economic activity, but count in the total population numbers so that, in addition to retroviral drugs, they receive the average per capita flow of consumption. The number of persons receiving retroviral therapy peaks at around 3.4 percent of the population in 2008 and remains above 1 percent until 2040. As shown in the table, per patient retroviral expenditures close to 10,000 U. S. dollars per year are sustainable, while giving both survivors and the invalided the living standards they would have enjoyed absent the epidemic.[46] To put these numbers in perspective, generic triple dose retroviral therapy drugs are available for US$292 or less per year.[47] Brazil's large-scale retroviral program, with auxiliary CD4, viral load, and drug-resistance testing, is currently delivered at a cost of less than US$1200 per patient year.[48] Clearly, the AIDS epidemic endows the South African economy with enough resources to both care for the victims of the epidemic and

46. I convert rand values to U. S. dollars using an exchange rate of 6.5 rand to the dollar, reflecting the value of the rand in the middle of 2004.

47. McNeil [2004] reports figures of US$292 and US$244 for Indian generic producers and notes that in October 2003 the Clinton foundation negotiated a price of US$140 per patient year for large orders paid in cash.

48. http://www.avert.org/aidsdrugsafrica2.htm (5/16/2004) and Marins, Jamal, Chen, et al. [2003]. Median survival times in Brazil rose from 18 months for AIDS patients diagnosed in 1995 to 58 months for AIDS patients diagnosed in 1996, when the free retroviral program was instituted [Marins, Jamal, Chen, et al. 2003]. The effectiveness of retroviral therapy improves with expenditure on auxiliary testing, treatment, and supervision. Consequently, the UNAIDS assumption of three-year survival might be unduly pessimistic for a sufficiently well-funded program, implying that the maximal expenditures calculated above are overestimates of sustainable expenditures per surviving patient. This, however, only reinforces the point made in the table; i.e., that sufficient resources exist to improve the circumstances of infected individuals without harming future generations.

still have means left over to raise the standard of living of future generations.[49]

## VII. Empirical Developments

Recent empirical trends in the South African economy support the analytic assumptions of the simulations laid out above and their positive view of the welfare of future, postepidemic, generations. Although gross national saving and fixed capital formation were definitely lower in the 1990s than in earlier decades (Figure XI),[50] this cannot be attributed to the epidemic, as the decline occurred in the late 1980s, long before HIV took root in South Africa. During the 1990s and into the early part of the new century, as the epidemic spread and entered the public consciousness, aggregate savings and capital formation have remained remarkably steady.[51] During the same period, despite

49. An unpublished appendix, available from the author, reviews the sensitivity of the results to some of the underlying assumptions. Allowing for perfect capital mobility (removing the closed economy assumption), fixes the wage and eliminates most of the Beckerian effects, producing per capita income results similar to the No Becker scenario. Capital mobility, however, eases intertemporal substitution, allowing the economy to sustain somewhat higher permanent increases in living standards (or smaller declines in the No Fert scenario). Reconsidering Table I's estimates of the reduction in fertility brought about by HIV as being a combination of behavioral and physiological (in infected women) influences produces fairly similar results to those shown above. Although this view is completely rejected by the HSRC 2002 community survey, I also consider the possibility that underlying community infection rates are almost double those indicated by the maternity clinic seroprevalence data. In this case, the results are worse, producing more orphans and considerably greater numbers of prospective patients, but the economy can still sustain retroviral expenditures of US$3100 per AIDS victim per year.

50. The data in the figure are based upon national accounts aggregates, available from http://www.reservebank.co.za, with public sector saving defined as general government saving plus fixed capital consumption and "private sector" saving defined as household and corporate saving (including public corporations), plus the fixed capital consumption of private business enterprises and public corporations. Together, these equal gross national saving or, equivalently, gross capital formation plus the current account. The savings data are in current prices, and gross fixed capital formation is in constant 1995 prices.

51. As the reader can see, public sector savings during this period rose (from negative to positive), while "private sector" savings (including public corporations) fell. From the point of view of understanding savings behavior, it is interesting to argue whether private savings fell in response to the epidemic or in response to the increase in public savings, or whether public savings rose in response to the decline in private savings. However, from the point of view of the implications for future generations, it does not matter what combination of public or private savings behavior produces a constant national savings rate. It is sufficient that it is simply constant. Hence, the Solovian assumption made in this paper.

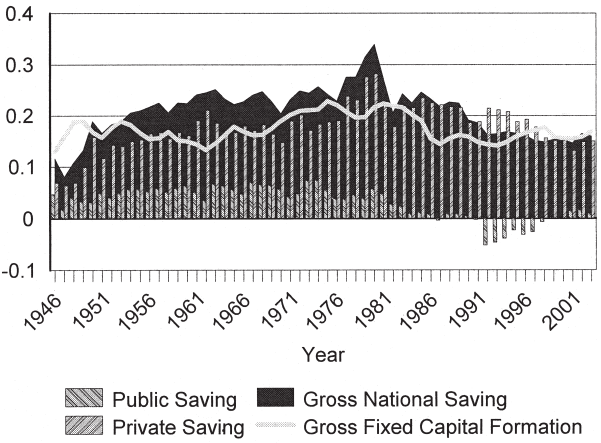



FIGURE XI
Saving and Investment/GDP

rising parental mortality,[52] school enrollment rates for the youngest age groups have not fallen, while those of older teenagers have declined by only 5 percent (Table IV), indicating that educational losses from orphaning are minimal.[53] Up to this point, the epidemic has had little detrimental effect on physical or human capital accumulation.

The impact of HIV on fertility, a key element in the analysis above, is confirmed in census data. Figure XII graphs the youth

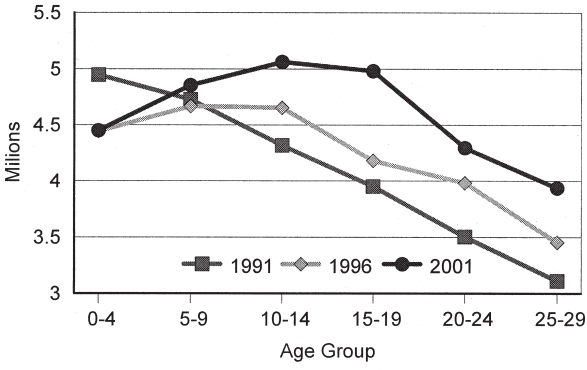
52. Official South African mortality data are seriously undermined by underreporting and unusual administrative procedures. The reporting of deaths is voluntary, motivated by the need for an official burial permit (necessary only in urban areas) or insurance and welfare benefit claims, and is believed to be largely incomplete [RSA SSA 1997, 2000]. Deaths are recorded years after the fact, and historically, published data only indicated the number of deaths reported within a given year, and not the year of occurrence. Further, the Department of Home Affairs allowed forms coming in after December to be added to the December process month for arbitrary periods of time (e.g., the microdata files for recorded deaths 1996 reveal that 97,000 of the 327,000 recorded deaths for 1996 were processed in "December"), confounding any analysis of the annual series. For the 1997–2000 period, recorded deaths have recently been reported in a cumulative fashion, based upon year of occurrence [RSA SSA 2001a]. As cumulated up to 2001, these data indicate a 50 to 100 percent increase in the number of male and female deaths in the 25–34 age group from 1997 to 2000 alone.

53. Or, alternatively, the reduction in the expected remaining lifespan of, for example, a living 16 year old male from 51.4 years prior to the epidemic to 39.4 years in 2000 has had little effect on the demand for investment in human capital.

TABLE IV
SCHOOL ENROLLMENT AND ORPHANHOOD RATES

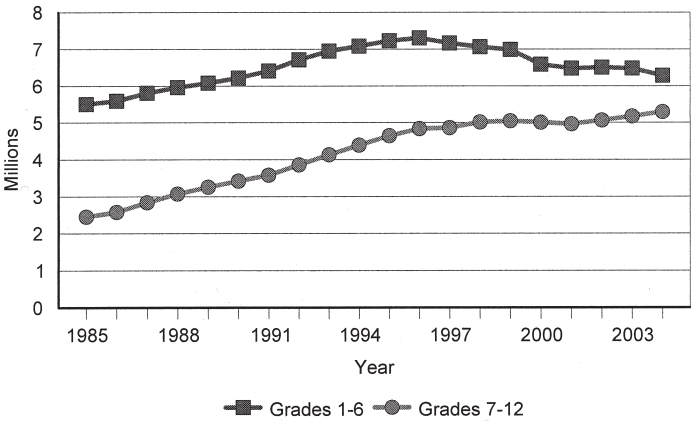
| | School enrollment | | Maternal orphan | | Paternal orphan | |
|---|---|---|---|---|---|---|
| | Ages 7–15 | Ages 16–20 | Ages 7–15 | Ages 16–20 | Ages 7–15 | Ages 16–20 |
| OHS 1993* | .95 | .72 | | | | |
| OHS 1994 | .96 | .75 | | | | |
| OHS 1995# | .97 | .76 | .034 | .057 | .14 | .21 |
| OHS 1996 | .96 | .77 | .026 | .045 | .12 | .18 |
| OHS 1997 | .94 | .77 | .032 | .055 | .13 | .20 |
| OHS 1998 | .93 | .74 | .032 | .054 | .12 | .20 |
| OHS 1999 | .95 | .74 | | | | |
| GHS 2002 | .97 | .71 | .057 | .084 | .15 | .24 |
| GHS 2003 | .97 | .72 | .061 | .084 | .16 | .22 |
| Census 1996 | .89 | .74 | .028 | .053 | .11 | .19 |
| Census 2001 | .94 | .70 | .045 | .070 | .14 | .21 |

Data calculated from the microdata files of each survey/census (available from http://www.statssa.gov.za), weighted by the person weights. OHS = October Household Survey; GHS = General Household Survey. Individuals with unknown or imputed enrollment or parental mortality are excluded from the denominators. The 1993–1994 and 1999 OHS did not collect information on parental mortality. * Enrollment explicitly restricted to full-time only; all other years include part-time students. # Microdata do not include any "don't know" or "no response," suggesting imputation. Maternal and paternal orphanhood data (which in these age groups typically include a few percent unknown) are probably not comparable with other years.




S: RSA CSS [1992], RSA SSA [2004c].

FIGURE XII
Census Population



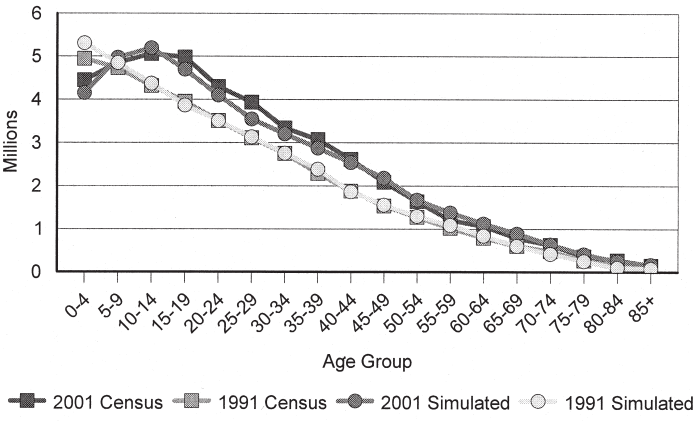

S: Statistics South Africa, RSA Statistics in Brief, and DOE documents.

FIGURE XIII
Aggregate School Enrollment

population distribution recorded in the past three censuses. In 1991 South Africa had the downward sloping profile typical of an economy with rapid population growth. During the 1990s this profile flattened until, by the time of the 2001 census, the number of teenagers exceeded the number of preteens. These trends are confirmed by data on aggregate school enrollment (Figure XIII) which show a movement from rapid growth to stagnation and decline.[54] These data, particularly the ongoing decline in the number of young primary school students, are consistent with a recent, and severe, reduction in fertility.[55] In Figure XIV I graph

54. Changes in the political boundaries of South Africa, along with the untimely death of the only programmer with access to the education database, have left the South African Department of Education without a time series on enrollment (personal communication with DOE officials). The South African Department of Education graciously provided me with scanned copies of numerous historical documents and reports which I used, along with the historical issues of South African Statistics and Statistics in Brief, to produce the series reported in the table. While there were some minor disagreements among sources, the differences are on the order of 1 percent or less. The data refer to ordinary (excluding special education) private and public school students, within the current political boundaries of the Republic of South Africa. The 1985 and 1986 data do not include White students enrolled in private schools, while the 1997 data do not include private school enrollment (equal to about 250,000 in recent years) for any racial group.

55. If the reader will compare Figures XII and XIII, she will see that the growth in the size of census cohorts aged 5–9 and 15–19 between 1991 and 2001 is broadly consistent with the stagnation in primary school enrollment and growth




S: RSA CSS [1992], RSA SSA [2004c], and author's simulations.

FIGURE XIV
Censuses and Simulations

the 1991 and 2001 population distributions produced by the forward and backward projection of the 1995 OHS in the base HIV simulation earlier above. These simulations, based upon my estimates of the impact of HIV on fertility drawn from a third, independent, source (the DHS), provide a close match to the change in relative cohort sizes present in census data. The inescapable conclusion is that the HIV epidemic has produced an abrupt reduction in fertility, endowing future generations with greater material resources per capita.

Having made a case in favor of the paper, I will now do my best to undermine it, focusing in particular on the fertility trends implied by recent census data. Censuses, especially in third-world countries, are bedeviled by the problem of the "undercount." The approach of the Central Statistical Service (now known as Statistics South Africa) in the 1991 census was to completely ignore the census totals[56] and, instead, project the population distribu-

of secondary school enrollment between these two dates, assuming roughly constant enrollment rates.

56. Apparently, 88 areas were "enumerated" using aerial photography supplemented by spot surveys used to convert houses to individuals, "sweeps" were conducted of entire magisterial districts without enumeration areas, and the census form, to be filled out during the census period by the households themselves, was printed only in English and Afrikaans [RSA CSS 1992; RSA SSA 2004d, 2004e].

tion from the 1970 census (considered to be accurate) using mortality and fertility estimates, with adjustment and validation against school enrollment data and private sector sample surveys [RSA CSS 1992]. The 1996 census adopted a different approach, using a postenumeration survey to adjust the population totals under the assumption of independence between the original census and postenumeration samples. Unfortunately, enumeration areas were not properly delineated *prior* to the Census,[57] which also only asked for individual first names or initials, with the result that 22 percent of the postenumeration sample could not be classified as either a "match" or a "miss" [RSA SSA 1998]. The 2001 census again used a postenumeration sample, but this time enhanced using bar codes affixed to houses during the original census, more detailed respondent information, and reconciliation visits to ensure matching, all of which reduced unclassified cases to less than 2 percent [RSA SSA 2004a]. While the 1996 census estimated an undercount of 10.7 percent, the more carefully executed 2001 census raised the undercount estimate to 17.6 percent.[58] The assumption, used in both censuses, of census and postenumeration independence is probably flawed, as censuses and surveys in third-world countries appear to systematically undercount young persons,[59] a systematic error which will not be solved by repeated sampling.[60] Clearly, differences in methodology, and serious concerns about accuracy, make intercensal comparisons of demographic trends highly questionable.

57. Bad weather prevented aerial mapping before the census, leading to a reliance on geographic descriptions and household lists given by local authorities.

58. The 2001 adjustment used an additional reenumeration sample to eliminate overcounts in the original census (e.g., falsifications and duplications), which was not done for the 1996 census. Calculated on the same basis as the 1996 census, the undercount was 20.0 percent in 2001 [RSA 2004a].

59. In my experience this is a consistent problem across a number of countries and samples (to confirm this, one simply has to compare the age distribution of each survey/census with later enumerations, noting the growth in the absolute size of younger cohorts).

60. As an extreme example, consider the case where enumerators never record persons under age five. In this case, two random samples of households will still reveal no one under age five and the postenumeration adjusted census population will show no individuals in this age group. The problem here is the assumption of independence, not in the selection of households, but in the coverage of individuals within households. This problem is recognized by Statistics South Africa, which has concluded that both the 1996 and 2001 censuses, after adjustment for the undercount, underestimate the number of 0–4 year olds [RSA SSA 2004b, 2004d]. If the reader looks carefully at the data in Figure XII, she will see that the absolute size of youth cohorts grows from 1996 to 2001 (simply shift the 1996 curve one position to the right). This is consistent with a systematic failure to enumerate youth, but, of course, could also be produced by the different adjustments for the undercount.

These problems do not negate all of the preceding discussion. The systematic failure of censuses to enumate youth does not explain why the relative size of the youngest cohorts is considerably smaller in the 2001 South African census than in the 1996 census, as the two censuses used similar methodologies. Additional data (e.g., on school enrollment and savings) support the assumptions and arguments made in the simulations. Nevertheless, it is the case that aggregate African data, even in one of the most data-rich African countries, are highly problematic. For this reason, I have focused on estimates and simulations based upon data from three careful surveys, the OHS, the DHS, and the seroprevalence studies of pregnant women. Aggregate data, such as they are, are strongly supportive of the most optimistic of these simulations, indicating a rapid decline in fertility and sustained investment in human and physical capital.

## VIII. Conclusion

Figure XV graphs my projections of the South African population with and without the HIV epidemic. In the absence of the epidemic, the population would have grown rapidly, reaching 110 million persons by 2050. With the epidemic, the combination of adult mortality, HIV infant mortality, the powerful effects of community HIV infection on fertility, and the endogenous re-

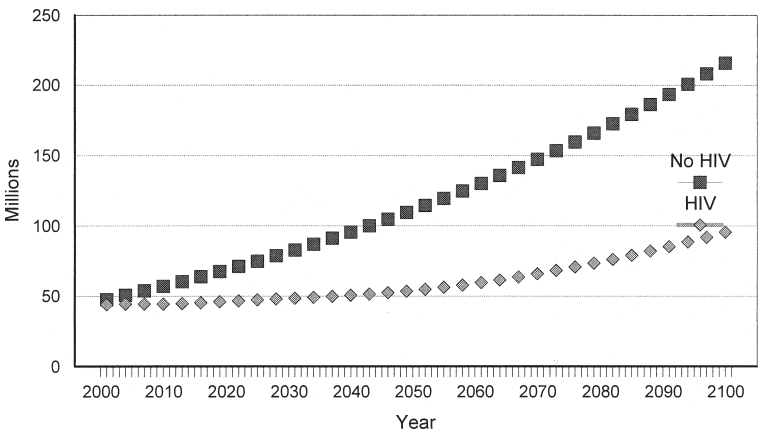



FIGURE XV
Population

sponse of fertility to higher wages contrive to keep the population below 50 million for almost 50 years. As shown in this paper, the positive effects of lower population growth are strong enough to counteract the most pessimistic forecasts of the human capital losses of AIDS-orphaned children, implicitly endowing the economy with extra resources which can be used to extend the lifespan of the afflicted and still leave reserves to raise the per capita welfare of future generations.

An emphasis on per capita outcomes is a particularly calloused notion of "welfare," one that does not care about the number of individuals, dead or unborn. It is by no means the way I would evaluate the agonized deaths of millions of young adults and children invaded by infections and cancers. There is, however, a long-standing emphasis on GDP per capita in the economics profession as a measure of economic well-being. In the classroom, this has led to the Solovian-based lessons on the merits of lower population growth taught to generations of graduate students, while, in the real world, it has supported the encouragement of fertility control through subsidies and, in some cases, draconian coercive measures. One cannot endlessly lament the scourge of high population growth in the developing world and then conclude that a reversal of such processes is an equal economic disaster. The AIDS epidemic is a humanitarian disaster of millennial proportions, one that cries for assistance. It is not, however, an economic disaster.

## Appendix: Construction of Variables from the Microdata Sets

The variables used in Section IV and Table I are constructed as follows.

Wages per Hour—Individuals in the OHS report whether they work for someone else or for themselves. Those working for someone else report their income from their main job, precisely or in terms of a range (e.g., R2000–R2499), and whether this income is daily, weekly, monthly, or annual. I multiply the daily, weekly, and monthly incomes (or income brackets) by 240, 48, and 12, respectively, to arrive at annual incomes. Individuals also report their weekly income in kind received in the form of transport, food, and other. I multiply these by 48 and add them to wage income, treating entries with no reports as values of zero. I take annual taxes as the reported amount deducted per day, week, month, or year (imputing a value of zero for blank entries),

multiplied by the factors listed above.[61] Dividing by the response to the question on hours of work in the last seven days the individual worked, yields my estimate of annual before- and after-tax wage income per weekly hour of work. I exclude from the sample a few hundred individuals who report that they work both for themselves and for others. The behavioral equations (3)–(6) of Table I exclude individuals under the age of 25, as their education, determining their lifetime behavior, is as yet incomplete. However, I include all individuals, including those under 25, in the wage regressions of columns (1)–(2), under the assumption that their incomes appropriately reflect the return to their current educational capital. Excluding individuals under 25 or, as noted in the paper, multistep procedures to adjust for selectivity bias, both have little effect on the estimated educational profile of wages.

Hours of work—The dependent variable in columns (3) and (4) of Table I is the number of hours of work during the last seven days that the individual worked (values of zero are entered for individuals who were not working). The sample covers all individuals over the age of 25 for which I have data on educational attainment.

Education—Individuals surveyed in the OHS and DHS report their highest standard passed or education level obtained, with some grouping of levels and some differences in choices between the surveys. I convert these into standardized years of education for the regressions of columns (1) and (2) of Table I as follows: (i) No schooling or less than one year completed = 0 years; (ii) Sub A/sub B/grade 1/grade 2/Std 1 = 2 years; (iii) Standards 2–10 = standard year + 2; (iv) Diploma/certificate with Std 9 or lower or further studies incomplete = 13 years; (v) Diploma/certificate with Std 10 or diploma/other postschool complete = 14 years; (vi) Degree or further degree complete = 16 years. These fourteen educational categories are then used to identify the impact of wage income in columns (3)–(6) of Table I and constitute the educational state space of column (6)'s ordered probit and the simulations in the paper.

Fertility—I use women's reports of their past pregnancies in the DHS to reconstruct the number of births in each year of their lives. I count pregnancies that were lost before term or resulted in

61. As noted in Section IV above, I get fairly similar model results when I use the tax code to calculate after-tax incomes.

stillbirths as births, under the assumption that the infant mortality estimates I derive from the OHS include such deaths. As shown in the unpublished appendix, the resulting estimates of fertility and mortality reproduce the OHS adult population distribution. If the definition of a pregnancy is restricted to a live birth, and the OHS infant mortality rates are then applied, predicted historical cohort sizes are too small. Although I exclude from the sample women younger than 25 at the time of the survey, fertility at earlier ages is captured by the retrospective histories of older women. I restrict the sample to completed years of life, i.e., do not include the fertility history since each woman's last birthday which, for obvious calendar reasons, shows systematically lower fertility rates.

Mother's Education—Individuals in the OHS report their relationship to the head of the household or reference person and the respondent numbers of their parents, if these are members of the household. I use this information to construct two sets of identified mothers: (1) Based upon relationship, where mothers are either the reference person where the respondent (the child) reports being the son or daughter of the reference person, or mothers are respondents who report that they are the parent of the reference person (the child). I eliminate cases where the age difference between the presumed mother and child is less than 12 or more than 60 years or where multiple women claim to be the mother of the reference person. (2) Based upon the reported respondent number of mothers, eliminating cases where the reported "mother" is male, where the age difference between mother and child is less than 12 or more than 60 years, or where the mother and child's individually reported relationships to the reference person are incompatible (e.g., when the mother claims to be the daughter of the reference person, but the child does not claim to be the grandchild of the reference person). I then merge the two sets of information, giving precedence to mothers identified on the basis of reported relationship in the 81 cases where the data sets disagree.

Non-AIDS Mortality—Although not shown in Table I, I use the OHS sample reports of deaths in the household in 1994–1995 to estimate mortality in the absence of AIDS. I add individuals who reportedly died in the period November 1994–October 1995 to the sample of surviving individuals and run probits for the probability of death in a given year on sex and a quadratic in age.

I divide the sample into two groups, aged 0–11 and 12–99, so as to allow simple quadratics in age to capture the rapid decline of youth mortality and rise of adult mortality with age. As the sex dummy is not a significant determinant of youth mortality, I exclude sex from the final estimating equation for that group. As shown in the unpublished appendix, my estimates of pre-AIDS mortality match the historical evolution of the South African population. In the simulations, I assume that mortality due to AIDS and non-AIDS causes are independently distributed.

CES data—To predict the behavior of the U. S. population using the S. A. coefficient estimates, I make use of the interview survey data files of the 2000 consumer expenditure survey, which includes five quarterly interviews running from first quarter of 2000 through the first quarter of 2001. I measure after-tax incomes as the family member's gross pay minus federal and state taxes deducted (grospayx-amtfed-sltaxx), multiplying by 48, 24, 12, 4, and 24 to convert weekly, biweekly, monthly, quarterly, and bimonthly reports to annual values ("other," i.e., unknown, payperiods are dropped from the sample). Hours of work are measured as the family member's usual weekly hours of work (inc_hrsq). I then use an interval regression (to account for topcoding) to estimate a wage regression as in column (2) of Table I, restricting the sample to employees (incomey $\leq 4$). Education is naturally coded in years, except for postsecondary, where I count some college or associate degrees as 14 years, bachelor's as 16 years, and master's and above as 18 years. After estimating the wage equation, I use it to predict the behavior of the 2000 U. S. population using the five quarters of the CES population sample with weights adjusted for months in the scope of the 2000 year (finlwt21*mo_scope/12). I identify mothers in cases where members identify themselves as the child of the household reference person by using the reference person (if female) or the spouse of the reference person (if the reference person is male). I also identify mothers using female individuals who report themselves as the mother of the reference person (the child). In all cases, I drop observations where the age difference between mother and child is less than 12 or more than 49 years.

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