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Technology Adoption, Mortality, and Population Dynamics*

John Hejkal† B. Ravikumar‡ G. Vandenbroucke§

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Abstract

We develop a quantitative theory of mortality trends and population dynamics. We emphasize diseases as causes of death and individuals’ decisions to reduce their mortality by adopting, at some cost, a modern health-related technology. Adoption confers a dynamic externality: Adoption becomes cheaper as more individuals acquire the modern technology. Our model generates an S-shaped diffusion curve, whose shape dictates the pace of mortality reduction in each country. We use the model to explain the gradual decline of mortality in Western Europe in the 19th and 20th centuries as well as the rapid decline in poor countries since 1960. Unlike a Malthusian theory of population, our model accounts for the well-known historical disconnect between mortality decline and economic takeoff and its recent equivalents: cross-country mortality convergence despite lack of income convergence and mortality miracles, i.e., declining mortality in countries with declining income. Our model is also consistent with the observed acceleration in world population, which cannot be explained solely by declining fertility à la Becker.

JEL codes: I12, I15, J11, E13
Keywords: Mortality, population dynamics, technology diffusion, convergence.

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1 Introduction

The search for an economic theory of population is as old as economics itself (e.g., Smith (1776) and Malthus (1798)). A theory of population dynamics is useful for answering (at least) two questions: what explains economic stagnation during the pre-industrial era; what explains the transition to modern economic growth in some countries since then? Malthus (1798) and Ricardo (1817) offered an answer to the first question with a theory of population and production. An answer to the second question requires a theory of population dynamics to link Malthus to models of modern growth à la Lucas (1988) and Romer (1986, 1990). Becker, Murphy and Tamura (1990) and Galor and Weil (1999), for instance, provided such a link by incorporating a theory of fertility into growth models.\footnote{See also Kremer (1993), Tamura (2000, 2002), Jones (2001), Hansen and Prescott (2002) and Lucas (2002).}

![Figure 1: The growth rate of world population and its components](https://gapminder.org/)

Note: The growth rate of world population is the difference between CBR and CDR.

Source: https://gapminder.org/.

Malthusian theory of population implies a positive correlation between income and population. Becker (1960)'s theory is on fertility decisions.\footnote{See surveys by Doepke (2015), Doepke and Tertilt (2016) and Greenwood, Guner and Vandenbroucke (2017).} Both Malthusian and Beckerian approaches to population are incomplete, however. Recall that the crude birth rate (CBR) is the number of births per person and the crude death rate (CDR) is the number of deaths per person. So, to a first approximation, the growth rate of population is the difference CBR – CDR, which has been increasing over time (see Figure 1).\footnote{Delventhal, Fernández-Villaverde and Guner (2021) document decline in CBR and CDR for a large number of countries spanning more than 250 years.} The Beckerian theory can explain the secular decrease in CBR, but this decrease by itself cannot explain the observed increase in the growth rate of population. Thus, a theory of population dynamics requires a theory of mortality reduction. Such a theory cannot be Malthusian since the
growth rate of population in poor countries over the last 60 years has been higher than that in the rich
despite the well-known lack of income convergence (e.g., Parente and Prescott (1993), Barro (2012)
and Jones (2016)).

A theory of mortality reduction has to confront several facts. First, poor countries accounted for half
of the increase in world population since 1960, and CDR reduction in poor countries was a major
contributor to that increase. Second, CDR in poor countries was higher than that in the rich in 1960,
but is below that in the rich in 2019. Furthermore, the average person born in a poor country in 1960
could expect to live for 42 years, whereas in a rich country he could expect 70 years. In 2019
these numbers had increased to 69 and 80, respectively. That is, the life expectancy in poor countries
had increased by 27 years while that in the rich countries had increased by only 10 years. Third,
there are mortality miracles, i.e., CDR reductions in countries whose incomes have been stagnant or
decreasing since 1960. Madagascar’s GDP for example, decreased at a rate of almost one 1% per year
while its CDR decreased at the rate of more than 2% per year. The mortality miracles are recent
equivalents of the well-known historical disconnect between mortality decline and economic takeoff
(e.g., Livi-Bacci (1991) and Mokyr (1993)), so the theory has to confront time series evidence over the
last two centuries as well.

We present a quantitative theory of population dynamics based on mortality. Our approach to mor-
tality follows Becker’s economic approach to fertility: It is based on individuals’ decisions. Specifi-
cally, our theory emphasizes diseases as causes of death and individuals’ decisions to adopt, at some
cost, health-related practices (technologies) that reduce their risk of dying. In our model there are
two technologies—obsolete and modern—characterized by age-specific survival probabilities, with the
modern technology offering a higher chance of survival at all ages. We refer to individuals using
the modern (obsolete) technology as modern (obsolete) individuals, for short. Our goal is to explain
world population dynamics over the last two centuries and the contribution of poor countries to world
population since 1960.

Our model’s exogenous driving variables are total factor productivity (TFP) and CBR. The initial
conditions are the proportion of modern individuals and the age distribution. The evolution of the
proportion of modern individuals depends on individual decisions: Obsolete individuals of any age must
spend resources—time and/or goods—to increase their odds of becoming modern. Cross-sectional
heterogeneity is thus limited to age and the proportion of modern individuals by age. The costs and
benefits of adopting the modern technology depend on TFP: Higher TFP raises the time cost and
lowers the good cost; it also raises the benefit of adoption by increasing the present value of income. We
further assume a dynamic externality: As more individuals adopt the modern technology, it becomes
cheaper to acquire. The evolution of the age distribution depends on CBR and age-specific mortality
rates, which are weighted averages of death probabilities, with weights given by the proportions of
obsolete and modern individuals.

Both the age distribution and age-specific mortality rates are endogenous in our model. Thus, our
testable implications are not only for these variables across countries and over time, but also for
two standard summary statistics of mortality: CDR and life expectancy at birth (LEB). The former depends on the age distribution and age-specific mortality rates; the latter depends only on age-specific mortality rates (see Appendix A for details).

The proportion of modern individuals is the key endogenous variable driving the testable implications. This proportion evolves according to an S-shaped diffusion curve, which is country specific and whose shape dictates the pace of mortality reduction. The S-shaped diffusion curve that describes the technology adoption in a country at each point in time depends on the country’s TFP and initial proportion of modern individuals. Rich countries are on a higher diffusion curve relative to poor countries because they have higher TFP and a higher initial proportion of modern individuals, implying a lower cost and a higher benefit of adopting the modern technology. Thus, rich countries have a lower mortality compared with poor countries, which explains the initial negative correlation in the data. Rich countries are on a flatter diffusion curve than poor countries because they start with a higher proportion of modern individuals. Thus, mortality declines at a slower rate in rich countries, which explains the eventual mortality convergence across countries and the speed of convergence.

How does the model generate mortality miracles? Because of the dynamic externality, the cost of adoption decreases as the proportion of modern individuals increases, which serves as an engine for more adoptions. Thus, mortality can decrease even with stagnant or decreasing income.

We calibrate the model to fit Sweden’s CDR from 1751 to 2017, taking the initial age distribution and the time series of CBR and TFP from data. Our proxies for the obsolete and modern technologies are the observed age-specific survival probabilities in 1751 and 2017, respectively. The critical parameters to calibrate are the initial proportion of modern individuals, and the parameters mapping resources into the probability of becoming modern. We then use the model to simulate mortality and population statistics for a recent sample of 87 countries since 1960, and for a sample of Western European countries and the world in the 19th and 20th centuries.

The model fits the data well for CDR, LEB, and age distribution in our sample of 87 countries. The model-generated population matches almost all of the increase in population. In the model and data, half of the population increase takes place in poor countries. Furthermore, the model reproduces the cross-country mortality convergence. In Section 2.4 we document that LEB convergence is largely accounted for by the reduction in age 0-4 mortality rates in poor countries; our model is consistent with this fact. Our model also reproduces mortality miracles: It implies a reduction in mortality for all countries that experienced a simultaneous decrease in GDP and mortality in the data.

The model is consistent with the acceleration of world population despite the decreasing CBR and reproduces the secular decline in mortality in the world and in Western European countries since the 19th century. Comparing Western Europe in the 19th century to poor countries in the 20th century, the decline in CDR was gradual in the former and rapid in the latter. For instance, in France, it took almost 90 years for the CDR to decline from 20 to 10; in poor countries the same decline took only 30 years. Our model is consistent with these observations.

Two remarks about our theory are in order. First, absent costly adoption by individuals, every one in
our model would adopt the modern technology immediately and diffusion would be the same across countries, regardless of TFP. There would be no cross-country differences in age-specific mortality rates or LEB. This would be at odds with the data.

Second, our theory is not about large-scale investments in sanitation and medical infrastructures. Explanations of mortality reductions based on such programs are not different from explanations based on economic growth (see Mokyr and Stein (1997)). Furthermore, these programs are unlikely to have been the main cause behind pre-20th century reductions of mortality in Europe (see Wohl (1983)). The modern technology in our model represents inexpensive individual-level practices that reduce the risk of dying, e.g., boiling water, washing hands. This is in line with Preston (1975), Easterlin (1999) and Mokyr (1993, 2000), for instance. Mokyr (1993) argues that these practices were promoted in Europe during the early 1800s by the hygienist movement (which predated germ theory) and were not based on expensive medical research.

2 Facts

We use historical data from a variety of sources and recent data (since 1960) from the World Bank. Specifically, using the World Development Indicators and the Health Nutrition and Population Statistics we assemble annual data on CBR, CDR, LEB, real gross domestic product per capita (GDP), and population by age from 1960 to 2019 for a group of 87 countries. We exclude China from the sample because, during the Great Leap Forward (1958-62) and the ensuing famine, China’s mortality increased by 150% in a span of a few years. As a share of the world excluding China, the 87 countries in our sample cover 74% of the population in 1960, 78% of deaths, and 79% of GDP. In 2019, these numbers are 78%, 76%, and 81%, respectively. Age-specific mortality rates for countries in our sample are from the United Nations’ World Population Prospects for 5-year age groups at five-year intervals from 1960 to 2015.

We group countries into quintiles of GDP in 1960 and keep the composition of quintiles constant at all dates. We refer to countries in the first (bottom) quintile as poor and those in the fifth (top) quintile as rich. Our sample spans large differences in GDP: The GDP of poor countries is 2.2% that of the rich in 1960.

2.1 Increasing population growth rate despite decreasing CBR

See Figure 1. It took 82 years for world population to increase from 1 billion to 1.5 billion, an average annual growth rate of less than 0.5%; the CBR during this period was above 40. In contrast, it took only 8 years for world population to increase from 3 billion to 3.5 billion; the population growth rate quadrupled to almost 2 percent per year, while the CBR in this period was 35. Since the growth rate of world population is the difference between CBR and CDR, it can only increase because the latter

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Footnote: See Appendix B for examples of diseases that can be mitigated by inexpensive means.
is decreasing faster than the former.

The pattern of increasing population growth rate despite a decreasing CBR can also be found among some poor countries recently. Between 1960 and 1970, the annual growth rate of population in poor countries was 2.51%. It then increased to 2.63% in each of the following two decades. At the same time, the average crude birth rate declined from 42 births per 1,000 people, to 39 and then 35.

2.2 Key to recent increase in population: CDR in poor countries

Poor countries account for 49% of the increase in total population in our sample between 1960 and 2019. Without the decrease in CDR in poor countries, the total population would have been almost one billion less in 2019.

To compute the contribution of poor countries to the change in total population in our sample, we abstract from migration, use the rate of natural increase, and construct population time series for each country \( i \), \( p_{i,t} \). That is, we assign to country \( i \) its observed population in 1960 and construct:

\[
p_{i,t+1} = p_{i,t} \left( 1 + \text{CBR}_{i,t} - \text{CDR}_{i,t} \right) \quad t = 1960, \ldots, 2018.
\]

Total population, \( p_t = \sum_i p_{i,t} \), increases by 3.11 billion. Let \( p_{t}^{\text{poor}} \) denote the population of poor countries, i.e., the sum of populations of countries belonging to the first quintile. The population of poor countries increased by 1.48 billion. The contribution of poor countries to the change in total population is then

\[
\frac{p_{2019}^{\text{poor}} - p_{1960}^{\text{poor}}}{p_{2019} - p_{1960}} = 0.49.
\]

To compute a back-of-the-envelope counterfactual population, we keep CDR in poor countries at their 1960 values in Equation (1). Then \( p_t \) increases by 2.18 billion instead of 3.11 billion. Thus, absent the decrease in CDR in poor countries, there would have been almost one billion fewer people in 2019. More importantly, there would have been almost one billion fewer poor people. Furthermore, the poor countries’ share of world population would have declined from 38% to 32%; instead, the share increased from 38% to 44% in the data due to the decline in CDR in poor countries.

Note that the rate of natural increase implies a total population increase of 3.11 billion, whereas the observed change in population is 3.14 billion. Given the minor role of migration, for the rest of the paper we will treat the rate of natural increase and population growth as synonymous.

2.3 Mortality convergence despite lack of income convergence

Panel A of Figure 2 shows GDP in rich and poor countries since 1960. Despite the lack of convergence in GDP there is convergence in CDR, as illustrated in Panel B: The CDR in poor countries was higher than that in the rich by 12 in 1960, but lower by 2 in 2019. The cross-country elasticity of CDR to
GDP increased from −0.25 in 1960 to 0 in 2019. Benin, for example, had a CDR three times and a GDP 4% that of the U.S. in 1960. In 2019, its CDR is the same as that of the U.S. but its GDP is only 2% of that of the U.S.

The convergence of LEB has been well-documented e.g., Preston (1980), Vallin and Meslé (2004), Riley (2005), and Ram (2006). Panel C of Figure 2 shows this in our sample: LEB in rich countries was 28 years higher than that in poor countries in 1960, but in 2019 the difference was 11 years. Other studies point out that the convergence in life expectancy occurred even though there has been no convergence in GDP, e.g., Kuznets (1973), Bourguignon and Morrisson (2002), Becker, Philipson and Soares (2005), and Acemoglu and Johnson (2007). The cross-country GDP elasticity of LEB in our sample declined from 0.14 in 1960 to 0.06 in 2019.
Panel D summarizes the convergence of mortality and divergence of income using the cross-country dispersion of CDR, LEB, and GDP.

### 2.4 Key to mortality convergence: age 0-4 mortality in poor countries

Figure 3 illustrates the change in age-specific mortality rates between 1960 and 2015 for rich and poor countries. The major differences are in the 0-4 mortality rate and above-80 mortality rate. For example, the decline in age 0-4 mortality rate is 51 in poor countries and only 5 in rich countries.

To understand the role of age-specific mortality rates for LEB convergence, consider the following experiment. Set the change in poor countries’ age 0-4 mortality rate to 5, the same as in the rich countries, and leave all other age-specific mortality rates to be the same as in the data for both groups of countries. The resulting LEB is 28 years higher in rich countries in 2015, instead of 12 in the data, i.e., there is hardly any convergence in LEB.\(^5\) In contrast, if we set the change in poor countries’ above-80 mortality rate to be the same as in the rich countries, and leave all other age-specific mortality rates as in the data, the resulting LEB is 12 years higher in rich countries. That is, the convergence is not affected by the change in above-80 mortality rate.

This result follows from the compounding effect of age-specific mortality on LEB: An increase in infant mortality has a larger effect than an increase in old-age mortality (Equation (A.3) in Appendix A).

\(^5\)The gap in the data in 2015 differs slightly from what we reported in Section 2.3 because (i) the time period and the source of the data are different, and (ii) since the data on age-specific mortality rates from the World Population Prospects are for 5-year age groups, we computed LEB by interpolating the mortality rates.
Thus, the key to LEB convergence is the decline in age 0-4 mortality rate in poor countries.

### 2.5 Mortality miracles

Livi-Bacci (1991) and Mokyr (1993) noted a disconnect between economic takeoff and mortality decline. As a case in point, Figure 4 shows demographic and economic data for England from the 16th to 19th centuries. Panel A indicates that CDR declined throughout the 18th century. Panel B, however, indicates that economic takeoff did not occur before the early part of the 19th century. Specifically, TFP in the agriculture and manufacturing sectors are flat throughout the 18th century and wages increase only at the onset of the 19th century.

Preston (1975) and Soares (2007) conclude that factors other than GDP have major effects on a country’s mortality, and it is true for rich and poor countries alike. Panel A of Figure 5 shows a scatter plot of CDR growth and GDP growth between 1960 and 2019 for each country in our sample. Faster economic growth does not correlate with faster CDR reductions. All of the countries with negative growth experienced a reduction in CDR. Conversely, there are countries with increasing CDR but none of them experienced negative growth. Note that the northwest quadrant of Panel A is empty.

Panel B of Figure 5 shows a similar scatter plot for LEB and GDP. All countries experienced increasing LEB, whether their GDP grew or not. How can LEB increase in countries experiencing an increase in CDR (northeast quadrant of Panel A)? The increase in CDR in these countries is due to changes in their age distributions. These countries experienced larger-than-average decreases in CBR during the period, implying an increasing proportion of older individuals and, thus, an increasing CDR. The increase in LEB, however, is due to declining age-specific mortality rates. In Japan, for example, CDR increased by 4 and LEB increased by 17 years. The proportion of people 65 and above increased from 5% to 24%. Age-specific mortality rates in 1960 were 0.002 and 0.03 at ages 25 and 65, respectively. In 2019 these rates had declined to 0.0003 and 0.007.

### 3 Model

Time is discrete and indexed by \( t \). The economy is populated by overlapping generations of individuals living up to a maximum age \( J \). The preferences of an individual of generation \( t \) (i.e., an individual of age 0 at date \( t \)) and age \( a \) are represented by

\[
E_{t,a} \left[ \sum_{j=a}^{J} \beta^{j-a} U(c_{t,j}) \right],
\]

where \( \beta \in (0, 1) \) is the subjective discount factor, \( U \) is a utility index, \( c_{t,j} \) represents consumption at age \( j \), and \( E_{t,a} \) is the expectation operator where the expectation is calculated using the appropriate
Figure 4: Mortality and the economy in England from the 16th to 19th centuries


Figure 5: Growth rates of crude death rate and life expectancy at birth vs. economic growth, by country, 1960-2019

Note: Panel A plots the growth rate of CDR between 1960 and 2019 against that of GDP. Panel B plots the growth rate of LEB against that of GDP.

There are two health technologies available at any point in time: “obsolete” and “modern,” indexed by $i \in \{o, m\}$. These technologies are characterized by age-specific survival probabilities, $\{s^i_j\}_{j=0}^J$, where $s^i_j$ denotes the probability of survival from age $j$ to $j + 1$, conditional on being alive at age $j$, for $i \in \{o, m\}$. We label individuals with the modern technology as “modern individuals.” Similarly, individuals with the obsolete technology are “obsolete individuals.” Since $J$ is the maximum length of life, we assume $s^o_J = s^m_J = 0$.

Individuals become “economically active” at age $k > 0$. From age $k$ to $J$ they are endowed with one unit of time each period. The wage rate is denoted by $w_t$ in period $t$. There is no saving. Modeling individuals of age $j \leq k$ is necessary to keep track of the entire age distribution, which in turn affects mortality and population dynamics.

Modern individuals allocate all their time to working; their choice problem is trivial. The value of a modern individual of generation $t$ and age $j \geq k$ is

$$V^m_{i,j} = U(w_{t+j}) + \beta s^m_j V^m_{i,j+1}.$$  \hfill (3)

An obsolete individual can successfully adopt the modern technology with some probability and become modern. The probability of successful adoption for an age $j > k$ member of generation $t$ is

$$A(y_{t,j}, h_{t,j}, \pi_{t+j}),$$  \hfill (4)

where $y_{t,j}$ is goods resources, $h_{t,j}$ is time, and $\pi_{t+j}$ is the proportion of economically active modern individuals at the beginning of period $t + j$. The value of an obsolete individual is

$$V^o_{i,j} = \max_{y,h} U(w_{t+j}(1 - h) - y) + \beta A(y_{t,j}, h_{t,j}, \pi_{t+j}) s^m_j V^m_{i,j+1}$$

$$+ \beta (1 - A(y_{t,j}, h_{t,j}, \pi_{t+j})) s^o_j V^o_{i,j+1}.$$  \hfill (5)

Since the probability of survival is 0 at age $J$, the terminal condition for modern and obsolete individuals is

$$V^m_{i,J} = V^o_{i,J} = U(w_{t+J}).$$

At an interior, the optimal choice of an obsolete individual is characterized by the first-order conditions

$$y_{t,j} : \quad 0 = U_1(w_{t+j}(1 - h_{t,j}) - y_{t,j}) - \beta A_1(y_{t,j}, h_{t,j}, \pi_{t+j}) \Delta_{t,j},$$  \hfill (6)

$$h_{t,j} : \quad 0 = U_1(w_{t+j}(1 - h_{t,j}) - y_{t,j}) w_{t+j} - \beta A_2(y_{t,j}, h_{t,j}, \pi_{t+j}) \Delta_{t,j},$$  \hfill (7)

where $\Delta_{t,j} \equiv s^m_j V^m_{i,j+1} - s^o_j V^o_{i,j+1}$. (We allow for corner solution in our quantitative experiments.)

A few remarks are in order at this stage. First, we assume that modern individuals do not expend resources every period to “operate” the modern technology. None of our results depend on this as-
sumption. Second, we assume that resources affect the probability of acquiring the modern technology; this is a modeling convenience. The alternative, i.e., adoption is certain once resources are expended, would imply that all individuals in an age group adopt simultaneously. Age-specific survival probabilities would then be step functions of time: low survival until the age group in question adopts, then high survival. This would be at odds with the data. One could introduce within-age heterogeneity to avoid this issue. This additional complexity does not yield additional insights. We model adoption as random so that the model is parsimonious. Our modeling can be interpreted as allowing people to choose over lotteries, à la Prescott and Townsend (1984) and Rogerson (1988). Third, constant \( w_t \) in Equations (6)-(7) does not imply that \( y \) and \( h \) would be constant over time. The evolution of \( \pi \) affects the individual choices due to the dynamic externality.

### 3.1 Population dynamics

Let \( p^i_{t,j} \) denote the population of obsolete and modern individuals of age \( j \) in generation \( t \). The age-\( j \) population of generation \( t \) is then \( p_{t,j} = p^m_{t,j} + p^o_{t,j} \). The age-\( j \) population at date \( t-j \) is \( p_{t-j,j} \), and the total population at date \( t \) is \( p_t = \sum_{j=0}^J p_{t-j,j} \).

We make the following simplifying assumptions on the number of births per person at time \( t \), CBR; (i) birth rates are not age specific, (ii) birth rates are the same for modern and obsolete individuals, and (iii) children of obsolete individuals are obsolete and children of modern individuals are modern. The last assumption represents the notion that a person cannot impart knowledge to their offspring that they do not themselves have, and a newborn acquires the health practices of their parents. The age-0 populations in cohort \( t \) are therefore

\[
p^i_{t,0} = \text{CBR}_{t-1} \sum_{j=0}^J p^i_{t-1-j,j} \quad \text{for } i = o, m. \tag{8}
\]

From age 0 to age \( k-1 \), the populations evolve according to

\[
p^i_{t,j+1} = s^i_j p^i_{t,j} \quad \text{for } j = 0, \ldots, k-1 \text{ and } i = o, m. \tag{9}
\]

The economically active (\( j \geq k \)) populations of generation \( t \) evolve according to

\[
\begin{align*}
p^o_{t,j+1} &= s^o_j p^o_{t,j} \left[ 1 - A(y_{t,j}, h_{t,j}, \pi_{t+j}) \right] \quad \text{(10)} \\
p^m_{t,j+1} &= s^m_j \left[ p^m_{t,j} + A(y_{t,j}, h_{t,j}, \pi_{t+j}) p^o_{t,j} \right]. \quad \text{(11)}
\end{align*}
\]

Equation (10) indicates that a fraction \( 1 - A(y_{t,j}, h_{t,j}, \pi_{t+j}) \) of the age-\( j \) obsolete population remains obsolete and a fraction \( s^o_j \) of those who remain obsolete survives to the next age. Equation (11) indicates that \( A(y_{t,j}, h_{t,j}, \pi_{t+j}) p^o_{t,j} \) age-\( j \) obsolete become modern and that, together with the \( p^m_{t,j} \) already-modern, they face survival probability \( s^m_j \). Recall that \( \pi_t \) is the proportion of economically
active modern individuals at the beginning of period \( t \):

\[
\pi_t = \frac{\sum_{j=k}^J p^m_t-j,j}{\sum_{j=k}^J p^m_t-j,j + \sum_{j=k}^J p^o_t-j,j}.
\] (12)

3.2 Mortality

It follows from Equations (10) and (11) that the deaths to account for in a period are: the deaths of those who started the period as modern individuals, those who started as obsolete and became modern, and those who started as obsolete and remained obsolete. The crude death rate is then

\[
\text{CDR}_t = \frac{1}{p_t} \sum_{j=0}^J \left( p^m_{t-j,j} + A_{t-j,j}p^o_{t-j,j} \right) \left( 1 - s^m_j \right) + \left( 1 - A_{t-j,j} \right) p^o_{t-j,j} \left( 1 - s^o_j \right),
\] (13)

where \( A_{t-j,j} \equiv A(y_{t-j,j}, h_{t-j,j}, \pi_t) \). The age-\( j \) mortality rate at date \( t \) is

\[
q_{t-j,j} = \frac{\left( p^m_{t-j,j} + A_{t-j,j}p^o_{t-j,j} \right) \left( 1 - s^m_j \right) + \left( 1 - A_{t-j,j} \right) p^o_{t-j,j} \left( 1 - s^o_j \right)}{p_{t-j,j}},
\] (14)

i.e., the fraction of the age-\( j \) population that died between dates \( t \) and \( t + 1 \). It follows that \( \text{CDR}_t \) defined in Equation (13) becomes

\[
\text{CDR}_t = \frac{1}{p_t} \sum_{j=0}^J p_{t-j,j} q_{t-j,j}.
\] (15)

Equations (15) shows how \( \text{CDR} \) depends upon age-specific mortality rates and age distribution. Both are endogenous. Age-specific mortality rates, \( q_{t-j,j} \), are governed by the proportion of modern individuals in each age group which in turn affects the age distribution, \( p_{t-j,j}/p_t \).

Life expectancy for just-born individuals at time \( t \) is the expected years of life under the assumption that they experience the age-specific mortality rates observed in the cross section at time \( t \). \( \text{LEB} \) at date \( t \) is then

\[
\text{LEB}_t = \sum_{j=1}^J jq_{t-j,j} \prod_{k=0}^{j-1} \left( 1 - q_{t-k,k} \right),
\] (16)

which simplifies to

\[
\text{LEB}_t = \sum_{j=1}^J \prod_{k=0}^{j-1} \left( 1 - q_{t-k,k} \right).
\] (17)

See Appendix A for details. Note that \( \text{LEB}_t \) depends on age-specific mortality rates but not on the age distribution.
3.3 Technology diffusion

The flow of individuals of age $j$ from obsolete to modern during period $t$ is

$$A(y_{t-j,j}, h_{t-j,j}, \pi_t) p_{t-j,j}^o.$$  

Let $A$ be of the form $A(y, h, \pi) = \pi G(y, h)$—as we assume in Section 4 for our quantitative exercise—then the flow from obsolete to modern is

$$A(y_{t-j,j}, h_{t-j,j}, \pi_t) p_{t-j,j}^o = \pi_t p_{t-j,j}^o G(y_{t-j,j}, h_{t-j,j}).$$

As in models of diffusion, our model exhibits the familiar S-shaped pattern. When the proportion of modern individuals is close to 0, the flow of adopters is “small.” When the proportion of modern individuals is close to 1, there are few obsolete individuals and, therefore, the flow of adopters is “small” as well.

The S-shaped pattern of diffusion is important to understand the convergence of mortality between poor and rich countries. Consider a country where the proportion of modern individuals is represented by the red line labeled “baseline” in Figure 6. Consider a second country with lower initial TFP, represented by the green line labeled “$w_{1/2}$,” but otherwise identical to the baseline. Lower initial TFP has three effects on adoption. First, the benefit of a longer life is lower than in the baseline because the present value of income/consumption is lower. Second, the marginal utility of consumption is higher than in the baseline, therefore diverting goods away from consumption into adopting the modern technology is costlier. Finally, the cost of allocating time to adopting the modern technology is lower than in the baseline because $w$ is lower. The first two effects imply fewer adoptions than in the
baseline. The third effect implies more adoptions. If the sum of all three effects is fewer adoptions (as it is the case in our quantitative analysis in Section 4) then the green line is below the red line in Figure 6. These effects of lower initial TFP are magnified if, in addition, the growth rate of TFP is lower than in the baseline.

Consider now a third country, identical to the baseline except for a lower initial proportion of modern individuals, represented by the blue line labeled “$\pi_1/2$.” There are more adoptions in the baseline because the cost of adoption is lower due to the dynamic externality. Therefore, the blue line is below the red line in Figure 6.

Consider a date $T$, in Figure 6, at which the proportion of modern individuals is higher in the baseline country (red) and, therefore, mortality is lower than in the second (green) and third (blue) countries. The slope is lower in the baseline country which is close to the flat part of its own diffusion curve: There are few obsolete individuals and, thus, the flow of adopters is small. In the other two countries the flow of adopters is higher because they are on the steeper part of their own diffusion curves. Thus, in this example, the lower-TFP country (poor) and the country that started with a lower initial proportion of modern individuals experience a faster decline in mortality than in the baseline (rich) country, which implies mortality convergence.

Note that at time $T$ the correlation between GDP and CDR is negative: A poor country, with a lower TFP and/or a lower initial proportion of modern individuals, will have a higher CDR than a rich country. This cross-country correlation does not imply, however, that mortality reductions over time are correlated with GDP growth. The S-curve is country-specific and depends on factors other than GDP growth.

4 Quantitative analysis

Our quantitative analysis proceeds in three steps. We first present functional forms and calibrate the model to Swedish historical data. Second, we use the calibrated model to analyze mortality and population growth in a set of Western European countries. Third, we analyze mortality and population growth from 1960 to 2019 in our sample of 87 countries.

4.1 Functional forms

The probability of adoption of the modern technology is

$$A(y, h, \pi) = \pi G(y, h)$$

where

$$G(y, h) = \Lambda \left(1 - e^{-\lambda(\alpha_y y^\theta + \alpha_h h^\theta)^{1/\theta}}\right).$$
We use a utility index from the CARA family to represent preferences:

$$U(c) = \Sigma - \exp (-\sigma c).$$

The CARA form for $U$, together with the formulation for $Q$ and $G$, yields an analytical solution to the first-order conditions of an individual’s optimization problem and, therefore, reduces the computational cost of fitting the model to the data. (We have to track the population size and the proportion of modern individuals at each age, at each point in time.) We check, in our computations, that $U$ is always positive. This is an important restriction in models of this nature, as noted by Rosen (1988): When the utility index is negative an extra year of life reduces utility. Appendix C describes the optimal $y$ and $h$.

### 4.2 Calibration

The Palgrave Macmillan Ltd (2013)’s vital statistics for Sweden are the longest of all developed countries, and Swedish data also illustrate a case where the decline in CDR precedes the economic takeoff. We use 1751 as our first date because of data availability on the age distribution.

We use the observed CBR of Sweden from 1751 to 2017 and Sweden’s GDP for $w_t$. Figure 7 shows Sweden’s GDP and CBR. The sequence of growth rates for $w_t$ that best reproduces (in a least-squares sense) Sweden’s GDP (labeled “$w_t$” on Panel A of Figure 7) are 0% prior to 1800, 1.2% from 1800 to 1900, and 2.19% from 1900 to 2017 (Maddison Project Database 2020).

Age-specific survival probabilities for Sweden in 1751 and 2017, which are available from the Human Mortality Database, represent the survival probabilities of obsolete and modern individuals, respec-
Figure 8: Survival probabilities and initial age distribution in Sweden

Note: The 1751 survival probabilities represent the technology for obsolete individuals, and the 2017 survival probabilities represent the technology for modern individuals. We truncate the survival probabilities at age 90, instead of age 111 in the model, for readability.

Source: Human Mortality Database (University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany); available at www.mortality.org or www.humanmortality.de.)

tively. The age-specific survival probabilities are available for all ages from 0 to 111. Thus, we set $J = 111$. Panel A of Figure 8 shows the survival probabilities.

We set the model period to be 1 year and the discount factor $\beta$ to 0.95. We consider the economy from date $t = 1, \ldots, T$, where date 1 corresponds to 1751 and date $T$ to 2017. Both population and $w$ are normalized to 1 at date 1. The age distribution of population at date 1 is that of Sweden in 1751 (see Panel B of Figure 8). We set the economically active age, $k$, equal to 15.

Let $\omega = (\lambda, \Lambda, \sigma, \Sigma, \alpha_t, \alpha_h, \theta, \pi_1)$ denote the list of parameters to be determined, where $\pi_1$ represents the initial proportion of modern individuals. We determine $\omega$ as the solution to the distance-minimization problem:

$$
\min_{\omega} \frac{1}{\sqrt{N}} \sum_{t=1}^{N} \left[ \text{CDR}_t(\omega) - \text{CDR}_{t}^{\text{data}} \right]^2,
$$

where $\text{CDR}_{t}^{\text{data}}$ represents the observed crude death rate in Sweden and $\text{CDR}_t(\omega)$ is the crude death rate implied by the model and defined in Equation (15). We use 8 parameters to match a time series of $N = 267$ observations.

Table 1 reports the calibrated parameters. Figure 9 shows the model’s fit to the time series of CDR. The correlation between model and data is 90.5%. There are, however, spikes in mortality that our model does not reproduce. For instance, two sharp increases in mortality occur in 1772-1773 and in 1834. Larsson (2020) argues that “dysentery (...), fevers, and smallpox caused the sharp rise in mortality in the severe crisis of 1772 and 1773.” The high mortality in 1834 is due to a cholera
Table 1: Model parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exogenous</td>
<td>$\beta = 0.950$, $k = 15$, $J = 111$</td>
</tr>
<tr>
<td>Calibrated Preferences</td>
<td>$\sigma = 3.296$, $\Sigma = 0.185$</td>
</tr>
<tr>
<td>Technology</td>
<td>$\alpha_y = 0.038$, $\alpha_h = 1.002$, $\theta = 0.201$, $\lambda = 0.018$, $\Lambda = 1.395$</td>
</tr>
<tr>
<td>Initial % of modern individuals</td>
<td>$\pi_1 = 0.462%$</td>
</tr>
</tbody>
</table>

*Note:* In addition to these parameters, the initial age distribution, survival probabilities in 1751 and 2017, and time series of TFP and CBR are taken directly from the data. See Figures 7 and 8.

![Crude death rate in Sweden, model and data](image)

*Source:* Palgrave Macmillan Ltd (2013) and authors’ calculations
epidemic. Our model does not have an exogenous shock during these specific years to account for the spikes in mortality. Finally, the model’s ability to fit the time series of CDR implies that it also fits well the growth rate of population in Sweden. The model-data correlation is 78%.

**Non-targeted moments** Panel A and B of Figure 10 compare the age distribution in the model to that in the data in 1850 and 1950. Recall that the age distribution in 1751 is exogenous, but the age distributions after 1751 are endogenous. The evolution of the age distribution is governed by births and age-specific mortality rates. The latter is the result of individual choices in the model, summarized by Equation (14). The fact that the model matches the evolution of the age distribution over time indicates that the role of migration, which is not accounted for in the model, is minor. The model is also consistent with the timing of mortality decline despite the absence of economic takeoff; see Panel C of Figure 10. CDR declined from 26 in 1751 to 18 in 1860; Jörberg (1965) notes that the Industrial Revolution occurred in Sweden in the 1870s. (See Delventhal et al. (2021) for the observed decline in CDR for more countries.)

### 4.3 Western Europe: 19th and 20th centuries

We test the model for a set of Western European countries. For country \(i\) we use CBR from Palgrave Macmillan Ltd (2013) and GDP from the Maddison Project Database as exogenous variables. The initial value for \(w_t\) is scaled to match country \(i\)'s GDP in year \(t_i\) (the first year for which data are available for country \(i\)) relative to that of Sweden in year \(t_i\). We choose a sequence of growth rates for \(w_t\) to best reproduce (in a least-squares sense) country \(i\)'s GDP. We use data from the Human Mortality Database for the initial age distribution of country \(i\) in year \(t_i\).

<table>
<thead>
<tr>
<th>Country</th>
<th>Model-data correlation of CDR(%)</th>
<th>Population increase accounted for by model (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>France (1816-2010)</td>
<td>96</td>
<td>96</td>
</tr>
<tr>
<td>Norway (1846-2010)</td>
<td>97</td>
<td>89</td>
</tr>
<tr>
<td>Switzerland (1876-2010)</td>
<td>89</td>
<td>97</td>
</tr>
<tr>
<td>Denmark (1835-2010)</td>
<td>97</td>
<td>97</td>
</tr>
</tbody>
</table>

*Note:* The preference and technology parameters are the same as in Table 1. The survival probabilities are the same as in Sweden (Panel A of Figure 8). The initial age distribution and time series of TFP and CBR are country specific, and taken directly from the data.

*Source:* Palgrave Macmillan Ltd (2013) and authors’ calculations.

Preference and technology parameters are the same as those in Table 1. We calibrate only one time-invariant parameter, namely the initial proportion of modern individuals, to fit the entire time series of country \(i\)'s CDR. In the case of France, for instance, this means that we use one parameter to
Figure 10: Age distribution, crude death rate, and total factor productivity in Sweden, model and data

Source: Authors’ calculations, Human Mortality Database (University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany); available at www.mortality.org or www.humanmortality.de.), Krantz (2017) and Schön and Krantz (2017).

match a time series of 195 observations from 1816 to 2010. Table 2 reports the model’s performance for France, Norway, Switzerland and Denmark. In Appendix D we report the entire time series of CDR for these four countries.

4.4 Mortality and population dynamics across countries: 1960-2019

We use the calibrated model to simulate time series of CDR and population from 1960 to 2019 for each one of the 87 countries in our sample. A country is identified by (i) its initial (1960) age distribution, which we take from data, (ii) its CBR path from 1960 to 2019, which we take from data, (iii) its GDP path from 1960 to 2019 ($w_t$), which we take from data, and (iv) its initial (1960) proportion of modern
individuals, $\pi_1$. We calibrate $\pi_1$ for each country to fit the time series of the country’s CDR from 1960 to 2019 in a least-squares sense. Compared with the calibration of Section 4.2, we use only one parameter to match a country’s entire CDR time series, taking as given the preference and technology parameters in Table 1.

Our calibration exhibits a positive cross-country correlation (81%) in 1960 between GDP and $\pi_1$. Thus, poor countries in 1960 tend to have low initial proportions of modern individuals. Since the CDR-GDP correlation is negative in the data in 1960, the correlation between CDR and $\pi_1$ is also negative in 1960 ($-93\%$). Thus, countries with high mortality in 1960 tend to have low initial proportions of modern individuals. It is important to note $\pi_1$ influences the entire path of CDR from 1960 to 2019, not just the 1960 CDR (see Figure 6).

4.4.1 Mortality

The CDR time series implied by the model for each country reproduces the data closely: The model-data correlation is 90.5% (Panel A of Figure 11). Recall that CDR depends upon the age distribution and age-specific mortality rates (Equation 15). The model fits the data well along both dimensions. The model-data correlation between the proportion of individuals by age in each country-year is 98.6% (Panel B of Figure 11). We also examine the implications of the model for LEB (which depends only on age-specific mortality rates): The model-data correlation is 95.3% (Panel C of Figure 11).

Convergence Panel A of Figure 12 shows the poor-to-rich ratio of CDR in the model and data. The model accounts for almost all of the convergence: The model-data correlation of the poor-to-rich CDR ratio is 94.3%.

Similarly, LEB in poor countries was 60% of the LEB in the rich in 1960; by 2019 it is more than 85%. Model-generated statistics exhibit the same qualitative pattern. The model-data correlation of the poor-to-rich ratio of LEB is 84.1% (see Panel A of Figure 13).

As noted in Section 2.3, the cross-country convergence of mortality occurs despite the absence of cross-country convergence in GDP. This feature is readily seen in Panel B, which shows the cross-country elasticity of CDR with respect to GDP in the model and data. The elasticity converges toward zero; the model-data correlation is 96.1%. For LEB, the model-data correlation of the GDP elasticity is 84.5% (see Panel B of Figure 13).

In Section 2.4 we noted that the decline in age 0-4 mortality rate in poor countries accounts for almost all of the LEB convergence. A natural question then is whether the model replicates the age 0-4 mortality rates in poor countries. Figure 14 illustrates the age 0-4 mortality rates in poor countries for model and data in five-year intervals from 1960 to 2015 (204 observations). The model-data correlation is 82%.

Recall from Section 3.3 that the S-shaped diffusion curve is country specific, indexed by the initial TFP.
and initial proportion of modern individuals (see Figure 6). Poor countries are on a lower diffusion curve, so their mortality in 1960 is higher than that of rich countries. However, their diffusion curve is steeper, so over time their mortality declines faster and converges to that of the rich. It is important to note that, even though convergence is bound to occur in our model, its timing is not bound to align with the data. The timing of convergence, that is the fact that poor countries reach the steep part of their diffusion while the rich reach the flat part of theirs, results from the choice of only one parameter for each country: the initial proportion of modern individuals. Recall that the calibrated technology and preference parameters are common to all countries.

Is it possible that the mortality convergence occurred because poor countries are investing more in health relative to rich countries? The answer is no. Figure 15 illustrates country-year observations on share of Gross Domestic Product devoted to health expenditures (including external aid) and GDP. Rich countries allocate a higher share relative to poor countries. Between 2000 and 2018, for instance,
A – Poor-to-rich ratio of CDR

B – GDP elasticity of CDR

Figure 12: Convergence in crude death rate

Note: Panel A shows the ratio of CDR in poor countries to that of the rich. Panel B shows the cross-country GDP elasticity of CDR.
Source: World Bank and authors’ calculations.

A – Poor-to-rich ratio of LEB

B – GDP elasticity of LEB

Figure 13: Convergence in life expectancy at birth

Note: Panel A shows the ratio of LEB in poor countries to that of the rich. Panel B shows the cross-country GDP elasticity of LEB.
Source: World Bank and authors’ calculations.
Figure 14: Age 0-4 mortality rates in poor countries, model and data

*Source:* United Nations and authors’ calculations.

Figure 15: Health expenditures versus gross domestic product per capita

*Note:* The figure shows current health expenditures (% of gross domestic product) vs. gross domestic product per capita. Each point represents a country-year.

*Source:* World Bank.
Madagascar’s share was only half that of France but its CDR declined from 10 to 6 whereas France’s CDR hardly changed. Preston (1980) points out that international financial aid for health purposes is a small fraction of poor countries’ health expenditures and does not explain the decline in mortality in poor countries.

**Mortality miracles** Section 2.5 points out that economic development is not necessary for mortality reduction. The southwest quadrant of Panel A of Figure 5 illustrates that there are countries with declining GDP that experience declining CDR. There is not a single country that experiences an increase in CDR and a decrease in GDP (northwest quadrant). Our model replicates these facts. Table 3 lists the countries with declining GDP.

<table>
<thead>
<tr>
<th></th>
<th>Growth rate of GDP</th>
<th>Change in CDR data</th>
<th>Change in CDR model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burundi</td>
<td>−0.04</td>
<td>−15.5</td>
<td>−7.2</td>
</tr>
<tr>
<td>Central African Republic</td>
<td>−0.74</td>
<td>−16.2</td>
<td>−7.0</td>
</tr>
<tr>
<td>Congo (Kinshasa)</td>
<td>−1.50</td>
<td>−13.5</td>
<td>−7.5</td>
</tr>
<tr>
<td>Haiti</td>
<td>−0.32</td>
<td>−14.2</td>
<td>−9.5</td>
</tr>
<tr>
<td>Madagascar</td>
<td>−0.85</td>
<td>−18.9</td>
<td>−9.4</td>
</tr>
<tr>
<td>Niger</td>
<td>−0.64</td>
<td>−20.9</td>
<td>−7.6</td>
</tr>
</tbody>
</table>

*Note:* The table presents the annual growth rate of GDP (percent) and the change in CDR (deaths per 1,000 persons) from 1960 to 2019 in both data and model.  
*Source:* World Bank and authors’ calculations.

Technology diffusion occurs in all countries even though GDP may decline. This is because of the dynamic externality (see Equations 6-7). Recall that the cost of adoption decreases as the proportion of modern individuals increases. The lower cost of adoption induces more obsolete individuals to spend resources to transition to becoming modern, thereby reducing the adoption cost further. This mechanism operates even if GDP is constant. A decreasing GDP is, of course, a force toward a higher cost of adoption and may offset the dynamic externality. Whether it does is a quantitative question. In our experiment it turns out that the dynamic externality dominates.

Conversely, the northeast quadrant of Panel A of Figure 5 illustrates that there are countries with increasing GDP and increasing CDR. This further suggests that GDP growth does not guarantee CDR reduction.

It is worth noting that LEB increases in all countries in the model as in the data. How does the model explain LEB increase in countries experiencing an increase in CDR (northeast quadrant of Panel A of Figure 5)? As noted in Section 2.5, the increase in CDR in these countries is due to an increasing proportion of older individuals, while the increase in LEB is due to declining age-specific mortality rates. Our model is consistent with these features of the data. In Japan, for example, CDR increased
by 9 in the model and LEB increased by 5 years between 1960 and 2019; the age-0 mortality rate declined from 0.02 to 0.004 and the age-65 mortality rate declined from 0.012 to 0.010. Recall that age-specific mortality rates are governed by the proportion of modern individuals in each age group, which increases as the modern technology diffuses. The increase in LEB follows from the decline in age-specific mortality rates (see Equation 16). The increase in CDR depends, in addition, on the age distribution (Equation 15), which is a function of CBR. During the period 1960-2019, Japan’s CBR decreased by 1.4% per year while the average decline among the 87 countries in our sample was 1%. As a result, the proportion of people 65 and above increased more than the average.

4.4.2 Population dynamics

![Figure 16: Growth rate of population, model and data](image)

*Note*: The figure plots the rate of natural increase in both model and data.

We compute model-generated time series of population as described in Equation (1): We assign to each country its observed population in 1960, its observed CBR and its model-implied CDR. We construct the data counterpart of population in the same manner using the observed CBR and CDR. (As noted in Section 2.2, the role of migration for the increase in population in our sample is minor.) Figure 16 shows the model fit for population growth by country-year. The correlation is 98%.

The model-generated population matches almost all of the increase in population between 1960 and 2019, as well as the distribution of population growth across countries. The increase in total population implied by the model is 3.07 billion whereas it is 3.11 billion in the data. Applying decomposition (2) we find that 49.3% of population growth takes place in the poor countries (vs. 49% in the data) and 4.3% takes place in rich countries (vs. 4.8% in the data).
4.5 The world: 19th and 20th centuries

We use our model to generate a time series of world population from 1800 to 2010. (The length of the time series is constrained by the data available from gapminder.org.) We proceed as follows. The preference and technology parameters are the same as those in Table 1. The exogenous time-varying variables we feed into the model are world CBR (Figure 1) and GDP from Maddison’s Project Database. The initial value for $w$ is scaled to match world GDP in 1800 relative to that of Sweden. The sequence of growth rates for $w_t$ that best reproduces (in a least-squares sense) world GDP are 1% prior to 1950 and 2.4% after. We use world population in 1800 as the initial population size. Finally, the two technologies—obsolete and modern—are the same as Sweden’s in 1800 and 2017, respectively, and Sweden’s age distribution in 1800 is the initial age distribution (we do not have age-distribution data for world population).

We find two time-invariant parameters to fit the time series of world CDR from 1800 to 2010 in a least-squares sense (211 observations). They are: the initial proportion of modern individuals, $\pi_1$, and a factor multiplying the sequence of age-specific survival probabilities of the obsolete technology.\footnote{World CDR in 1800 is abnormally high at 37. Even with 100% of obsolete individuals in 1800, the model cannot generate a CDR of this magnitude. In other words, the world’s obsolete technology in 1800 was worse than that of Sweden in 1800.}

We find $\pi_1 = 0.08\%$ and the factor multiplying the survival probabilities of the obsolete technology is 0.99. Figure 17 shows the model fit: The correlation is 98.5%.

Acceleration of world population Table 4 illustrates the acceleration of world population since 1800. In both the model and data it took 82 years for world population to increase from 1 billion to 1.5 billion and only 38 years for the next half billion. It took 8 years in the data for world population to increase from 3 to 3.5 billion while it took 9 years in the model. The model replicates the accelerating population despite a declining CBR due to the reduction in CDR as illustrated in Figure 17.

5 Conclusion

We developed a theory of population dynamics based on individual choice. In our theory, individuals decide to incur goods and/or time costs to adopt a health technology to reduce their mortality rates. Technology adoption creates a knowledge diffusion across individuals, which leads to further adoptions. Population dynamics are the result of the endogenous evolution of age-specific mortality rates and age distribution.

Our theory delivers mortality reduction and population growth that are consistent with the observed (i) acceleration in population despite decreasing birth rates, (ii) cross-country convergence in mortality despite lack of convergence in income, (iii) mortality miracles, i.e., reduction in mortality in countries with declining income, (iv) substantial contribution of poor countries to world population growth, (v)
Figure 17: World crude death rate, model and data

*Source:* https://gapminder.org/ and authors’ calculations.

Table 4: World population growth, model and data

<table>
<thead>
<tr>
<th>Population change (bil.)</th>
<th>Data</th>
<th></th>
<th></th>
<th>Data</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Annual growth</td>
<td>years</td>
<td>Annual growth</td>
<td>years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.0 to 1.5</td>
<td>0.49%</td>
<td>82</td>
<td>0.49%</td>
<td>82</td>
<td>0.49%</td>
<td></td>
</tr>
<tr>
<td>1.5 to 2.0</td>
<td>0.76%</td>
<td>38</td>
<td>0.76%</td>
<td>38</td>
<td>0.76%</td>
<td></td>
</tr>
<tr>
<td>2.0 to 2.5</td>
<td>0.93%</td>
<td>24</td>
<td>1.01%</td>
<td>22</td>
<td>1.01%</td>
<td></td>
</tr>
<tr>
<td>2.5 to 3.0</td>
<td>1.66%</td>
<td>11</td>
<td>1.52%</td>
<td>12</td>
<td>1.52%</td>
<td></td>
</tr>
<tr>
<td>3.0 to 3.5</td>
<td>1.93%</td>
<td>8</td>
<td>1.93%</td>
<td>8</td>
<td>1.93%</td>
<td></td>
</tr>
</tbody>
</table>

*Source:* https://gapminder.org/ and authors’ calculations.
reduction in age 0-4 mortality rate that is critical to increase in life expectancy in poor countries, and
(vi) gradual reduction in mortality in Western Europe during the 19th and 20th centuries.

In our model of individual choice, the decline in mortality has value. An implication of the increase
in life expectancy is that welfare is better measured by lifetime income instead of income. Since life
expectancy in poor countries is converging to that of the rich, it is conceivable that the lifetime income
is converging even though income is not. Jones and Klenow (2016) go beyond income and measure
well-being differences across countries using consumption, leisure, mortality, and inequality. They
argue that mortality plays the most important role in accounting for the well-being differences.

Our theory of population based on mortality is complementary to theories of population based on
fertility. We treated fertility as exogenous in our model. A complete theory of population would
have both mortality and fertility be endogenous. With endogenous fertility in our model, the role of
technology diffusion for population growth could be stronger. If individuals have preferences over the
number of surviving children, as in Eckstein, Mira and Wolpin (1999), Doepke (2005) and Bar and
Leukhina (2010), then fertility directly depends on mortality. In such a model, technology diffusion
that reduces the age 0-4 mortality also has the potential to reduce fertility and account for the declining
trends in both CBR and CDR.
References


A Population statistics

The population statistics we discuss in this appendix are the crude death rate (CDR) and life expectancy at birth (LEB). For the sake of simplicity we consider a stationary population (i.e., a stationary age distribution). Let $p_j$ denote the size of the age-$j$ population, and let $p = \sum_j p_j$ denote total population. Let $q_j$ denote the age-specific mortality rate, i.e., the probability of death between age $j$ and age $j+1$. The corresponding survival probability is $s_j = 1 - q_j$. Recall that $s_J = 0$. It is convenient to define the probability of surviving from age 0 to $j$ as

$$S_j \equiv \prod_{k=0}^{j-1} s_j.$$ 

Note that $S_{j+1} = S_j s_j$, that is, the probability of surviving to age $j+1$ is the probability of surviving to age $j$ times the probability of surviving from $j$ to $j+1$.

CDR, that is the number of deaths per person, is

$$\text{CDR} = \frac{1}{p} \sum_j p_j q_j.$$ 

Note that CDR is a function of the age distribution ($p_j/p$) and age-specific mortality rates ($q_j$).

LEB is the expected years of life for a person just born:

$$\text{LEB} = \sum_{j=1}^{J} j(1 - s_j) \prod_{k=0}^{j-1} s_j,$$

$$= \sum_{j=1}^{J} j(1 - s_j) S_j. \tag{A.1}$$

Equation (A.1) contains only survival probabilities, so LEB does not depend on the age distribution. Note that since $S_{j+1} = S_j s_j$,

$$\text{LEB} = \sum_{j=1}^{J} jS_j - \sum_{j=1}^{J} jS_{j+1},$$

$$= S_1 + \sum_{j=2}^{J} jS_j - \sum_{j=1}^{J} jS_{j+1},$$

$$= S_1 + \sum_{j=1}^{J-1} (j + 1)S_{j+1} - \sum_{j=1}^{J-1} jS_{j+1},$$

$$= S_1 + \sum_{j=1}^{J-1} (j + 1)S_{j+1} - \sum_{j=1}^{J-1} jS_{j+1},$$
where the last line follows from $s_j = 0$ so that $S_{J+1} = 0$. It follows that

$$\text{LEB} = \sum_{j=1}^{J} S_j.$$  \hfill (A.2)

**Elasticity of LEB with respect to age-specific survival probability**

It follows from Equation (A.2) that

$$\frac{\partial \text{LEB}}{\partial s_i} = \sum_{j=1}^{J} \frac{\partial S_j}{\partial s_i}$$

where

$$\frac{\partial S_j}{\partial s_i} = \begin{cases} 0 & \text{if } i < j - 1, \\ \frac{S_j}{s_i} & \text{if } i \geq j - 1. \end{cases}$$

Thus,

$$\frac{\partial \text{LEB}}{\partial s_i} = \frac{1}{s_i} \sum_{j=i+1}^{J} S_j = \frac{1}{s_i} \left( \text{LEB} - \sum_{j=1}^{i} S_j \right).$$

Finally, the elasticity of LEB with respect to $s_i$ is

$$\epsilon_i \equiv \frac{\partial \text{LEB}/\text{LEB}}{\partial s_i/s_i} = 1 - \frac{1}{\text{LEB}} \sum_{j=1}^{i} S_j.$$  \hfill (A.3)

Note that $\epsilon_i$ is monotonically decreasing in $i$: An increase in young-age mortality has a larger effect than an increase in old-age mortality. Note also that $\epsilon_i \in [0, 1)$ and $\epsilon_J = 0$. 


B例 Example of life-saving practices

The information below is available from the Centers for Disease Control and Prevention, the North Dakota Department of Health, and the Kentucky Department of Public Health.

AIDS

AIDS is caused by the human immunodeficiency virus (HIV). The spread of the virus can be prevented by abstinence or using condoms.

Cholera

Cholera is a disease that one contracts by drinking water or eating food contaminated with the bacteria Vibrio Cholerae. Casual contact between people does not spread cholera. One can reduce the risk of contracting cholera by drinking only clean (bottled, boiled, or chemically treated) water, washing hands often, using clean water to wash dishes, brush teeth, wash and prepare food, and make ice and, finally, dispose of feces in a manner that prevents contamination of water and food.

Ebola

The ebola virus disease (EVD) spreads to people through direct contact with the blood, bodily fluids, and tissues of an infected animal. The virus then spreads from person to person through direct contact with blood or bodily fluids, and contaminated objects.

To reduce the risk of contracting EVD, one should: avoid contact with blood and bodily fluids of sick people and people who died from EVD; avoid contact with contaminated objects; avoid funeral or burial practices that involve touching the body of someone who died from EVD; avoid contact with bats, forest antelopes, and nonhuman primates blood, fluids, or raw meat prepared from these or unknown animals (bushmeat).

The plague (Yersinia pestis)

The plague bacteria can be transmitted to humans via flea bites, contact with contaminated fluid or tissue and infectious droplets. Transmission of these droplets is the only way that plague can spread between people. One can reduce the risk of becoming infected by limiting rodent habitat around homes, work places, and recreational areas. One should wear gloves when handling infected animals, use repellent and keep fleas off of pets. Wearing a mask also reduces the risk of transmitting and/or contracting Yersinia pestis.

Polio (Poliomyelitis)

Polio is caused by the Poliovirus which is found in the stool and throat of infected people. Besides the vaccine, one can prevent the spread of polio by washing hands with soap and water after using the bathroom and changing diapers, and before preparing food and eating.
Consider the first-order conditions (6)-(7), abstracting from time and generation subscripts for simplicity,

\[ 0 = U_1 (w (1 - h) - y) - \beta A_1 (y, h, \pi) \Delta \]
\[ 0 = U_1 (w (1 - h) - y) w - \beta A_2 (y, h, \pi) \Delta. \]

Given the functional form for \( A \) (Section 4), these conditions imply

\[ 0 = U_1 (w (1 - h) - y) - \beta \pi \lambda \exp (-\lambda \chi (y, h)) \chi_1 (y, h) \Delta \]
\[ 0 = U_1 (w (1 - h) - y) w - \beta \pi \lambda \exp (-\lambda \chi (y, h)) \chi_2 (y, h) \Delta \]

where \( \chi (y, h) = (\alpha_y y^\theta + \alpha_h h^\theta)^{1/\theta} \). This implies \( 1/w = \chi_1 (y, h)/\chi_2 (y, h) \), where \( \chi_1 (y, h) = \chi (y, h)^{1-\theta} \alpha_y y^{\theta-1} \) and \( \chi_2 (y, h) = \chi (y, h)^{1-\theta} \alpha_h h^{\theta-1} \). Hence, \( h = y X (w) \) where,

\[ X (w) = \left( w \frac{\alpha_y}{\alpha_h} \right)^{1/(\theta-1)}. \]

Note that, at the optimum,

\[ \chi (y, h) = y \left( \alpha_y + \alpha_h X (w)^\theta \right)^{1/\theta} \]

and

\[ \chi_1 (y, h) = \left( \alpha_y + \alpha_h X (w)^\theta \right)^{1/(\theta-1)} \alpha_y \equiv \chi_1 (w), \]

where the identity, abusing notations, indicates that the first-derivative of \( \chi \) with respect to \( y \) is a function of \( w \) only at the optimum. Consumption is \( c = w - y (1 + w X (w)) \). Given the functional form for \( U \) (Section 4), the first-order condition for \( y \) is

\[ \sigma \exp (-\sigma w + \sigma y (1 + w X (w))) = \beta \Delta \pi \lambda \exp \left( -\lambda y \left( \alpha_y + \alpha_h X (w)^\theta \right)^{1/\theta} \right) \chi_1 (w) \]

or

\[ y \left[ \sigma (1 + w X (w)) + \lambda \left( \alpha_y + \alpha_h X (w)^\theta \right)^{1/\theta} \right] = \sigma w + \ln \left( \frac{\beta \Delta \pi \lambda \chi_1 (w)}{\sigma} \right). \]
### Figure D.1: Crude death rates in Europe, model and data

Source: Palgrave Macmillan Ltd (2013) and authors’ calculations.