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Technology Adoption, Mortality, and Population Dynamics*

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Abstract

We develop a quantitative theory of mortality and population dynamics. We emphasize individuals' decisions to reduce their mortality by adopting better health technology. Adoption confers a dynamic externality: Adoption becomes cheaper as more individuals use better technology. Our model generates a diffusion curve, whose shape dictates the pace of mortality reduction. The model explains historical trends in mortality rates and life expectancies at various ages, and population dynamics in Western Europe. Unlike Malthusian theories based solely on income, ours is consistent with the observed disconnect between mortality and income. Unlike Beckerian theories based solely on fertility, ours accounts for the observed acceleration in population.

JEL codes: I12, I15, J11, E13
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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 linking Malthus to modern-growth models à la Lucas (1988) and Romer (1986). Becker, Murphy, and Tamura (1990), Galor and Weil (1999), and Tamura (2002) provide such a link via a theory of fertility and human capital.

Theories of fertility, such as Becker (1960), Becker and Barro (1988), and Barro and Becker (1989), explain the secular *decrease* in the world crude birth rate (CBR), from 4% before 1850 to 3% after 1950, but cannot explain the *increase* in the growth rate of world population during the same period, from 0.5% to 1.8%.[1] (The growth rate of world population is CBR − CDR (crude death rate).) Thus, a theory of population dynamics requires a theory of mortality reduction.

What we mean by a "theory" of population dynamics is a model that is consistent not only with the time series of CDR but also with the dynamics of age distribution and life expectancies at all ages.[2] For such a theory, a key endogenous variable is the time series of mortality rate at *every* age, not just univariate CDR.

Our theory is inspired by Mokyr's (1993) approach to mortality: It is based on *individuals*' decisions to adopt health technologies that reduce their mortality risk at each age. The endogenous evolution of age-specific mortality rates determines CDR and population dynamics, in addition to other statistics such as life expectancies at various ages and age composition of the population. We use long-run trends—more

[1] For quantitative models of fertility, see surveys by Doepke and Tertilt (2016) and Greenwood, Guner, and Vandenbroucke (2017). The acceleration in population despite a declining CBR is also observed in recent data.

[2] An aggregate model of the univariate time series of CDR would be unsatisfactory because it would be inconsistent with other mortality statistics such as life expectancies at various ages. For instance, suppose one delivers CDR by endogenizing mortality rates in perpetual-youth models of Yaari (1965) or Blanchard (1985). Such models would imply that the time series of life expectancies at all ages would be identical—see Appendix A.1.

than two centuries of data for 7 European countries—to confront our theory.

The popular view is that mortality decline is mostly due to medical innovations. But Sweden's CDR had already declined by 80 basis points from the mid 18th century to the 1870s. The decline was almost the same over the next 130 years, which saw medical innovations such as Germ Theory, penicillin, and sulfa drugs. Why? Our answer is that individual actions, based on the filth-disease correlation known before the 1870s, helped diffuse the best health practices. This choice-theoretic view is also articulated by Mokyr (1993), Mokyr and Stein (1997), and Mokyr (2000). They argue that the hygienist movement promoted the awareness of the correlation between filth and disease which gave rise to health practices in early 19th century Europe, reducing mortality. Furthermore, McKeown, Brown, and Record (1972), Mokyr and Stein (1997), and Riley (2005a) note that major causes of death had disappeared before effective medical cures and vaccines became widely available.[3]

Our model has two technologies—obsolete and modern—represented by age-specific survival probabilities, with modern technology offering a higher chance of survival at all ages. The modern technology represents the outcome of individual-level practices, such as boiling water, safe handling of milk and food, washing hands, and using soap (Mosley, Jamison, and Henderson, 1990, Table 6), that reduce the risk of dying. We also allow mortality to decline exogenously for reasons other than individual choices.

We refer to individuals using the modern (obsolete) technology as modern (obsolete). Obsolete of any age spend time and/or goods to increase their odds of becoming modern. Cross-sectional heterogeneity is thus limited to age and the proportion of modern by age. Age-specific mortality is a weighted average of mortality rates of the obsolete and modern. These weights evolve endogenously as the technology diffuses.

We assume a dynamic externality: At any point in time, the proportion of modern determines the incentives for the obsolete to adopt the modern technology. As the proportion increases, all else equal, the dynamic externality implies more individuals adopt the modern technology, so mortality declines. The proportion of modern evolves

[3]For medical innovations such as vaccines, the vaccination rate is determined by individual choices. For instance, smallpox vaccinations started in Sweden in 1801, but Lazuka and Jensen (2021) note that the vaccination rate among children age 10 or below was only 60% even after 25 years (see their Figure 2). The uptake was low despite the vaccination being mandatory starting in 1816. One of the reasons noted in Lazuka and Jensen (2021) is "the loss in income due to the absence from work when taking a child to a vaccinator" (p. 9), which points to individual choices.

according to the familiar S-shaped pattern of technology diffusion. With few modern, the dynamic externality is "small" and, hence, the flow of adopters is "small." With few obsolete, the flow of adopters is "small" as well.

Our model's exogenous driving variables are total factor productivity (TFP) and CBR. TFP affects the costs and benefits of adopting the modern technology. Higher TFP raises the time cost and lowers the goods cost; it also raises the benefit of adoption by increasing lifetime income. The only role of CBR is quantitative: It delivers the flow of newborns each period and does not affect individual decisions. The endogenous evolution of age-specific mortality rates, combined with the exogenous flow of newborns, determines the age distribution in the model. Age-specific mortality rates and age distribution together pin down CDR and, hence, population dynamics.

We calibrate the model to a univariate time series of long-run trend in Sweden's child (age 0 to 4) mortality. We use CMR to refer to child mortality. Our proxies for the obsolete and modern technologies are the observed age-specific survival probabilities in 1751 and 2018, respectively. (This is similar to Hansen and Prescott, 2002, where both Malthus and Solow technologies are always available.) The critical parameters to calibrate are the initial proportion of modern and the time-invariant parameters mapping time and goods into the probability of becoming modern.

In line with our notion of a theory of population dynamics, we examine the model's ability to match the time series of Sweden's mortality at different ages, life expectancy at different ages, CDR, and population. Note that CMR is targeted in our calibration, but mortality rates for those above age 4 are neither targets nor mechanically implied by CMR. While mortality rates at all ages decline over time, the evolution is heterogeneous. For instance, (i) a child was twice as likely to die as those 60-64 years of age in the mid 18th century (32% versus 16%); by World War I the mortality rates for the two groups were practically identical. (ii) Until the end of the 19th century, the mortality rate increased with age except for CMR. Our model matches the heterogeneity in age-specific mortality trends, which implies that we deliver the life expectancies at all ages. For instance, life expectancy at age 30 increased by 8 years between the mid 18th century and late 19th century in the model and data. The endogenous evolution of age-specific mortality and the exogenous CBR deliver the time series of age distribution and, hence, CDR.

For almost the entire 19th century, CBR remained constant or declined in Sweden, but the population growth rate increased steadily. Our model delivers this acceleration: CDR declines more than CBR in the model and data. The exogenous channel for mortality decline accounts for 36% of the population growth rate in Sweden.

Income plays a minor role in mortality reduction in our model, as in the data. In a counterfactual where income remains constant in Sweden at its 18th century level, we show that, for more than 125 years, CDR is the same as in the baseline model with income growth. This implication contradicts theories à la Malthus, but is consistent with the historical disconnect between mortality decline and economic takeoff, noted by Livi-Bacci (1991) for Europe from the Middle Ages to the Industrial Revolution and Wrigley, Davies, Oeppen, and Schofield (1997) and Leukhina and Turnovsky (2016) for England in the 18th and 19th centuries. Furthermore, Delventhal, Fernández-Villaverde, and Guner (2021) find that, in 186 countries over a few centuries, real gross domestic product per capita (GDP) does not predict the start of the CDR decline. While the role of income for mortality decline is minor, the GDP-CDR correlation is not zero in the data; without individual choice the mortality-income correlation in the model would be zero.

The model also delivers the long-run trends in CDR, age-specific mortality rates, life expectancies, and population for several European countries, including in France and Denmark. Finally, while world CBR has been declining since the late 19th century, world CDR has been declining more rapidly. The model reproduces the CDR decline and the resulting acceleration in world population.

In related work on Sweden, Eckstein, Mira, and Wolpin (1999) develop a model of fertility dynamics taking age-specific mortality rates as exogenous. Cervellati and Sunde (2015) study a model of economic transition driven by fertility choices via quantity-quality tradeoffs. Theirs is not a theory of CDR or population acceleration, so it is not set up to explain the observed increase in population growth in Sweden despite a constant CBR.

Several remarks about our theory are in order. First, our theory does not have an explicit role for medicine. However, one could argue that modern medicine plays an implicit role because individuals in our model have access to the 2018 survival probabilities. To exclude any role for modern medicine, we represent the modern

technology by 1930 survival rates (before the appearance of penicillin and sulfa drugs) and re-calibrate our model. The re-calibrated model matches the mortality statistics for Sweden before 1930. In particular, the population growth is almost the same in the re-calibrated model and baseline.

Second, our theory does not have large-scale public investments in sanitation. As noted by Mokyr and Stein (1997), explanations of mortality reductions based solely on such programs are not different from explanations based solely on income, which are inconsistent with the historical evidence for the United Kingdom, Germany, and France. Furthermore, most sewers in England were built in the late 19th century (Wohl, 1983), while life expectancy at birth (LEB) increased 25% between 1725 and 1850 (Fogel, 2004, Table 1.1). A similar critique applies to nutrition-based theories.

Third, one might argue that ours is an "incomplete" theory of population since we take CBR as exogenous. However, endogenizing CBR in our model would not yield *additional* target moments for demographic variables. In Appendix D, we add an assumption that parents care about the number of surviving children, as in Eckstein et al. (1999), Doepke (2005), Bar and Leukhina (2010), and Albanesi and Olivetti (2016), where mortality is exogenous. With this addition, we endogenize both CBR and CDR. While this model can be used to confront the data on CBR, it yields no additional moments to confront the data on population growth, age distribution, and life expectancies, relative to our baseline model with exogenous CBR.

We develop the model in Section 2. We present functional forms, calibrate to historical Swedish data, and test the model in Section 3. We also discuss the roles of individual choice (Section 3.4), medicine (Section 3.5), sanitation infrastructure (Section 3.6), and nutrition (Section 3.7) in reducing mortality. In Section 4 we quantitatively examine mortality in Denmark, France, and other Western European countries and the acceleration of the world population. We conclude in Section 5.

2 Model

Our goal is to develop a model that is quantitatively consistent with mortality statistics, such as CDR and life expectancy at all ages, and age distribution across countries and over time. It is useful to recall how CDR and life expectancy are measured. Let

$p_{t,j}$ denote the population of birth-cohort t and age j, so total population at time t is $p_t = \sum_j p_{t-j,j}$. Let $q_{t,j}$ denote the mortality rate of age-j individuals of birth-cohort t, i.e., the probability of a cohort-t person not surviving from age j to $j+1$, conditional on surviving to age j. Thus, total deaths at time t are $\sum_j p_{t-j,j} q_{t-j,j}$. The crude death rate at time t is the total number of deaths per person, so

$$\text{CDR}_t = \frac{\text{Total deaths at time } t}{p_t} = \frac{1}{p_t} \sum_j p_{t-j,j} q_{t-j,j}. \tag{1}$$

Life expectancy at age a at time t is the expected additional years of life for individuals of age a if they experience the death probabilities observed at time t.

$$\text{LE}_{t,a} = \sum_{j=a}^{\infty} (j+1-a) q_{t-(j+1),j+1} \prod_{k=0}^{j-a} (1 - q_{t-(k+a),k+a}). \tag{2}$$

LEB_t is the expected years of life for age-0 individuals at time t: $\text{LEB}_t \equiv \text{LE}_{t,0}$.

Note that CDR depends on age-specific mortality rates and age distribution of population. Life expectancy at every age, on the other hand, depends only on age-specific mortality. Note also that date-t mortality statistics in (1) and (2) depend only on date-t variables in the cross section.

We show in Appendix A that simple aggregate models of univariate CDR or LEB are inconsistent with the data on mortality statistics defined in Equations (1) and (2). What is needed is a model where age-specific mortality evolves over time. We develop a parsimonious model that delivers precisely that.

2.1 Theoretical framework

Time is discrete and indexed by t. The economy is populated by overlapping generations of individuals living up to a maximum age J.

There are two health technologies available at any point in time: "obsolete" and "modern," indexed by $i \in \{o, m\}$. These technologies are age-specific survival probabilities, $\{s_j^i\}_{j=0}^J$, where s_j^i denotes the probability of survival from age j to $j+1$, conditional on surviving to age j, for $i \in \{o, m\}$. We label individuals using the modern technology as "modern" and those using the obsolete technology as "obsolete."

We assume $s_j^m > s_j^o$ for all $j < J$ and $s_J^o = s_J^m = 0$.

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.

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

$$V_{t,j}^m = U(w_{t+j}) + \beta s_j^m V_{t,j+1}^m, \tag{3}$$

where U is the period utility index and $\beta \in (0,1)$ is the subjective discount factor. The obsolete can adopt the modern technology and become modern in the *next period* with probability $A(\cdot)$. For an age $j > k$ member of generation t, the probability is $A(y_{t,j}, h_{t,j}, \pi_{t+j})$, where $y_{t,j}$ is goods, $h_{t,j}$ is time, and π_{t+j} is the proportion of economically active modern at the beginning of period $t+j$. Note that $y_{t,j}$ and $h_{t,j}$ are choices at time $t+j$, while w_{t+j} is the (exogenous) wage rate and π_{t+j} is an *aggregate* proportion at date $t+j$.

We assume that the probability of adoption is increasing in goods and time:

$$\frac{\partial}{\partial y} A(y,h,\pi) > 0 \text{ and } \frac{\partial}{\partial h} A(y,h,\pi) > 0. \tag{4}$$

We also assume

$$\frac{\partial^2}{\partial \pi \partial y} A(y,h,\pi) > 0 \text{ and } \frac{\partial^2}{\partial \pi \partial h} A(y,h,\pi) > 0. \tag{5}$$

That is, the proportion of modern creates a positive externality: With a higher proportion, the marginal effect of time and goods on the probability of adoption increases. This is a "dynamic externality" since a higher proportion of modern at the beginning of date t implies more adoption at t and even more modern at $t+1$.

In our quantitative exercise, we allow for $A(0,0,0) \geq 0$, i.e., the obsolete can become modern without allocating any goods or time. Thus, mortality can decline for reasons such as public health investments, unrelated to individual choices.

The age-j obsolete consume $w_{t+j}(1-h)-y$ and their value is

$$V^o_{t,j} = \max_{y,h} U\left(w_{t+j}(1-h)-y\right) + \beta s^o_j \left[A(y,h,\pi_{t+j})V^m_{t,j+1} + \left(1-A(y,h,\pi_{t+j})\right)V^o_{t,j+1}\right]. \tag{6}$$

Since $s^o_J = s^m_J = 0$, the terminal condition for modern and obsolete individuals is

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

At an interior, the optimal choice of an obsolete individual is characterized by

$$y_{t,j} \quad : \quad 0 = U_1\left(w_{t+j}\left(1-h_{t,j}\right)-y_{t,j}\right) - \beta s^o_j A_1(y_{t,j},h_{t,j},\pi_{t+j})\Delta_{t,j}, \tag{7}$$

$$h_{t,j} \quad : \quad 0 = U_1\left(w_{t+j}\left(1-h_{t,j}\right)-y_{t,j}\right)w_{t+j} - \beta s^o_j A_2(y_{t,j},h_{t,j},\pi_{t+j})\Delta_{t,j}, \tag{8}$$

where $\Delta_{t,j} \equiv V^m_{t,j+1} - V^o_{t,j+1}$. (We allow for corner solutions as well.)

A few remarks are in order at this stage. First, we assume that resources are not expended every period to "operate" the modern technology. None of our results depend on this assumption: Any resources required to operate the modern technology can be easily incorporated into the dynamic program (3), which might affect the magnitude of $V^m_{t,j}$ but not the choices at the margin. Second, resources affect the *probability* of acquiring the modern technology; this is a modeling convenience. The alternative—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—which would be at odds with the data. Third, constant w_t in (7)-(8) does not imply that y and h would be constant over time. The evolution of π affects the individual choices due to the dynamic externality. Finally, an additional year of life increases welfare since individuals enjoy consumption longer (Equation 6), so our model has both quantity and quality of life, as in Becker, Philipson, and Soares (2005). However, unlike Murphy and Topel (2006) where preferences depend upon health status, U in our model is the same for both the obsolete and modern.

2.2 Population dynamics

Let $p^i_{t,j}$, $i = o, m$, denote the population of obsolete and modern, respectively, 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 is $p_{t-j,j}$, and the total population at date t is $p_t = \sum_{j=0}^{J} p_{t-j,j}$. The proportion of economically active modern at the beginning of period t is

$$\pi_t = \frac{\sum_{j=k}^{J} p^m_{t-j,j}}{\sum_{j=k}^{J} p^m_{t-j,j} + \sum_{j=k}^{J} p^o_{t-j,j}}. \tag{9}$$

We make the following simplifying assumptions on the exogenous number of births per person at time t, CBR_t: (i) Birth rates are not age specific, (ii) birth rates are the same for the modern and obsolete, and (iii) children of obsolete are obsolete and children of modern are modern. Assumption (i) is innocuous: Age-specific birth rates do not matter for age distribution, age-specific mortality, CDR, and population dynamics. Assumption (ii) can be relaxed by adding type-specific birth rates, but this would add more parameters without targets or additional insights. The last assumption represents the notion that children acquire the health practices of their parents. The age-0 populations in cohort t are

$$p^i_{t,0} = \text{CBR}_t \sum_{j=0}^{J} p^i_{t-j,j} \qquad \text{for } i = o, m. \tag{10}$$

From age 0 to age $k-1$, the populations evolve according to

$$p^i_{t,j+1} = s^i_j p^i_{t,j} \qquad \text{for } j = 0, \ldots, k-1 \text{ and } i = o, m. \tag{11}$$

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

$$p^o_{t,j+1} = s^o_j p^o_{t,j} \left[1 - A(y_{t,j}, h_{t,j}, \pi_{t+j})\right], \tag{12}$$

$$p^m_{t,j+1} = s^m_j p^m_{t,j} + s^o_j p^o_{t,j} A(y_{t,j}, h_{t,j}, \pi_{t+j}). \tag{13}$$

Equation (12) 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 (13) indicates that a fraction s^m_j of the age-j modern population survives to the next period and a fraction $s^o_j A(y_{t,j}, h_{t,j}, \pi_{t+j})$ of the age-j obsolete population survives and becomes modern in the next period.

Equilibrium Given an initial population at date 1, $\{p^m_{-j,j}, p^o_{-j,j}\}$, a path for the wage rate, $\{w_t\}$, and birth rate, $\{\text{CBR}_t\}$, an equilibrium is an allocation, $\{y_{t,j}, h_{t,j}\}$, and population sizes, $\{p^m_{t,j}, p^o_{t,j}\}$, for $t \geq 1$ and age $j = 1, \ldots, J$, such that

1. $\{y_{t,j}, h_{t,j}\}$ is optimal, i.e., it solves (6) given $\{\pi_t\}$, $\{w_t\}$ and $\{\text{CBR}_t\}$;
2. $\{p^m_{t,j}, p^o_{t,j}\}$ evolves according to (10)-(13);
3. $\{\pi_t\}$ is consistent with individual decisions, i.e., it satisfies (9).

Mortality The age-j mortality rate at date t, that is the fraction of the age-j population that dies between dates t and $t+1$, is

$$q_{t-j,j} \equiv \frac{p^m_{t-j,j}\left(1 - s^m_j\right) + p^o_{t-j,j}\left(1 - s^o_j\right)}{p_{t-j,j}}. \tag{14}$$

Equation (14) is similar to the result in Hansen and Prescott (2002). In their model and in ours, two different technologies are available at each point in time. In their model, there is an endogenous allocation of capital to each technology. In ours, there is an endogenous allocation of individuals at each age.

The evolution of population combined with (14) helps us compute the mortality statistics described in (1) and (2), and mortality rates for subgroups, e.g., CMR.[4]

To summarize, our theory is about the evolution of age-specific mortality rates. We take two health practices—obsolete and modern—as exogenously given and endogenize the proportions of individuals in each age using these two practices. The evolution of these proportions determines the evolution of mortality rates at different ages. The evolution starts with an initial condition: the aggregate proportion of modern. Note that the initial stock of modern, together with assumption (iii) on CBR, has a Darwinian implication: Even in the absence of any flow of adopters, the

[4] Cervellati and Sunde (2015) consider only two mortality statistics for Sweden, CMR and LE_5, both of which are *assumed to be functions of past economy-wide schooling and per-capita income.* By construction, theirs is not a theory of CDR or population acceleration. Furthermore, the evidence on schooling and income as the main determinants of mortality decline is weak: De La Croix, Lindh, and Malmberg (2008) note that it is the changes in longevity that affect the observed rise in schooling in Sweden and Delventhal et al. (2021) show that income does not predict mortality. In our model, individual decisions result in different mortality rates for different age groups and different life expectancies conditional on age. Our model is consistent with long-run trends in population growth, age composition of the population, CDR, and life expectancies and mortality rates at various ages.

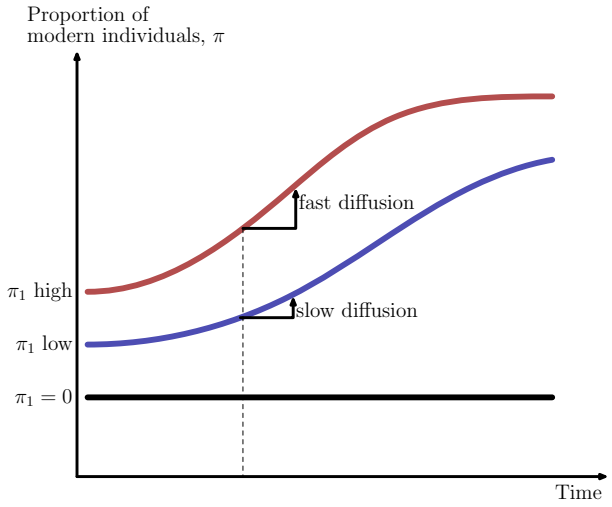


Figure 1: The diffusion of the modern technology

modern would dominate the population. In Equations (12) and (13), if $A \equiv 0$ then

$$\frac{p^m_{t,j+1}}{p^o_{t,j+1}} = \frac{p^m_{t,j}}{p^o_{t,j}} \frac{s^m_j}{s^o_j} > \frac{p^m_{t,j}}{p^o_{t,j}}.$$

2.3 Mortality-Income disconnect

In Section 1 we noted the historical disconnect between mortality and income. To understand how the model generates the disconnect, let $A(y, h, \pi) \equiv \pi G(y, h)$ for illustrative purposes. The flow of age-j obsolete to the modern technology in period t is $\pi_t G(y_{t-j,j}, h_{t-j,j}) p^o_{t-j,j}$. Adoption exhibits an S-shaped pattern: When the proportion of modern is close to 0, the flow of adopters is "small." When the proportion is close to 1, there are few obsolete, so the flow of adopters is "small" as well.

Consider three countries identical in all respects, except for the initial proportion of modern, π_1, which is zero, low, or high. Figure 1 represents the corresponding stylized diffusion curves. If $\pi_1 = 0$, there are no modern, so the modern technology does not diffuse and mortality is both high and constant over time. For low and positive π_1, initial mortality is lower. The dynamic externality incentivizes the adoption of the modern technology and, hence, mortality decreases. For high and positive π_1, initial mortality is further reduced and the incentives to adopt are further increased because

of the dynamic externality. Thus, the diffusion curves are not only higher for higher values of π_1, they are also *steeper*. In sum, differences in π_1 yield differences in both the level *and* rate of change of the proportion of modern, and, therefore, differences in the level and rate of mortality decline regardless of income.

Although differences in π_1 (or exogenous flow of adopters) can generate differences in mortality dynamics independently of income, the CDR-GDP correlation is not zero in the data. To understand the role of income on mortality, consider two countries identical in all respects, except for the initial income, w_1. A lower w_1 has three effects on adoption. First, the present value of income/consumption is lower, so incentives for increasing lifespan are lower. Second, the marginal utility of consumption is higher, therefore diverting goods away from consumption is costlier. Finally, the cost of allocating time is lower. The first two effects imply fewer adoptions, while the third effect implies more. If the sum of all three effects is fewer adoptions, then the country with lower w_1 experiences higher mortality. In addition to the level effects of w, the benefit of a longer life is higher when w grows faster.

3 Quantitative analysis: Sweden

We present functional forms and calibrate the model to Swedish historical data. We then study the quantitative implications for mortality statistics in Sweden.

3.1 Functional forms

The probability of adoption of the modern technology is

$$A(y,h,\pi) = \kappa + \pi\Lambda\left[1 - e^{-\left(\alpha_y y^\theta + \alpha_h h^\theta\right)^{1/\theta}}\right], \tag{15}$$

where $\Lambda, \alpha_y, \alpha_h > 0$ and $\theta \leq 1$. This function satisfies (4), (5), and $A(0,0,0) = \kappa \geq 0$.

The parameter κ is the per-period flow of obsolete who become modern without any individual actions. The flow κ could represent, for instance, biological mutations or public health investments that make individuals resilient to infections and diseases, and survive longer.

Note that even though both the exogenous and endogenous components of $A(y, h, \pi)$ are flows, they have a compounding effect. As summarized in Section 2.2, a one-time increase in $A(y, h, \pi)$ increases the proportion of modern forever and, given the higher survival probabilities, the modern would dominate the population.

Recall that the proportion of modern is an endogenous state variable, so we have to specify an initial condition. The initial value, π_1, is partly the result of past individual actions. As we noted in Section 2.3, π_1 matters for both the level and rate of decline of mortality: A high value of π_1 implies "low- and fast-declining" mortality, while a low value implies "high- and slow-declining" mortality. To fit cases of high- and fast-declining mortality, Λ can be adjusted. This is because Λ affects the adoption probability and, hence, the rate of decline of mortality, but not its initial level.

We use a utility index from the CARA family to represent preferences:

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

The form for U, together with the CES specification in Equation (15), yields an analytical solution to the individual's problem (Appendix B) and reduces the computational cost of fitting the model to the data. (We have to track the population size and proportion of modern at each age, every period.) We check in our computations that U is always positive. As noted by Rosen (1988), this is an important restriction: When U is negative, an extra year of life reduces utility. Hall and Jones (2007) also use a functional form with an additive constant to account for the increase in the share of health expenditures in post-World War II U.S. data. Córdoba and Ripoll (2017) use Epstein-Zin preferences and exogenous mortality to model the value of life.

3.2 Calibration

The model period is 1 year. Age-specific survival probabilities for ages 0 to 110 for Sweden are available from the Human Mortality Database since 1751. In the data $s_{110} = 0$, so $J = 110$. The obsolete and modern technologies are the age-specific survival probabilities in 1751 and 2018, respectively (Panel A of Figure 2).

We set the discount factor β to 0.95. We consider the economy from date $t = 1, \ldots, T$, where date 1 corresponds to 1755 and date T to 2015. The age distribution of

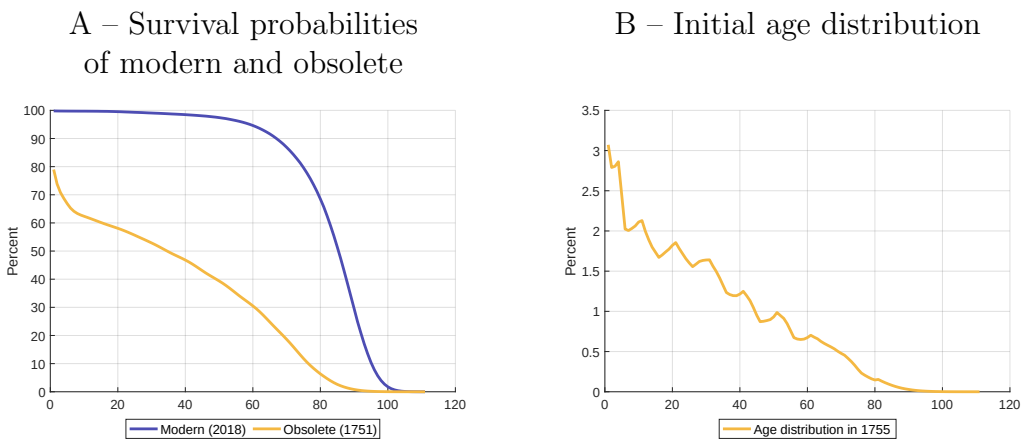


Figure 2: Survival probabilities and initial age distribution in Sweden

Note: Panel A shows the probabilities of survival to each age j, $\prod_{k=0}^{j-1} s_k$, in 1751 (obsolete) and 2018 (modern). The initial age distribution in Panel B is Sweden's age distribution in 1755.
Source: Human Mortality Database (2022).

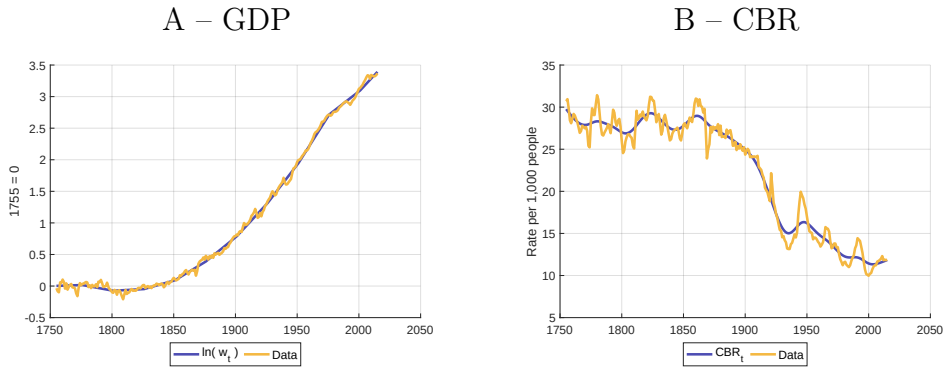


Figure 3: Exogenous variables: gross domestic product per capita and crude birth rate in Sweden, 1755-2015

Source: Krantz (2017) and Schön and Krantz (2017).

population at date 1 is that of Sweden in 1755 (Panel B of Figure 2). We set the economically active age, k, equal to 15.

Both population and w are normalized to 1 at date 1. For w_t, we use the trend component of Sweden's GDP, which we represent with a sequence of constant growth rates changing every 25 years. For CBR_t, we use the HP-trend component of Sweden's CBR. Figure 3 shows Sweden's GDP, CBR, and their trends.

We calibrate the model to CMR data for 1755-2015. Let $\omega = (\sigma, \Sigma, \alpha_y, \alpha_h, \theta, \Lambda, \kappa, \pi_1)$ denote the list of parameters to be calibrated. We determine ω as the solution to the distance-minimization problem:

$$\min_{\omega} \frac{1}{\sqrt{T}} \sum_{t=1}^{T} \left[\text{CMR}_t(\omega) - \text{CMR}_t^{\text{data}}\right]^2, \quad \text{where} \quad \text{CMR}_t = 1 - \prod_{j=0}^{4}(1 - q_{t-j,j}).$$

Recall that CMR_t is child mortality at date t, i.e., the probability that a newborn does not survive past age 4, given the age-specific mortality rates at date t in the cross section. We compute $\text{CMR}_t^{\text{data}}$ with Swedish mortality rates and its model counterpart, $\text{CMR}_t(\omega)$, using the mortality rates implied by the model (Equation 14). In sum, we use 8 parameters to target a time series of $T = 261$ observations.

Table 1: Model parameters

Exogenous	$\beta = 0.950$, $k = 15$, $J = 110$
Calibrated	
Preferences	$\sigma = 6.487$, $\Sigma = 0.111$
Technology	$\alpha_y = 0.052$, $\alpha_h = 0.323$,
	$\theta = 0.230$, $\Lambda = 3.331$, $\kappa = 0.07\%$
Initial modern	$\pi_1 = 1.79\%$

Note: In addition to these parameters, the initial age distribution, survival probabilities of modern and obsolete, and time series of GDP and CBR are taken from the data—see Figures 2 and 3.

Table 1 reports the calibrated parameters. Specifically, the initial proportion of modern is $\pi_1 = 1.79\%$, and the exogenous flow from obsolete to modern in each period is $\kappa = 0.07\%$. Even though the value of κ seems "small," it has a compounding effect. We examine the quantitative role of κ in Section 3.4.

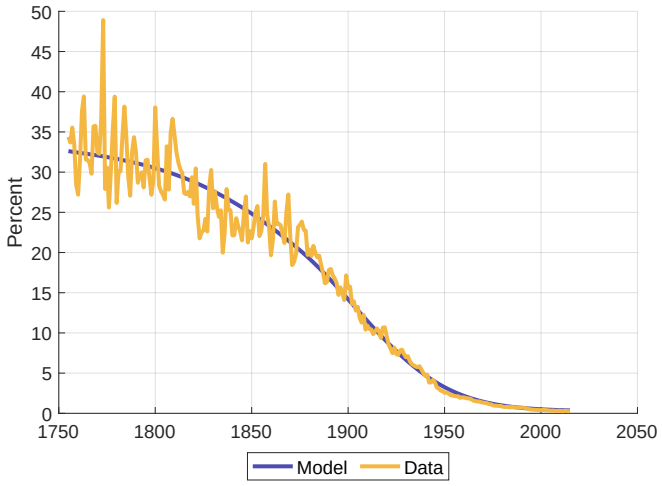


Figure 4: Child mortality rate in Sweden, model and data, 1755-2015

Source: Authors' calculations and Human Mortality Database (2022).

Figure 4 shows the model's fit to CMR. The correlation between model and data is 98%. The downward CMR trend in the model matches the observed timing: The decline starts as early as the mid 18th century in both model and data.[5] The onset of CMR reduction is a century before the Industrial Revolution started in Sweden (Jörberg, 1965). Panel A of Figure 3 shows the stagnation of GDP until the second half of the 19th century. These observations suggest that income growth cannot be the sole explanation for the decline in mortality; see Section 3.4.

3.3 Non-targeted moments

As a test of the model we examine several statistics that are not mechanically implied by CMR. These include LEB, CDR, population dynamics, mortality rates beyond age 4, life expectancies beyond birth, and age distribution in Sweden.

[5]Our theory is about the long-run trend in mortality and does not have year-specific shocks. So, it cannot reproduce the spikes in mortality, e.g., a 33% increase in CMR in 1772-73 (Larsson, 2020).

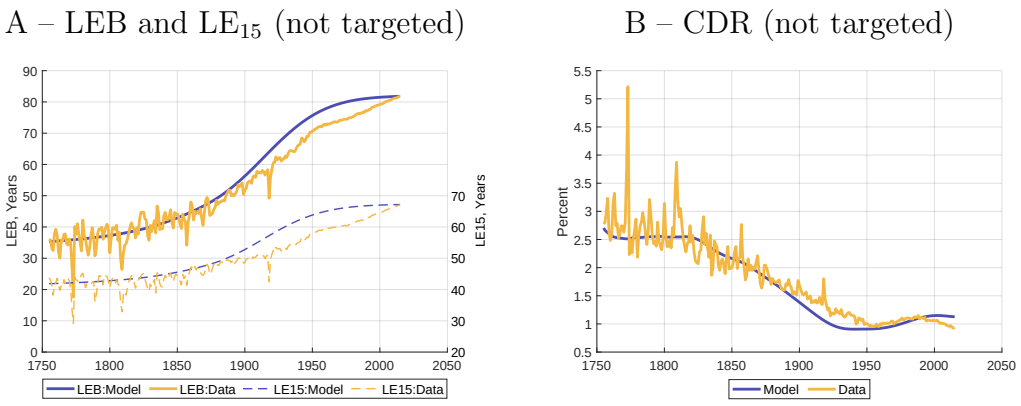


Figure 5: LEB, LE_{15}, and CDR in Sweden, model and data, 1755-2015

Note: Panel A: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

Life expectancy at birth A widely used mortality statistic is LEB, defined in Equation (2). Unlike CMR, which is measured using only mortality *below age* 5, LEB is measured using mortality at *all* ages. Thus, the model's fit for CMR does not imply a fit for LEB. Specifically, our model's mortality rates above age 4 could differ from those in the data and would imply discrepancies in Panel A of Figure 5.

Mortality rates at different ages contribute differently to the increase in LEB over time. Between 1800 and 1850, CMR accounts for more than 72% of the increase in LEB, while old-age (60+) mortality accounts for less than 8%. Between 1950 and 2000, CMR and old-age mortality account for less than 20% and more than 50%, respectively.[6] Our model is consistent with this pattern.

Besides LEB, one might be interested in LE_{15} since we assume children inherit their parents' technology and they are not economically active until age 15. LE_{15} depends on mortality at ages 15 and above. Panel A of Figure 5 shows that the model reproduces the trends in LEB and LE_{15} well, even though they are not targeted in the calibration.

[6]Between 1800 and 1850, the increase in LEB was 12.5 years. Holding CMR at its 1800 level, the increase in LEB is 3.5 years; holding old-age mortality fixed, the increase is 11.5 years. Between 1950 and 2000, the increase in LEB was 8.6 years. Holding CMR at its 1950 level, the increase in LEB is 6.9 years; holding old-age mortality fixed, the increase is 4 years.

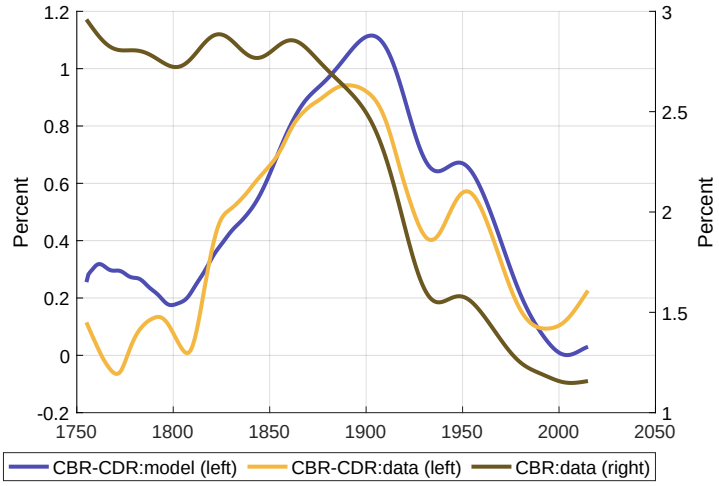


Figure 6: Population growth in Sweden (not targeted), model and data, 1755-2015

Note: Rate of population growth is the rate of natural increase.
Source: Authors' calculations and Human Mortality Database (2022).

Crude death rate CDR, defined in (1), depends on age distribution and age-specific mortality. In the model, age distribution is a function of mortality at all ages and the exogenous CBR. Again, the model's fit for CMR does not imply a fit for CDR. Panel B of Figure 5 shows that the model reproduces the trend in CDR.

Population dynamics Is the model consistent with population acceleration despite stagnant or declining CBR? Figure 6 illustrates the population growth—computed using the rate of natural increase—in the model and data. During the 19th century, observed population growth *increased* even though CBR did not; our model reproduces this fact. CDR declines more than CBR in the model, as in the data (Panel B of Figure 5). During the 20th century, the population decelerated in both model and data. This is predominantly due to the decline in CBR.

It is also clear from Panel B of Figure 5 that CDR in the model is less than that in the data in the late 18th century and early 20th century. The compounding effect of lower CDR in the model implies higher population level relative to the data.

Mortality at other ages Figure 7 shows the time series of mortality rates at ages 10-14, 30-34, and 50-54. As we indicated earlier, the observed age-specific mortality rates declined for all ages and their evolution is heterogeneous. The model reproduces this fact. Specifically, even though we calibrate only to CMR data, the model is consistent with different speeds of decline in mortality at other ages.

In the 18th and 19th centuries, mortality in Sweden is increasing with age except for CMR. By the mid 20th century, CMR falls below the mortality rate for those 60-64 years of age; by 2000, CMR falls below the mortality rate for those 40-44 years of age. Our model is consistent with this pattern.

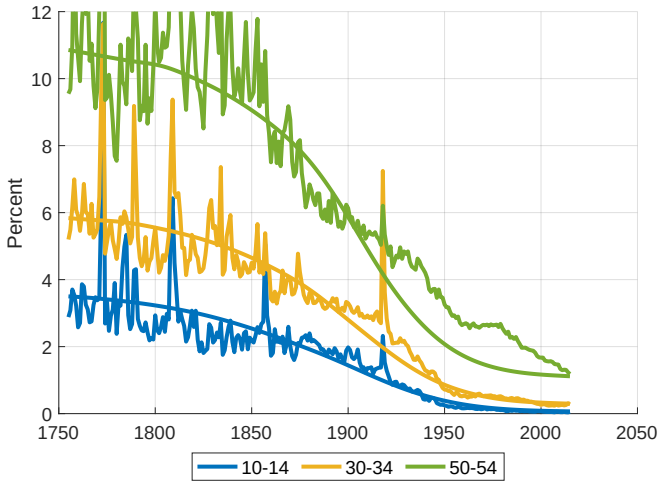


Figure 7: Age-specific mortality in Sweden (not targeted), model and data, 1755-2015

Note: We adjusted the scale of the vertical axis for clarity of display. In 1773, for instance, the 50-54 mortality rate was almost 25%, which is abnormally high.
Source: Authors' calculations and Human Mortality Database (2022).

Life expectancy at older ages Note that mortality rates at older ages are lower in the model relative to the data in the 20th century. As a result, life expectancies at older ages are higher in the model; see Figure 8 for life expectancy at age 30 and 50 in the model and data. We conjecture that older-age mortality is less in the model due to the fact that the relative price of health, i.e., the goods cost of accessing the modern technology, is constant in the model but increasing in the data. (In the

data, the price of health relative to goods increased 4-fold since World War I.) Thus, individuals acquire the modern technology at a faster rate in the model, resulting in a more rapid mortality decline. As suggestive evidence supporting our conjecture, recalibrating the model to 1755-1925 data (before the dramatic rise in the relative price of health) shows that the model is in line with the life expectancies at ages 30 and 50—see Section 3.5.

How can a non-decreasing CDR (Panel B of Figure 5) be consistent with increasing life expectancies? For instance, CDR does not decline in the second half of the 20th century, but life expectancies increase. This is due to population aging: The median age increased from 26 in 1900 to 40 in 2000 due to the decline of CBR.

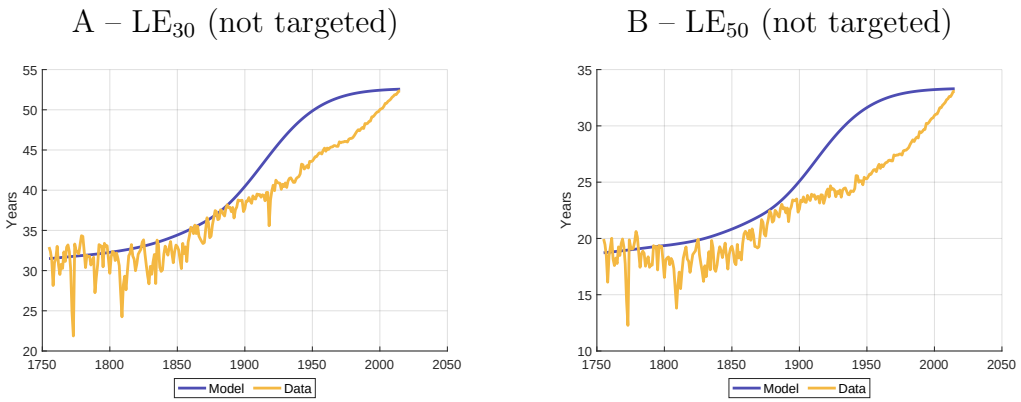


Figure 8: Life expectancy at ages 30 and 50 in Sweden, model and data, 1755-2015

Source: Authors' calculations and Human Mortality Database (2022).

Age distribution Figure 9 illustrates the age distribution in the model and data from 1755 to 2015. The initial age distribution is taken directly from the data, but the subsequent age distributions are not targeted and are functions of endogenous age-specific mortality and exogenous CBR. There are almost 29,000 points in the figure, and most of them are clustered around the 45° line.

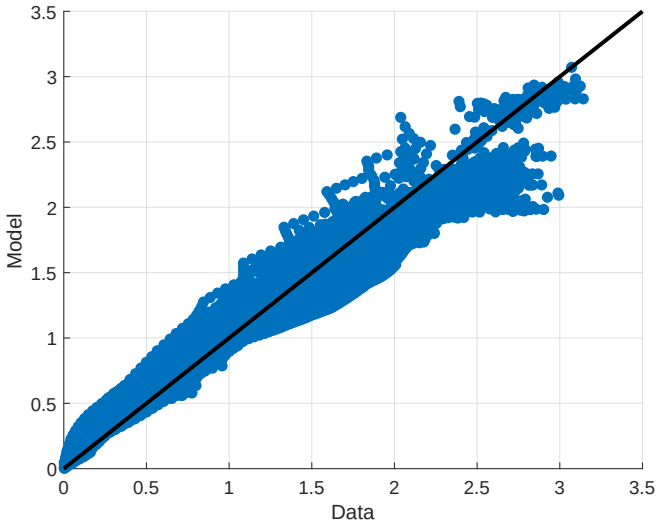


Figure 9: Age distribution in Sweden, model and data, 1755-2015

Note: Each point is a proportion of individuals by age in a year. There are 261 years and 111 ages per year, for a total of 28,971 points.
Source: Authors' calculations and Human Mortality Database (2022).

3.4 Model mechanics

Role of individual choices In our model, κ is the flow from obsolete to modern in every period without any individual actions, providing an exogenous channel for mortality decline. Recall that κ has a compounding effect leading to a mortality decline: A fraction κ of the obsolete population becomes modern every period, adding to the stock of modern and depleting the stock of obsolete. Furthermore, κ strengthens the Darwinian implication relative to the case where $A \equiv 0$. If $A \equiv \kappa > 0$, then (12) and (13) imply that the modern would dominate the population faster.

$$\frac{p^m_{t,j+1}}{p^o_{t,j+1}} = \frac{p^m_{t,j}s^m_j + \kappa p^o_{t,j}s^o_j}{p^o_{t,j}(1-\kappa)s^o_j} = \frac{p^m_{t,j}}{p^o_{t,j}}\frac{s^m_j}{s^o_j}\frac{1}{1-\kappa} + \frac{\kappa}{1-\kappa} > \frac{p^m_{t,j}}{p^o_{t,j}}.$$

To quantify the role of individual choices, we assume $\pi_1 = \Lambda = 0$ in Equation (15) and leave κ at its calibrated value. In this experiment, the average annual population growth rate is 0.19%, whereas in the baseline the growth rate is 0.52%. That is, κ explains 36%, so individual choices account for the rest of population growth.

However, our model with only κ would imply a zero correlation between CDR and GDP, which is inconsistent with the data.

Role of income We fix GDP at its 1755 value for all t. Panel A of Figure 10 shows that despite stagnant GDP, CDR declines from 2.7% in 1755 to 1.75% in 1880, almost identical to the baseline. In the baseline, CDR declines to a low of 0.91% in the 1950s, whereas in the constant-income counterfactual, CDR declines to 1.16%. Thus, 86% $(\frac{2.7-1.16}{2.7-0.91})$ of the decline occurs without any GDP growth. Panel B shows that the trend in LEB is virtually the same until 1880 in the baseline and counterfactual, and that the increase in LEB occurs without an increase in income.

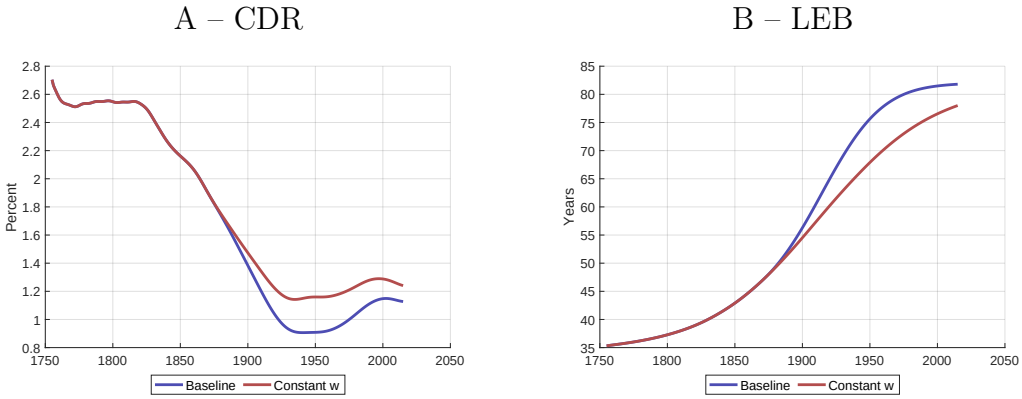


Figure 10: CDR and LEB in Sweden, baseline and constant GDP, 1755-2015

Source: Authors' calculations.

3.5 Medical theory of mortality

In our baseline calibration of Section 3.2 we use 2018 age-specific survival rates to represent the modern technology. To the extent that the higher survival rates in 2018 were due to medical advances and pharmaceuticals such as penicillin and sulfa drugs, some of the decline in CDR could have been due to modern medicine.

Penicillin was discovered in 1928 and sulfa drugs appeared in the 1930s. The Swedish data suggests that the role of modern medicine for the long-run trend in mortality is likely to be small. In Sweden, mortality has been declining for almost 200 years

Table 2: Trend in Swedish mortality prior to modern medicine

	CMR, %	Age 40-44, %	CDR, %
1750s	32.1	7.8	2.7
1790s	30.3	7.0	2.6
1850s	24.2	6.4	2.2
1910s	10.2	3.6	1.5

Note: Each row is an average of the death rates over the corresponding decade.
Source: Authors' calculations and Human Mortality Database (2022).

for all age groups before 1930 (see Table 2). In the United States prior to 1950, Jayachandran, Lleras-Muney, and Smith (2010) find that sulfa drugs contributed little to overall mortality reduction.

To abstract from modern medicine, we calibrate the model to fit CMR data for 1755-1925 in Sweden, using 1930 survival rates to represent the modern technology. We hold all of the parameters to be the same as in the baseline, except for π_1 and Λ. Since we changed the modern technology, it is natural to recalibrate the initial proportion of modern. However, recall that π_1 affects the level and rate of change of CMR, so we use Λ to discipline the rate of change.

The pre- modern medicine calibration reproduces the data well: Unlike our baseline calibration, LEB, LE_{15}, LE_{30}, and LE_{50} do not exceed the data (Figure 11). Furthermore, the level of population in 1925 is almost the same under the two calibrations: They differ by less than 7% (Figure 12).

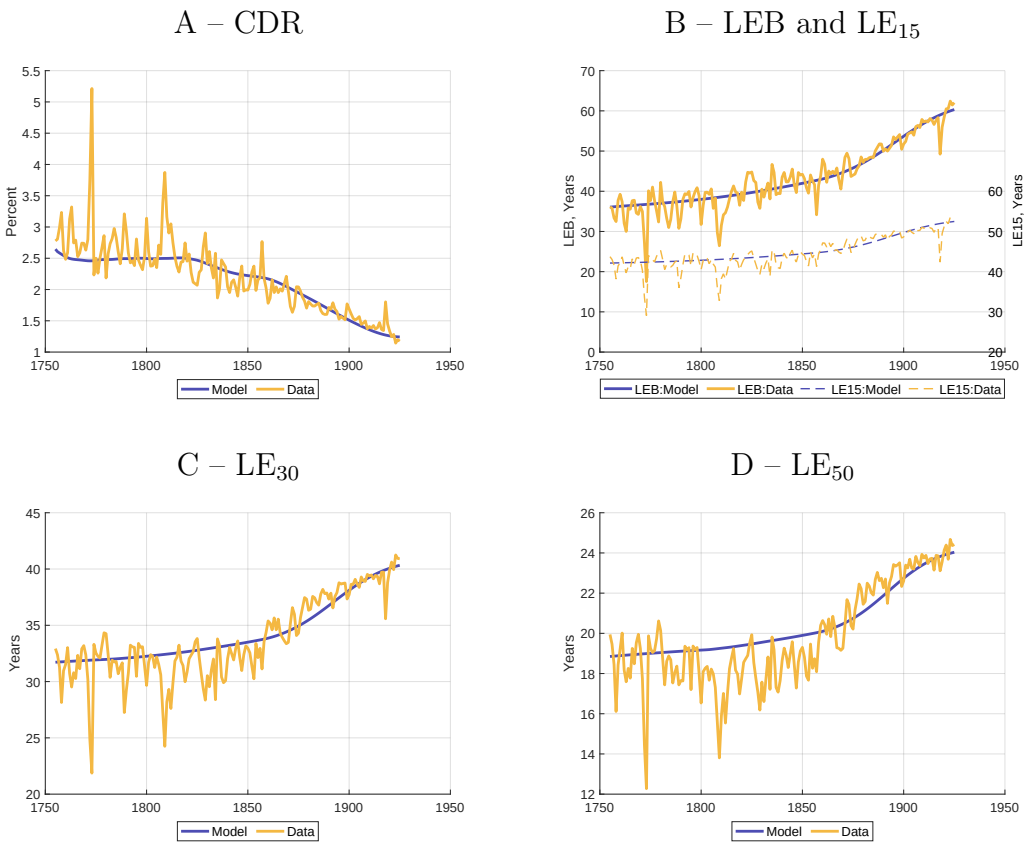


Figure 11: Non-targeted mortality statistics in Sweden before modern medicine, model and data, 1755-1925

Source: Authors' calculations and Human Mortality Database (2022).

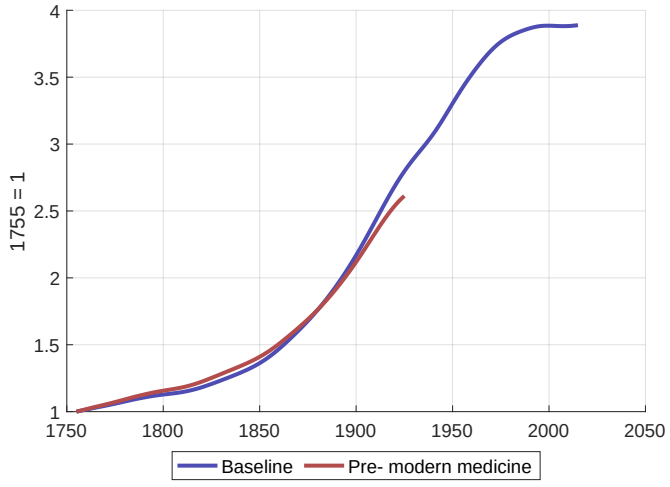


Figure 12: Population in Sweden, baseline and pre- modern medicine calibrations

There have, of course, been medical innovations before the 20th century that have reduced deaths from specific diseases. It does not imply, however, that medical innovations are the main drivers of long-run trends in mortality statistics. There are several reasons for this. Consider the case of smallpox vaccine, discovered in the late 18th and early 19th centuries. First, while Ager, Worm Hansen, and Sandholt Jensen (2018) show that smallpox mortality declined by 60% upon the introduction of the vaccine in Sweden (see their Figure 1), they also document that *smallpox mortality* had declined by 50% between 1750 and 1800, prior to the introduction of the vaccine. Second, Ager et al. (2018) note that smallpox "killed approximately 10 percent of the population... in the second half of the 18th century" in Sweden (p. 488); Guy (1882) cites a similar number for London during the 18th century when smallpox was at its height. That is, 90% of deaths were not due to smallpox. Third, while the vaccine changed the trend in smallpox mortality, it did not result in a trend break in the 1800s in the data on aggregate mortality statistics (Figures 4, 5, 7, and 8).

3.6 Infrastructure theory of mortality

Another alternative to our theory is that public infrastructures, particularly in sanitation, were the main drivers of mortality reduction. There are three problematic issues with this alternative theory of long-run trend in mortality.

First, Woods and Woodward (1984) conclude that public works, such as sewer systems, have limited efficacy because they reduce exposure to food- and waterborne infections (e.g., dysentery and cholera) but not to airborne infections (e.g., tuberculosis, measles, whooping cough, pneumonia, influenza). Even in the early 20th century, despite public infrastructures, airborne diseases caused a significant number of deaths. For instance, in Sweden around 1900, airborne diseases were three times as fatal as waterborne diseases (Helgertz and Önnerfors, 2019, Figure 3); in England and Wales in 1901, the deaths from airborne diseases were more than three times the deaths from diarrhoeal diseases (Preston, Keyfitz, and Schoen, 1972). Similar to the smallpox vaccine in Section 3.5, it is conceivable that public infrastructure reduces deaths from specific diseases in specific regions at specific points in time. However, such reductions are unlikely to have a substantial impact on the long-run trend in mortality statistics.

Second, in Sweden, water and sewer infrastructures were introduced in the 1850s.

Helgertz and Önnerfors (2019, p. 307) find "a 9 percent reduction in waterborne disease mortality associated with the implementation of water and/or a sewerage system" between 1875 and 1930. Furthermore, CMR had already declined from 32% in the 1750s to 24% by the 1850s (see Table 2) and there is no evidence of a break in the long-run trend in mortality statistics in the 1850s (Figures 4, 5, 7, and 8).

Finally, insofar as these infrastructures are large-scale investments, they are income-based drivers of mortality. And, Livi-Bacci (1991), Wrigley et al. (1997), Leukhina and Turnovsky (2016), and Delventhal et al. (2021), among others, have noted the limited role of income for mortality.

3.7 Nutrition theory of mortality

Improved nutrition has been proposed as the leading cause for the historical decline of European mortality (McKeown, 1976). This view has been criticized by a number of authors. Food consumption in Britain was flat before 1850 (Clark, Huberman, and Lindert, 1995), but infant mortality had been declining since the early 18th century (Wrigley et al., 1997). Livi-Bacci (1983) points out that, between 1550 and 1750, the British peerage had a life expectancy identical to that of the general population despite having access to better nutrition.

As in Section 3.6, the nutrition theory is also based on income to the extent that food production increases with economic development, so the critique in Section 3.6 applies. If improvements in nutrition are consequences of individuals' cleanliness and safe preservation of food items, then our theory applies.

4 Quantitative analysis: Western Europe and the World

As a further test of the model, we examine mortality in two countries for which we have almost 200 years of data—France (1820-2018) and Denmark (1835-2018)—and the acceleration of world population despite a declining CBR, noted in Section 1. We also examine mortality in Norway, Netherlands, Switzerland, and Finland for which we have shorter time series. The results for these countries are in Appendix C.

Mortality in Denmark and France For each country, we obtain demographic data from the Human Mortality Database and GDP data from the Maddison Project Database. We represent a country's GDP in a manner similar to that for Sweden. Specifically, the initial value of w for country i is scaled relative to that of Sweden:

$$\frac{w^i_{t_i}}{w^{\text{Sweden}}_{t_i}} = \frac{\text{GDP}^i_{t_i}}{\text{GDP}^{\text{Sweden}}_{t_i}}, \tag{16}$$

where t_i is the starting year for country i. We choose the sequence of growth rates to best reproduce country i's GDP.

We then proceed as follows: Country i is endowed with its GDP trend as above, its trend CBR, and its observed age distribution in year t_i. It is also endowed with Sweden's obsolete and modern technologies (Panel A of Figure 2).

We choose π_1 and Λ such that country i's CMR implied by the model best reproduces the observed CMR, holding all other parameters at the values calibrated for Sweden. That is, only π_1 and Λ are country-specific. As noted in Section 2.3, we can use country-specific π_1 to account for the fact that CMR in 1835 for Denmark and in 1820 for France are different from that in Sweden in 1755. But, π_1 also affects the speed of decline of CMR. We use country-specific Λ to match the speed of decline.

Figure 13 displays the model's ability to fit non-targeted mortality statistics: CDR, LEB, and LE_{15}. As in the case of Sweden, our model fits the data for France and Denmark well. In Denmark, for instance, the model captures the decline in CDR from the mid 19th century to mid 20th century. It also captures the increase post 1950s, which suggests that the model's age distribution is consistent with the data. This explains, as in the case of Sweden, the simultaneous increase in CDR and LEB.

World population acceleration We obtain world data on CBR and CDR from the United Nations and on GDP from the Maddison Project Database. We do not have data on age distribution or age-specific mortality.

The exogenous time-varying variables are world CBR and w. The first observation we have for the world is in 1800. We compute w for the world in 1800 as in Equation (16). We then represent world GDP growth as we did for Sweden. The obsolete

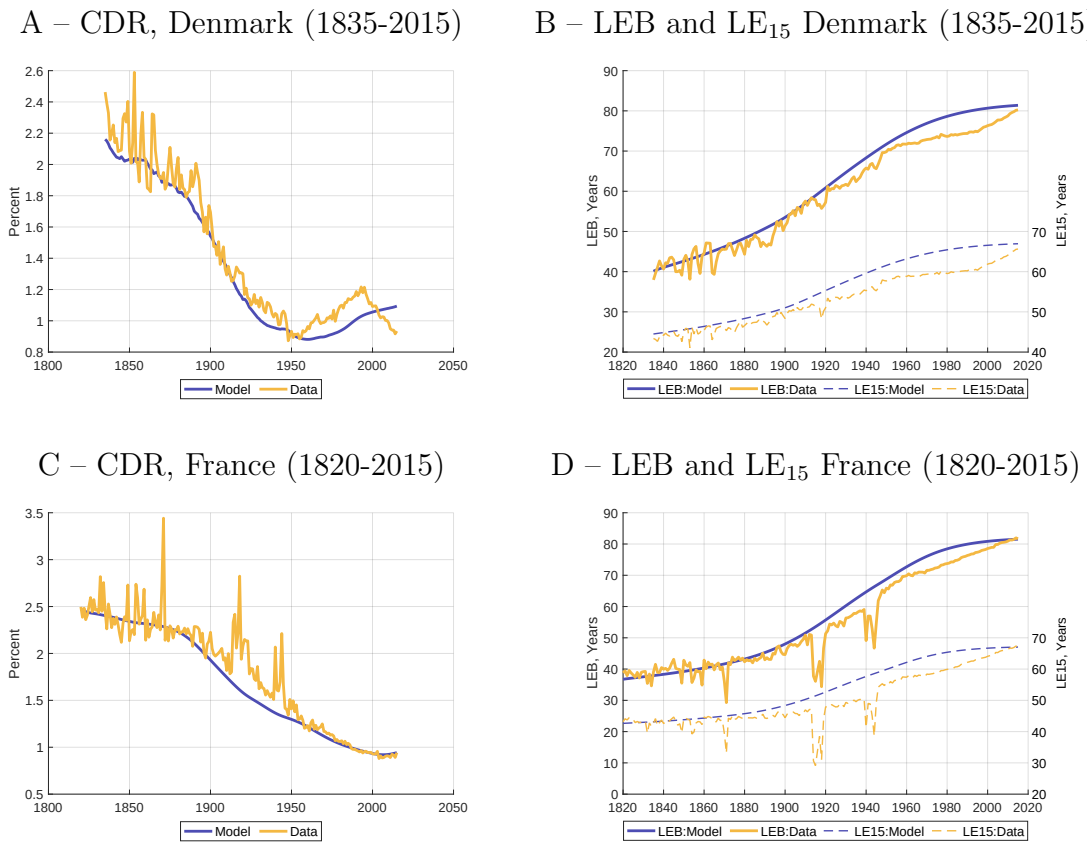


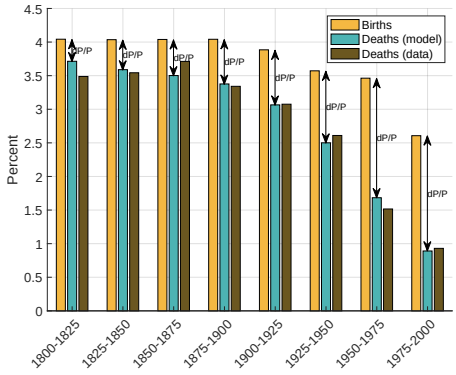
Figure 13: The model's fit to non-targeted data for Denmark and France

Note: Panels B and D: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

and modern technologies are the same as Sweden's. The initial age distribution is Sweden's age distribution in 1800.

The world's CDR in 1800 is "too high": Even with 100% obsolete individuals in 1800, the model cannot generate the observed CDR of 3.7%. In other words, the world's obsolete technology in 1800 was worse than that of Sweden in 1751. To remedy this problem, we use a scaling factor less than one to reduce the survival probabilities in the obsolete technology. We choose π_1, Λ, and the scaling factor such that the world's CDR implied by the model best reproduces the observed CDR, holding all other parameters at the values calibrated for Sweden. (We do not have CMR data for

A – Population growth



B – LEB

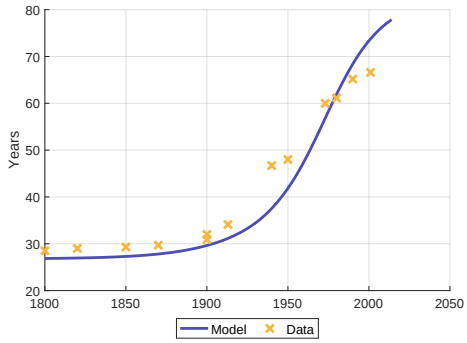


Figure 14: World population growth and life expectancy at birth, 1800-2000

Source: Panel A: Authors' calculations and United Nations. Panel B: Authors' calculations, Riley (2005b, Table 1), and Acemoglu and Johnson (2007, Table 1).

the world, so we cannot follow the same calibration strategy as we did for Sweden.) We find $\pi_1 = 0.3\%$, $\Lambda = 3.9$, and the scaling factor $= 0.99$.

Panel A of Figure 14 illustrates that the model reproduces the acceleration of world population. The observed CBR has been stagnant or declining since the early 19th century. Our model reproduces the fact that the secular decline in CDR is more than that in CBR. As a result, population accelerates in the model and data.

As a test of the model, Panel B of Figure 14 illustrates the fit to the evolution of LEB in the world since 1800. The increase in LEB in the model is consistent with the estimates in Riley (2005b) and Acemoglu and Johnson (2007).

5 Conclusion

The economic demography literature is largely concerned with fertility, assuming mortality to be either exogenous or Malthusian, i.e., a decreasing function of income. We argue that both Beckerian theories of fertility and Malthusian theories of mortality are incomplete explanations of population dynamics. We propose a theory of mortality reduction that accounts for population growth over long periods of time in Western Europe and the world.

In our model, individuals choose to incur goods and/or time costs to adopt better health technology to reduce their mortality risk at each age. There is also an exogenous flow of adopters, so mortality declines for reasons other than individual choices. Adopters create a dynamic externality: They induce others to adopt. This determines the speed of technology diffusion and evolution of age-specific mortality rates. These, in turn, determine the population dynamics.

Our theory delivers mortality and population growth consistent with (i) life expectancies at various ages and CDR, (ii) the acceleration of population despite a declining CBR, (iii) the evolution of age distribution, and (iv) the minor, but non-zero, role of income for mortality. Quantitatively, roughly two-thirds of the population growth is due to the individual-choice channel and the remaining one-third is due to the exogenous channel.

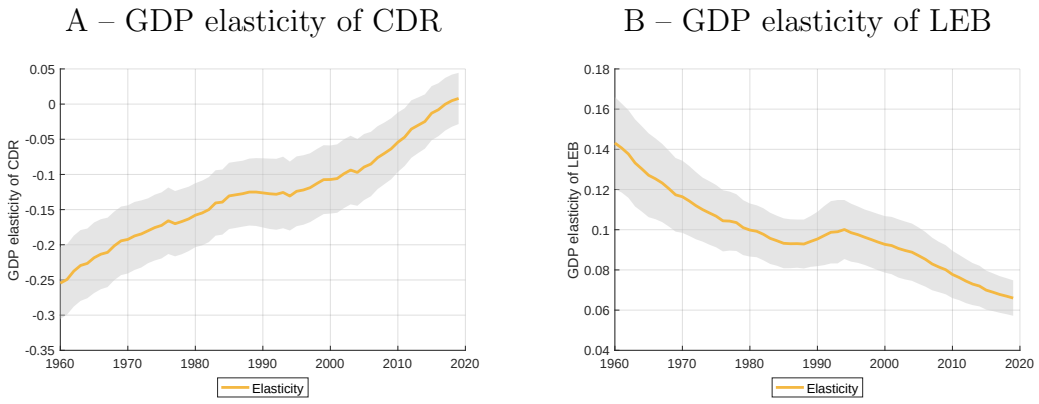


Figure 15: Convergence in mortality

Note: Panel A shows the cross-country GDP elasticity of CDR. Panel B shows the cross-country GDP elasticity of LEB. The gray areas represent 95% confidence intervals.
Source: World Bank and authors' calculations.

Our theory could be useful in explaining several demographic facts since 1960: (i) survival miracles, i.e., CDR reductions in countries whose incomes have been decreasing; (ii) the convergence of CDR and life expectancies despite the lack of income convergence—see Figure 15; and (iii) the large contribution of poor countries to world population growth, despite the decline in fertility in poor countries.[7]

[7]The convergence in LEB despite the absence of income convergence was noted earlier by Becker et al. (2005) and Acemoglu and Johnson (2007).

Endogenizing fertility in our model would strengthen the role of individual choices for population growth. If individuals have preferences over the number of surviving children, then technology diffusion that reduces CMR would also reduce fertility and would have the potential to account for the declining trends in both CDR and CBR.

Our theory could also be used to study economic transition. We could augment Hansen and Prescott (2002), for example, with our model of technology adoption and endogenize population growth as a function of both mortality and income. Such a model would deliver a theory of demographic transition based on mortality instead of fertility and economic transition from Malthusian stagnation to modern growth.

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A Population statistics

Recall that the crude death rate at time t is

$$\text{CDR}_t = \frac{\text{Total deaths at time } t}{p_t} = \frac{1}{p_t}\sum_j p_{t-j,j}q_{t-j,j}, \tag{A.1}$$

LEB at time t is

$$\text{LEB}_t = \sum_{j=0}^{\infty}(j+1)q_{t-(j+1),j+1}\prod_{k=0}^{j}(1-q_{t-k,k}) \tag{A.2}$$

and life expectancy conditional on being alive at any age i in period t is

$$\text{LE}_{t,i} = \sum_{j=i}^{\infty}(j+1-i)q_{t-(j+1),j+1}\prod_{k=0}^{j-i}(1-q_{t-(k+i),k+i}). \tag{A.3}$$

A.1 Simple aggregate specifications

Consider perpetual-youth models as in Yaari (1965) and Blanchard (1985). First, suppose that the mortality rate is just time varying (Panel A of Table A.1): $q_{t-j,j} = q_t$.

Table A.1: Mortality specifications

	Age			
Time	0	1	2	...
t	q_t	q_t	q_t	...
$t+1$	q_{t+1}	q_{t+1}	q_{t+1}	...
$t+2$	q_{t+2}	q_{t+2}	q_{t+2}	...
$\vdots$				

A – Time-specific mortality–$q_{t-j,j} = q_t$

	Age			
Time	0	1	2	...
t	q_t	q_{t-1}	q_{t-2}	...
$t+1$	q_{t+1}	q_t	q_{t-1}	...
$t+2$	q_{t+2}	q_{t+1}	q_t	...
$\vdots$				

B – Cohort-specific mortality–$q_{t-j,j} = q_{t-j}$

Then, from Equation (A.1), we see that $\text{CDR}_t = q_t$ and does not depend on the age distribution. From Equation (A.2),

$$\text{LEB}_t = q_t(1-q_t) + 2q_t(1-q_t)^2 + 3q_t(1-q_t)^3 + \cdots = q_t\sum_{k=1}^{\infty}k(1-q_t)^k = \frac{1-q_t}{q_t}.$$

Note that there is a one-to-one mapping between CDR and LEB. If the model delivers the observed CDR, then it would be inconsistent with LEB. For instance, using CDR data for Sweden, the implied LEB in 1950 would be 98 instead of the observed 71. Furthermore, life expectancy at every age at time t would be equal to LEB_t. Our model in Section 2 does not have a one-to-one mapping between CDR and LEB.

Second, consider an alternative as in Panel B of Table A.1: $q_{t-j,j} = q_{t-j}$. In this alternative, for individuals of cohort $t-j$, survival rate is the same no matter how old they are. However, at any point in time, mortality rate is heterogeneous across age. Suppose this second specification delivers the observed time series of LEB and CDR. It is easy to see from Panel B of Table A.1 that $\text{LEB}_t = \text{LE}_{t+1,1} = \text{LE}_{t+2,2} = \cdots$ This would be at odds with observed life expectancies at various ages. Our model in Section 2 does not have this implication.

In sum, neither specification is quantitatively consistent with the set of mortality statistics described in Equations (A.1), (A.2), and (A.3).

B Optimization

Given the functional form for A (Section 3), the first-order conditions (7)-(8), abstracting from time and generation subscripts, imply

$$\begin{aligned} 0 &= U_1\left(w\left(1-h\right)-y\right)-\beta s^o \pi \Lambda \exp\left(-\chi\left(y,h\right)\right)\chi_1\left(y,h\right)\Delta \\ 0 &= U_1\left(w\left(1-h\right)-y\right)w-\beta s^o \pi \Lambda \exp\left(-\chi\left(y,h\right)\right)\chi_2\left(y,h\right)\Delta \end{aligned}$$

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

$$h = yX\left(w\right) \text{ where } X\left(w\right) = \left(w\frac{\alpha_y}{\alpha_h}\right)^{1/(\theta-1)}.$$

Note that, at the optimum,

$$\chi\left(y,h\right) = y\left(\alpha_y + \alpha_h X\left(w\right)^\theta\right)^{1/\theta} \text{ and } \chi_1\left(y,h\right) = \alpha_y\left(\alpha_y + \alpha_h X\left(w\right)^\theta\right)^{1/\theta-1} \equiv \chi_1(w),$$

where $\chi_1(w)$ is a function of only w at the optimum. Given the functional form for U (Section 3), the first-order condition for y is

$$\sigma \exp\left(-\sigma w + \sigma y\left(1+wX\left(w\right)\right)\right) = \beta s^o \Delta \pi \Lambda \exp\left(-y\left(\alpha_y + \alpha_h X\left(w\right)^\theta\right)^{1/\theta}\right)\chi_1\left(w\right)$$

$$\text{or, } y\left[\sigma\left(1+wX\left(w\right)\right)+\left(\alpha_y+\alpha_h X\left(w\right)^{\theta}\right)^{1/\theta}\right]=\sigma w+\ln\left(\frac{\beta s^o \Delta\pi\Lambda}{\sigma}\chi_1\left(w\right)\right).$$

C Western Europe

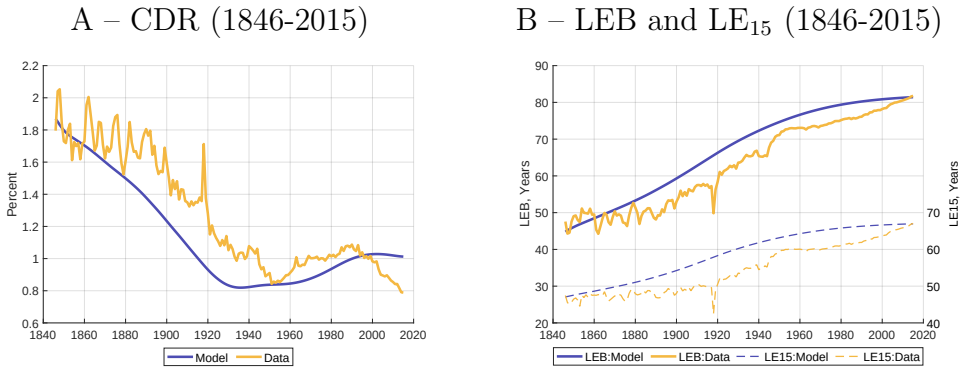

Figure C.1: CDR, LEB, and LE_{15} in Norway, model and data, 1846-2015

Note: Panel B: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

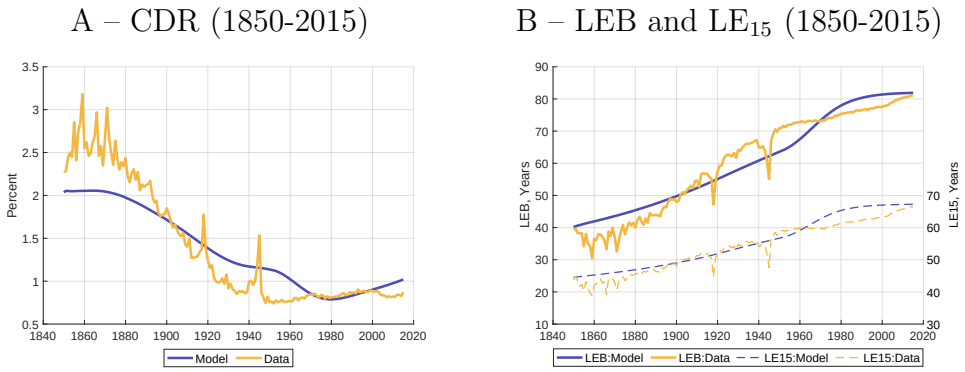

Figure C.2: CDR, LEB, and LE_{15} in Netherlands, model and data, 1850-2015

Note: Panel B: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

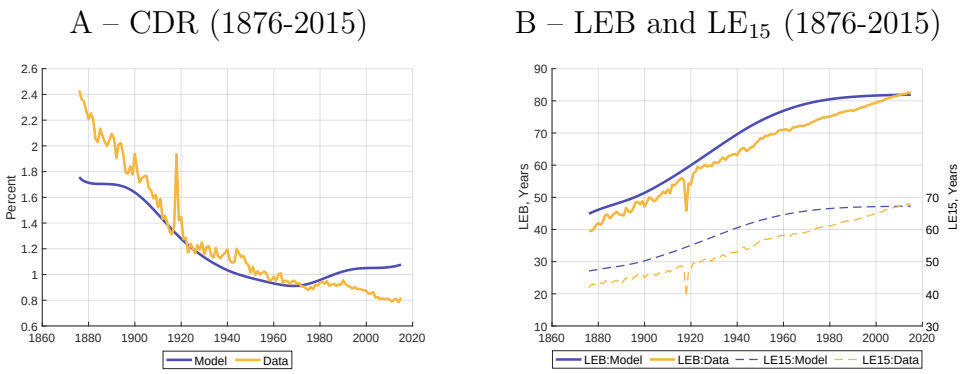

Figure C.3: CDR, LEB, and LE_{15} in Switzerland, model and data, 1876-2015

Note: Panel B: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

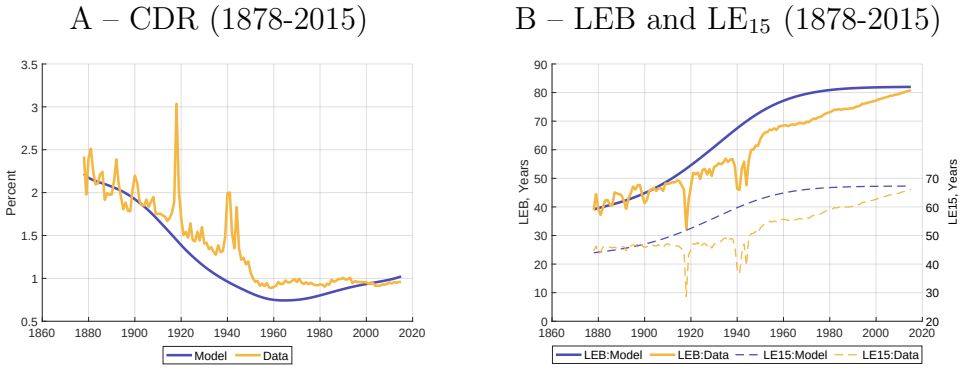

Figure C.4: CDR, LEB, and LE_{15} in Finland, model and data, 1878-2015

Note: Panel B: LEB on left axis and LE_{15} on right axis.
Source: Authors' calculations and Human Mortality Database (2022).

D A model with endogenous fertility and mortality

We add two features to our baseline model in Section 2 to endogenize fertility. (i) Parents' utility increases with the number of surviving children. (ii) Cost of raising children increases with the number of surviving children. These features deliver an endogenous CBR that is linked to CDR. And, they also deliver the negative relationship between income and CBR.

As before, s_j^i denotes the probability of surviving to age $j+1$ conditional on being alive at j, under technology i, and $S_j^i \equiv \prod_{k=0}^{j-1} s_k^i$ denotes the unconditional probability of surviving to age j. Let $S_0^i = 1$.

Newborn remain economically inactive children, living with their parents until age $k-1$. Children of modern (obsolete) parents are modern (obsolete). At age k, children become adults endowed with one unit of productive time for each remaining period of their lives. Adults have preferences over consumption and the number of children living with them. The wage rate is w_t and there are no savings.

At age k, obsolete adults spend time and/or goods to increase their probability of becoming modern. The probability of adopting the modern technology at time t is $A(y, h, \pi_t)$. At age $k+1$, adults of both types choose their fertility, b. At age $2k+1$, adults no longer have any children living with them.

The value of type i adults of generation t and age $2k+1$ is

$$\mathcal{V}_{t,2k+1}^i = \frac{1}{S_{2k+1}^i} \sum_{j=2k+1} S_j^i \beta^{j-(2k+1)} U\left(w_{t+j}\right), \tag{D.4}$$

where β is the subjective discount factor and U is the period utility index. Fertility decisions take place at age $k+1$, for $i \in \{o, m\}$:

$$\begin{aligned} \mathcal{V}_{t,k+1}^i \quad = \quad & \max_b \ \frac{1}{S_{k+1}^i} \sum_{j=k+1}^{2k} S_j^i \beta^{j-(k+1)} \left[U\left(w_{t+j} - \mathcal{C}_{t+j}\left(n_j\right)\right) + \gamma V\left(n_j\right)\right] \qquad \text{(D.5)} \\ & + \beta^k \frac{S_{2k+1}^i}{S_{k+1}^i} \mathcal{V}_{t,2k+1}^i, \\ \text{s.t.} \qquad & n_j = b S_{j-k-1}, \qquad \text{(D.6)} \end{aligned}$$

where n_j is the number of surviving children living with age-j parents. The functions V and $\mathcal{C}_t$ are the period utility index and the cost, respectively, of surviving children. The cost function is indexed by time because the evolution of wages affects the opportunity cost of time spent raising children. Let b_t^i denote the optimal fertility decision of generation t, for $i \in \{o, m\}$.

The adoption decision of obsolete age-k adults is

$$\mathcal{V}^o_{t,k} = \max_{y_t, h_t} U(w_{t+k}(1-h) - y_t) + \beta s^o_k \Big[A\left(y_t, h_t, \pi_{t+k}\right) \mathcal{V}^m_{t,k+1} + \beta \left(1 - A\left(y_t, h_t, \pi_{t+k}\right)\right) \mathcal{V}^o_{t,k+1} \Big]. \quad \text{(D.7)}$$

D.1 Population dynamics

Let $p^i_{t,j}$ denote the age-j population of cohort t and type i. The age-0 population at date t is

$$p^i_{t,0} = \text{CBR}^i_t \sum_{j=0}^{J} p^i_{t-j,j} \quad \text{(D.8)}$$

where CBR^i_t denote the "type-specific CBR" of period t:

$$\text{CBR}^i_t = \frac{b^i_{t-(k+1)} p^i_{t-(k+1),k+1}}{\sum_{j=0}^{J} p^i_{t-j,j}}.$$

At all ages, except age k, the cohort size evolves according to

$$p^i_{t,j+1} = s^i_j p^i_{t,j}, \qquad \text{for } j \neq k \text{ and } i = o, m. \quad \text{(D.9)}$$

The proportion of economically active modern adults, π, can be defined as in Equation (9). At age k, the obsolete of cohort t may remain obsolete or become modern:

$$p^o_{t,k+1} = s^o_k p^o_{t,k} \left[1 - A(y_t, h_t, \pi_{t+k})\right], \quad \text{(D.10)}$$

$$p^m_{t,k+1} = s^m_k p^m_{t,k} + s^o_k p^o_{t,k} A(y_t, h_t, \pi_{t+k}) p^o_{t,k}. \quad \text{(D.11)}$$

Note that Equation (D.8) is the type-specific version of Equation (10). Equations (D.9)-(D.11) correspond to (11)-(13), adjusting for the fact that adoption takes place only at age k in the model with endogenous births.