Technology adoption, mortality, and population dynamics

Technology Adoption, Mortality, and Population Dynamics[*]

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Abstract

We develop a quantitative theory of mortality and population dynamics, emphasizing individuals' decisions to reduce their mortality by adopting better health technology. Expanded use of this technology reduces the cost of adoption and confers a dynamic externality by increasing the future number of individuals who use the 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 of 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 (2000), 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. For such a theory, a key endogenous variable is the time series of mortality rate at *every* age, not just univariate CDR.[2]

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

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[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 filth-disease correlation, which gave rise to health practices that reduced mortality in early 19th century Europe.

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 to become 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 modern technology diffuses.

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, technology adoption becomes cheaper, so more individuals adopt the modern technology and mortality declines. Current actions by individuals increase the future proportion of modern, which is not internalized by any individual, creating a dynamic externality. The proportion of modern evolves according to the familiar S-shaped pattern of technology diffusion: With few modern, as well as with few obsolete, the flow of adopters is "small."

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 to deliver the flow of newborns; CBR does not affect the individual decisions. Age-specific mortality rates and the flow of newborns determine the age distribution.

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 targeted 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 our model is consistent with 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 in Sweden remained constant or declined, but the population growth rate increased steadily. Our model delivers this acceleration: CDR declines more than CBR in the model and data.

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 hereafter) does not predict the start of the CDR decline.

The model also delivers the long-run trends in CDR, age-specific mortality rates, life expectancies, and population for several European countries, using the same time-invariant parameters as those of Sweden. 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. 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.

Three remarks about our theory are in order. First, we do not have an explicit role for medicine or large-scale public investment in water and sewer infrastructure. One could argue that they play an implicit role in our model because individuals have access to the 2018 survival probabilities, which might have been the result of medicine and infrastructure. To exclude any role for these factors, we represent the modern technology by the 1850 survival rates. The period 1750-1850 represents a time in Sweden (i) when GDP did not grow, (ii) before the Pasteur revolution, and (iii) before the construction of infrastructure. The implications for mortality statistics over this period are practically the same as those in the baseline. We elaborate more on these and other factors in Sections 3.4, 4.1, 4.2, 4.3, 4.4, and 4.5.

Second, one might argue that ours is an "incomplete" theory of population since we take CBR as exogenous. While a model of CBR and CDR could be used to confront the data on CBR, it would not yield any additional moments to confront the data on population growth, age distribution, and life expectancies, relative to our baseline with exogenous CBR.

Third, we allow for the possibility that mortality declines and, hence, population

grows for reasons other than the individual-choice mechanism. In each period, there is an exogenous flow of obsolete who become modern. This flow could represent, for instance, biological mutations or public health investments that make individuals survive longer; and it has a compounding effect. In a counterfactual where we shut down the individual-choice mechanism, the exogenous channel accounts for 36% of the population growth rate in Sweden.

We develop the model in Section 2. We present functional forms, calibrate to Swedish CMR, and test the model in Section 3. We discuss the role of alternative mechanisms in Section 4. In Section 5 we quantitatively examine mortality in Denmark, France, and other Western European countries and the acceleration of the world population. We conclude in Section 6.

2   MODEL

As noted in the previous section, our goal is to develop a quantitative theory of 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 $\sum_j p_{t-j,j} q_{t-j,j}$. CDR 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 j at time t ($\text{LE}_{t,j}$) is the expected additional years of life for individuals of age j if they experience the death probabilities observed at time t.

$$\text{LE}_{t,j} = 1 - q_{t-j,j} + (1 - q_{t-j,j}) \, \text{LE}_{t,j+1}. \tag{2}$$

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

CDR depends on age-specific mortality rates and age distribution of population. Life

expectancy at every age, on the other hand, does not depend on age distribution. Note also that date-t CDR and life expectancies 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 CDR and life expectancies. 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 time $t+j$.

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

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

The function A depends positively on π. This is a reduced-form approach to modeling learning through random meetings. Imagine the proportion of people who wash their hands with soap regularly and survive longer is 10%, i.e., $\pi = 0.1$. The odds of running into such people and learning the practice of hand washing are low. The odds of learning the practice are higher if the proportion is 90%.

We also assume

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

The incentive to spend resources to adopt the modern technology increases with the proportion of modern. With a higher proportion, the marginal effect of time and goods on the probability of adoption increases. The proportion of modern also creates a positive externality. Current actions by individuals affect the future proportion of people who use soap regularly and survive longer. This effect on the future proportion is not internalized by any individual.

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

$$V_{t,j}^o = \max_{y,h} U\left(w_{t+j}(1-h) - y\right) + \beta s_j^o \left[A(y,h,\pi_{t+j})V_{t,j+1}^m + (1 - A(y,h,\pi_{t+j}))\,V_{t,j+1}^o\right]. \tag{6}$$

Since $s_J^o = s_J^m = 0$, the terminal condition for modern and obsolete individuals is

$$V_{t,J}^m = V_{t,J}^o = U(w_{t+J}).$$

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

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

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

where $\Delta_{t,j} \equiv V_{t,j+1}^m - V_{t,j+1}^o$. (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).

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 alters the individual choices.

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 $p_{t,j}^i$, $i = o, m$, denote the population of obsolete and modern, respectively, of age j in generation t. The age-j population of generation t is then $p_{t,j} = p_{t,j}^m + p_{t,j}^o$. 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

$$\pi_t = \frac{\sum_{j=k}^{J} p_{t-j,j}^m}{\sum_{j=k}^{J} p_{t-j,j}^m + \sum_{j=k}^{J} p_{t-j,j}^o}. \tag{9}$$

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 additional observables or insights. The last assumption represents the notion that children acquire the health practices of their parents. The age-0 populations in cohort t are

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

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

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

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

$$p_{t,j+1}^o = s_j^o p_{t,j}^o \left[1 - A(y_{t,j}, h_{t,j}, \pi_{t+j}) \right], \tag{12}$$
$$p_{t,j+1}^m = s_j^m p_{t,j}^m + s_j^o p_{t,j}^o A(y_{t,j}, h_{t,j}, \pi_{t+j}). \tag{13}$$

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_j^o of those who remain obsolete survives to the next age. Equation 13 indicates that a fraction s_j^m of the age-j modern population survives to the next period and a fraction $s_j^o A(y_{t,j}, h_{t,j}, \pi_{t+j})$ of the age-j obsolete population survives and becomes modern in the next period.

Equilibrium   Given an initial population at date 1, $\{p_{1-j,j}^m, p_{1-j,j}^o\}$, 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, $\{p_{t,j}^m, p_{t,j}^o\}$, 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. $\{p_{t,j}^m, p_{t,j}^o\}$ evolves according to (10)-(13);
3. $\{\pi_t\}$ is consistent with individual decisions, i.e., it satisfies (9).

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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 all times. 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.

To summarize, we have a model of diffusion. It yields age-specific mortality rates over time. 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 modern would dominate the population. In (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
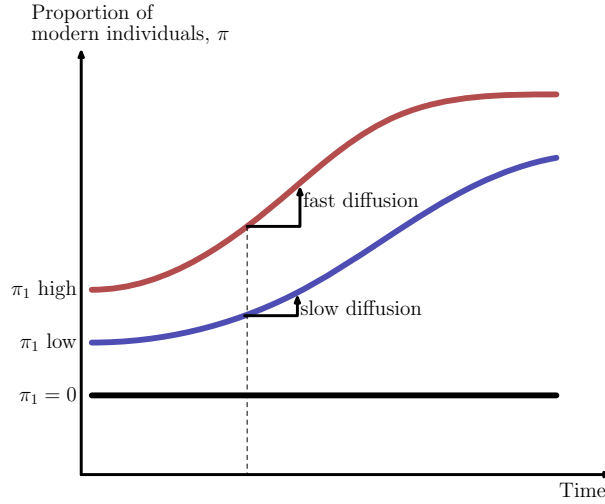
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Figure 1: The diffusion of the modern technology

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 correlation between CDR and income is not zero in the time series 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 to Swedish historical data. We then study the quantitative implications for mortality statistics and population 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. A fraction κ of the obsolete population becomes modern every period, adding to the stock of modern and depleting the stock of obsolete. The flow κ could represent, for instance, biological mutations or public health investments every period 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 rate of decline of mortality, but not its initial level.

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

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

The form for U, along with the CES specification in (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 our computations to ensure 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).[3]

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 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 real gross domestic product per capita (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

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[3]The availability of the 2018 survival rates does NOT mean availability of MRI, CAT scan, or cancer treatments. What we mean is actions such as boiling water, washing hands, and using soap, for example, that are conducive to higher survival.
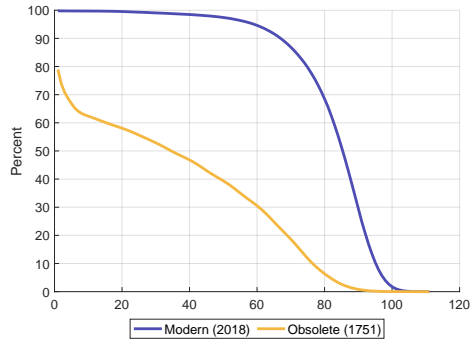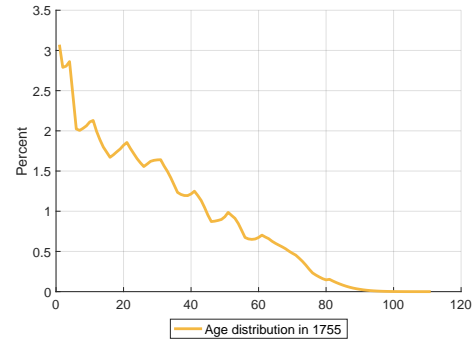
Figure 2: Survival probabilities and initial age distribution in Sweden

Note: Panel A shows the *unconditional* probabilities of survival to each age j, $\prod_{k=0}^{j-1} s_k$, in 1751 (obsolete) and 2018 (modern). The survival probability used in (14) is the conditional probability s_j, i.e., the probability that a person of age j would survive to age $j+1$. The initial age distribution in Panel B is Sweden's age distribution in 1755.
Source: Human Mortality Database (2022).
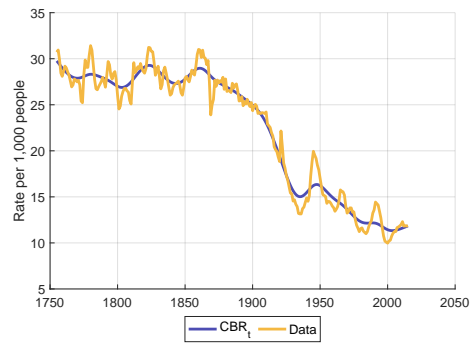
A – GDP
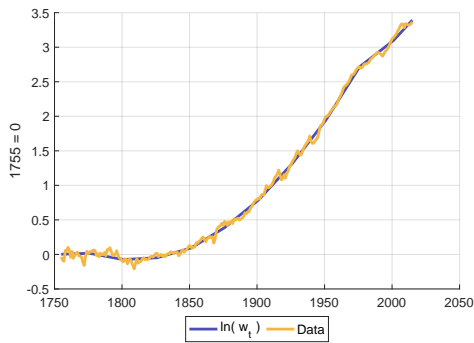
B – CBR

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

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

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 to age 5. This depends only on 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 time-invariant parameters to target a time series of length $T = 261$.

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, we take the initial age distribution, survival probabilities of modern and obsolete, and time series of GDP and CBR 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," recall that it has a compounding effect. We examine the quantitative role of κ in Section 3.4.

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.[4]

---

[4]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. Furthermore, the spikes are short-lived and do not affect the trend.
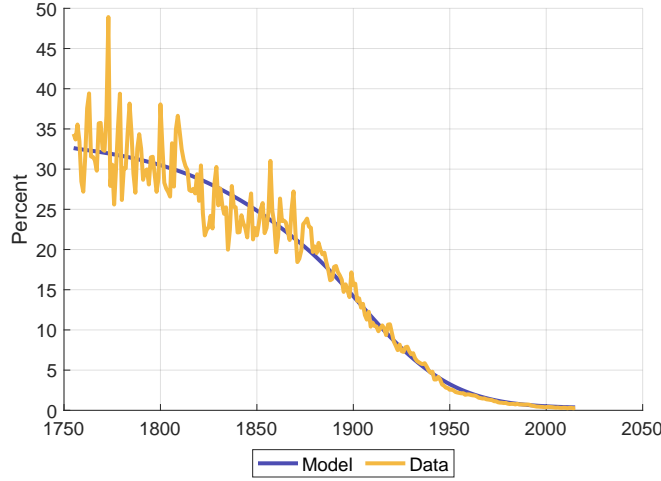
Figure 4: Child mortality rate in Sweden, model and data, 1755-2015

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

3.3 Non-targeted moments

Recall that age-specific mortality rates at time t in the model as well as in the data is a vector: $\{q_{t-j,j}\}_{j=0}^{110}$. For our calibration target, we have used the time series of $\{q_{t-j,j}\}_{j=0}^{4}$. To test the model, we examine several non-targeted moments; the model's fit for CMR does not imply a fit for the moments below.

1. Age-specific mortalities: Time series trends in $\{q_{t-j,j}\}_{j=5}^{110}$ are not the same as the trend in $\{q_{t-j,j}\}_{j=0}^{4}$ in the model and they are not the same in the data.

2. Age distributions, which depend on initial conditions, *all* age-specific mortalities, and CBR.

3. LEB: At each time t, it is a function of $\{q_{t-j,j}\}_{j=0}^{110}$, of which we have used only five elements, $\{q_{t-j,j}\}_{j=0}^{4}$, as targets.

4. LE_i at ages $i \geq 5$: At each time t, LE_i is a function of $\{q_{t-j,j}\}_{j=i}^{110}$, of which we have not used any elements as targets.
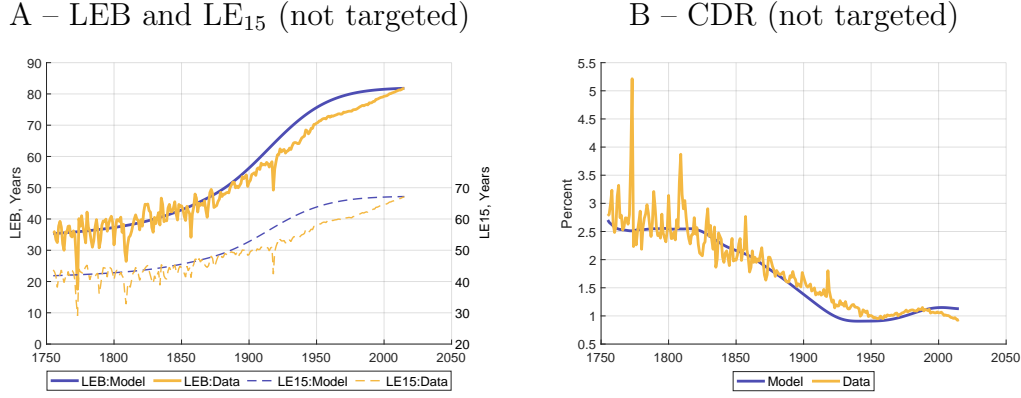
A – LEB and LE$_{15}$ (not targeted)　　　　B – CDR (not targeted)

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).

5. CDR: At each time t it is a function of $\{q_{t-j,j}\}_{j=0}^{110}$ and age distribution, of which we have used only five elements, $\{q_{t-j,j}\}_{j=0}^{4}$, as targets.

6. Population growth: A function of CDR and CBR time series.

Life expectancy at birth A widely used mortality statistic is LEB, defined in Equation (2). Panel A of Figure 5 shows that the model reproduces the trend in LEB.[5] Our model's mortality rates above age 4 could differ from those in the data and would imply discrepancies in the figure.

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.

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[5]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. Panel A of Figure 5 shows that the model reproduces the trend in LE$_{15}$ well.

[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.
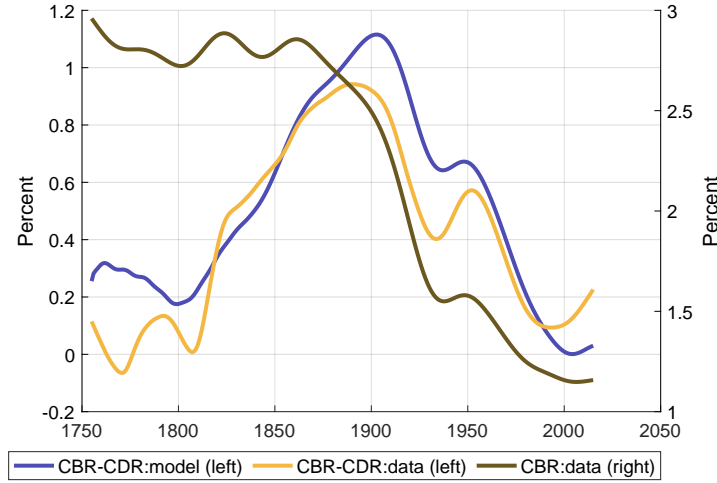
Crude death rate   CDR at time t, defined in (1), depends on age distribution and age-specific mortality at time t. Panel B of Figure 5 shows that the model reproduces the trend in CDR.

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. 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 19th 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, the model is consistent with different speeds of decline in mortality at other ages; in both the model and data,

CMR trend $\neq$ Age 10-14 mortality trend $\neq$ Age 30-34 mortality trend

$$\neq \text{Age 50-54 mortality trend.}$$

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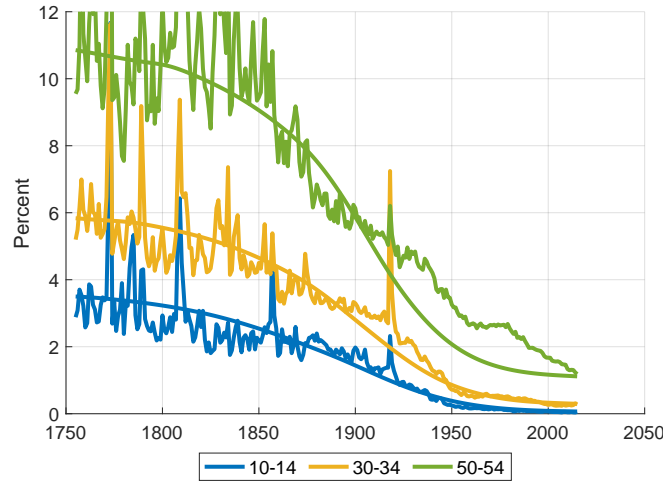
Figure 7: Age-specific mortality in Sweden (not targeted), model and data, 1755-2015

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.[7] We conjecture that older-age mortality is less in the model due to the fact that the relative price of health—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, we compute the model's implications using the survival probabilities in 1930 as the modern technology (before the dramatic rise in the relative price of health). This alternative is in line with the life expectancies at ages 30 and 50; see also Section 4.1.
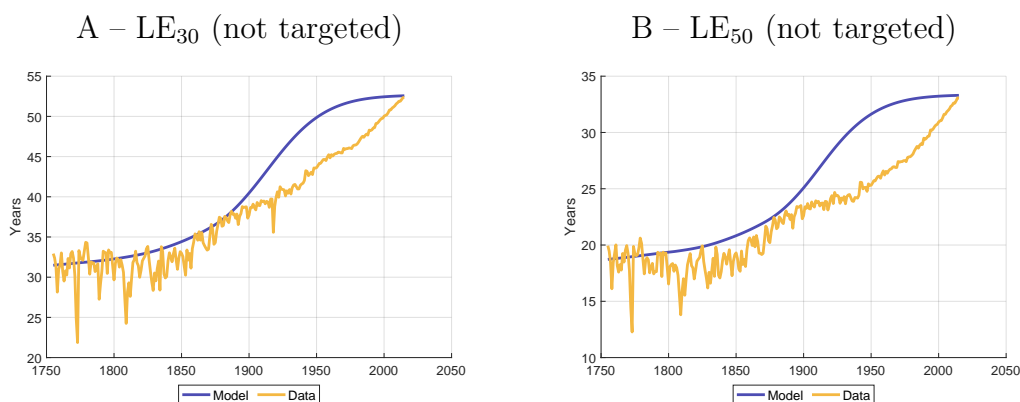
<div align="center">

A – LE$_{30}$ (not targeted)   B – LE$_{50}$ (not targeted)

</div>

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.

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[7]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.
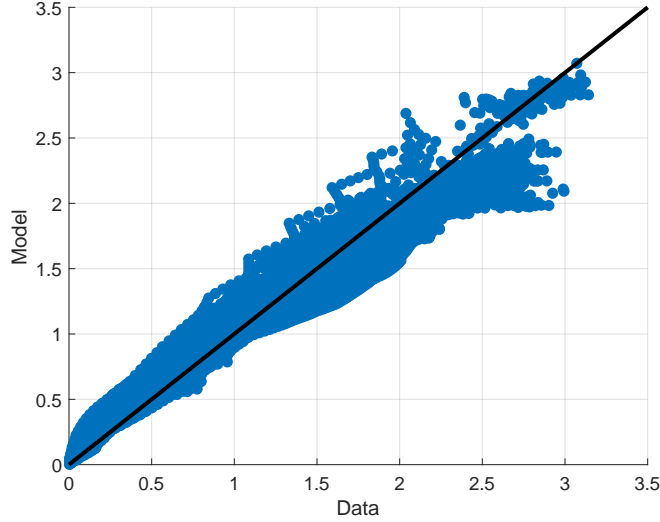
Figure 9: Age distribution in Sweden (not targeted), model and data, 1755-2015

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).

Recall that CDR is a function of age distribution and age-specific mortality. In Panel B of Figure 5 CDR in the model declines more rapidly than in the data starting around 1900. This raises a question whether the departure is due to age-specific mortality or age distribution. To answer this we plot Figure C.1 in Appendix C. This figure is a variant of Figure 9 that distinguishes between pre- and post-1900. In Figure C.2, we plot the age distribution for four different years: 1825, 1850, 1925, and 1950. The model's implications are roughly the same pre- and post-1900. So, the model-data gap in CDR post 1900 is due to age-specific mortality.

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 and 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.

$$\frac{p_{t,j+1}^m}{p_{t,j+1}^o} = \frac{p_{t,j}^m s_j^m + \kappa p_{t,j}^o s_j^o}{p_{t,j}^o (1-\kappa) s_j^o} = \frac{p_{t,j}^m}{p_{t,j}^o} \frac{s_j^m}{s_j^o} \frac{1}{1-\kappa} + \frac{\kappa}{1-\kappa} > \frac{p_{t,j}^m}{p_{t,j}^o}. \tag{16}$$

To quantify the role of individual choices, we assume $\pi_1 = \Lambda = 0$ in (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.[8]

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. CDR declines to a low of 0.91% in the 1950s in the baseline, whereas in the constant-income counterfactual, CDR declines to 1.16%. Thus, 86% $\left(\frac{2.7-1.16}{2.7-0.91}\right)$ 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. See also Section 4.5.

4   ALTERNATIVE MECHANISMS

In this section we discuss alternative mechanisms, such as medicine, infrastructure, nutrition, and schooling, that are often cited as explanations for mortality decline. It is conceivable that each mechanism could reduce mortality for specific diseases, in specific periods, in specific regions, and for specific age groups. Our focus, however, is on the long-run *trend* in various mortality statistics (independent of any specific disease) and population over more than two centuries for entire countries and for all age groups.

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[8]Equation (16) suggests that the proportion of modern increases with age in every cohort. Figure D.1 shows the evolution of the proportion of modern by age. At any point in time, the proportion tends to increase with age. At a cohort level, the proportion of modern among the 20-year-olds in 1800 is less than the proportion among the 60-year-olds in 1840 (8% and 17%, respectively). The same is true for the 20-year-olds in 1900 vs. the 40-year-olds in 1940.
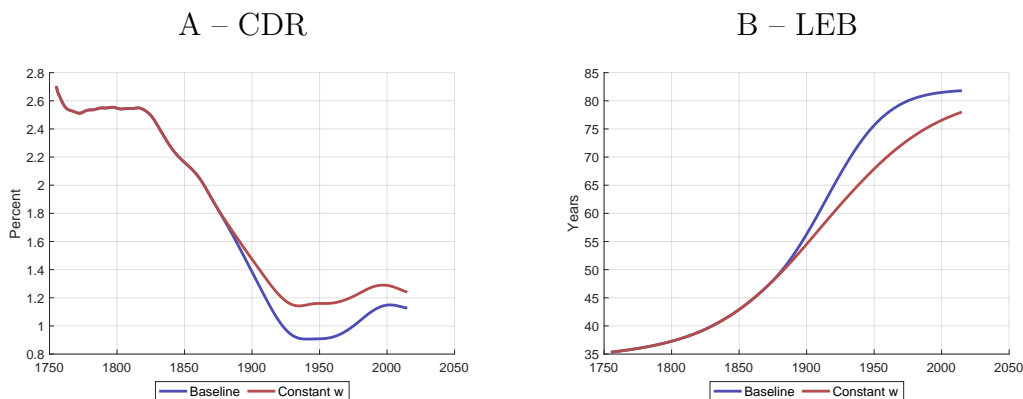
<center>A – CDR   B – LEB</center>

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

Source: Authors' calculations.

4.1 Medical theory of mortality

In our baseline in 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 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 use the same obsolete technology as in the baseline but use the 1930 survival probabilities as the modern technology, instead of the 2018 survival probabilities. We then choose π_1 and Λ to fit CMR data for 1755-1925 in Sweden, holding all other parameters at the baseline values (Table 1). Since we changed the modern technology, it is natural to adjust 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. By construction, medical innovations such

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).

as penicillin and sulfa drugs do not play a role in this exercise since these innovations appeared only in the 1930s. So, (i) these medical innovations do not play a role in the model's fit of the mortality statistics from 1755-1925 and (ii) there is no guarantee that this exercise would deliver the pre-1930 mortality in the data.

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

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.

Smallpox Consider the case of smallpox vaccine, discovered in the late 18th and early 19th centuries. First, Ager, Worm Hansen and Sandholt Jensen (2018) document that *smallpox mortality* had declined by 50% between 1750 and 1800, prior to the introduction of the vaccine (see their Figure 1). Second, they 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 mortality statistics such as CDR or LEB (Figures 4, 5, 7, and 8).
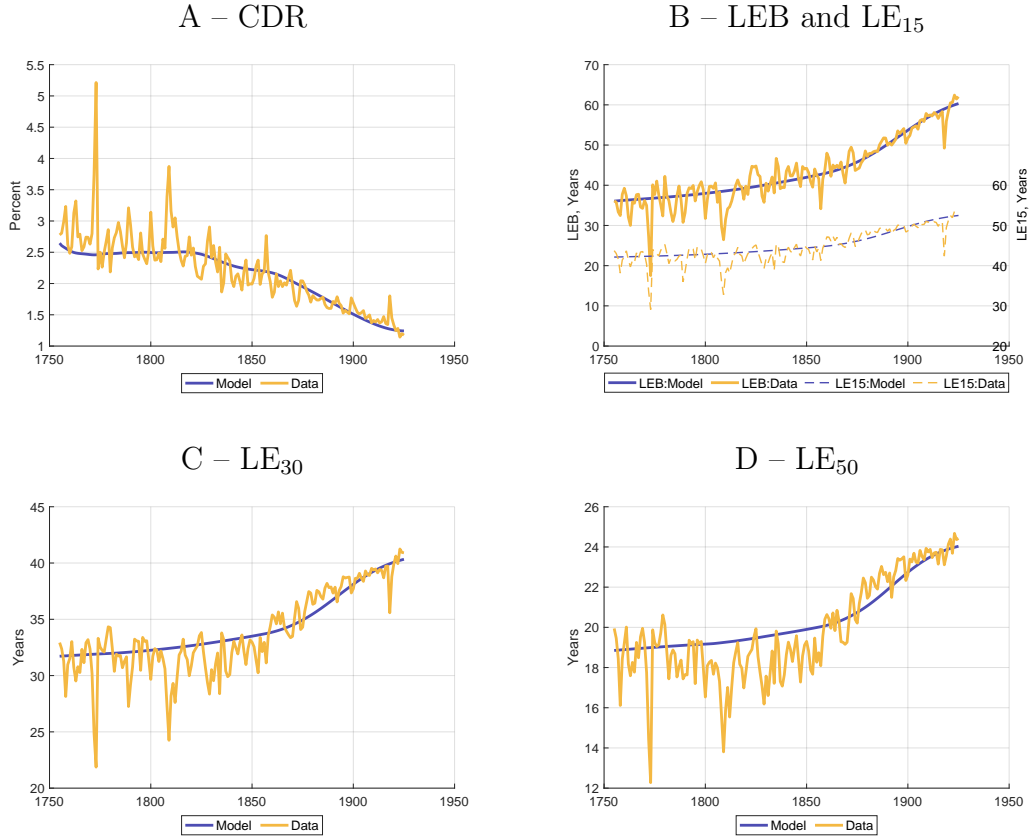
Figure 11: Non-targeted mortality statistics in Sweden before modern medicine, model and data, 1755-1925

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

4.2 Infrastructure theory of mortality

Another alternative to our theory is that public infrastructures, such as water and sewer systems, were the main drivers of mortality reduction. There are a few problematic issues with this alternative.

First, one might conjecture that since water and sewer systems are usually concentrated in cities, large populations might benefit from such systems and survive longer. In Swedish cities, 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. However, their Figure 3 documents that

the mortality decline in Swedish cities from just waterborne diseases over the same period is 90%, far in excess of 9%. Furthermore, deaths due to waterborne diseases are less than 10% of all deaths in Swedish cities (see their Figures 3 and A1).[9]

Second, Woods and Woodward, eds (1984) conclude that water and 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 the infrastructure, 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).

Third, in Sweden, water and sewer systems were introduced in the 1850s. But, 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, 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 (see Mokyr and Stein, 1997). And, Livi-Bacci (1991), Wrigley et al. (1997), Leukhina and Turnovsky (2016), and Delventhal et al. (2021), among others, have noted the minor role of income for mortality.

Similar to the smallpox vaccine in Section 4.1, mortality reductions in waterborne diseases are unlikely to have a substantial impact on the long-run trend in mortality statistics such as CDR or LEB.

4.3 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

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[9]While we could add a spatial dimension to our model, we do not have a long time series of mortality data for locations in Sweden with different densities, so we cannot study the density-mortality relationship, i.e., Farr's law (Brownlee, 1920).

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 4.2, the nutrition theory is also based on income to the extent that food production increases with economic development, so the critique in Section 4.2 applies. If improvements in nutrition are consequences of individuals' cleanliness and safe preservation of food items, then our theory applies.

4.4 Schooling theory of mortality

Schooling has been suggested as an important driver of mortality. For instance, Cervellati and Sunde (2015) assume that CMR and LE_5 are functions of past economy-wide schooling (and income). Our focus here is on long-run trends in mortality and life expectancy at all ages, population, and age distribution. We explain below why schooling is unlikely to be of first-order importance for these trends.

First, the channel for mortality decline in our model—diffusion of health practices—does not necessarily require schooling. In fact, the diffusion preceded the rise in schooling throughout Europe (Mokyr, 1993).

Second, in France, the Jules Ferry Laws on public education were introduced in 1881-82. However, LEB had increased from 26 in 1750 to 42 in 1850 (see Fogel, 2004, Table 1.1), an increase of 62% before the Jules Ferry Laws.

Finally, in De La Croix, Lindh and Malmberg (2008), the direction of causality is from mortality to schooling: It is the changes in longevity that affect the observed rise in schooling.

4.5 Role of 2018 technology

We use the vector of survival probabilities in 2018 to represent the modern technology. It could be the case that the 2018 survival probabilities are due to economic development, medicine, and infrastructure.

While we cannot assess *how much* of the 2018 survival probabilities is due to economic development, medicine, and infrastructure, we can study the implications of the model

during a time period in Sweden (i) when GDP did not grow (Panel A of Figure 3), (ii) before Germ Theory, and (iii) before the construction of water and sewer infrastructure. The period we have in mind is 1750-1850. In the data, the period 1750-1850 accounts for 43% of the CDR decline and 36% of the CMR decline, relative to the entire sample period 1750-2018.

Suppose that the obsolete technology is the same as that in the baseline, but the modern technology is the vector of survival rates in 1850, instead of the baseline 2018 survival probabilities. We adjust π_1 and λ, holding the other time-invariant parameters at their baseline values, so that the model delivers CMR from 1755 to 1845. The model's implications for mortality over this time period are practically the same as those in the baseline. With the 1850 survival probabilities, CDR declines from 2.7% in 1755 to 2.1% in 1845, while in the baseline CDR declines from 2.7% to 2.2% over the same period.

This result is consistent with the evidence. The onset of CMR reduction is a century before the Industrial Revolution started in Sweden (Jörberg, 1965). GDP growth as a channel of first-order importance for the long-run trend in mortality has been dismissed by Livi-Bacci (1991) for Europe and Wrigley et al. (1997) and Leukhina and Turnovsky (2016) for England. More recently, Becker, Philipson and Soares (2005) and Acemoglu and Johnson (2007) have noted the cross-country convergence in LEB in post World War II data despite the absence of income convergence. Furthermore, major causes of death had disappeared before medical cures and vaccines became widely available, as noted by McKeown, Brown and Record (1972), Mokyr and Stein (1997), and Riley (2005a).

5 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 E.

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:

$$\frac{w_{t_i}^i}{w_{t_i}^{\text{Sweden}}} = \frac{\text{GDP}_{t_i}^i}{\text{GDP}_{t_i}^{\text{Sweden}}}, \tag{17}$$

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).
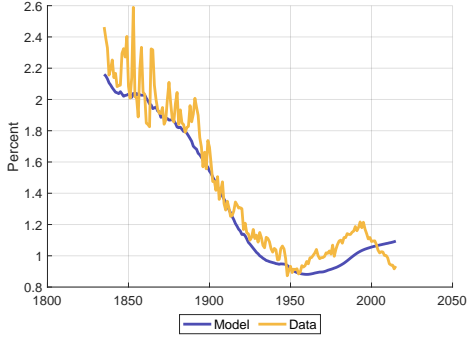
We choose π_1 and Λ such that country i's CMR implied by the model best reproduces the observed CMR, holding all other parameters at the values calibrated for Sweden (Table 1). That is, only π_1 and Λ are country-specific. As noted in Section 2.3, we can use country-specific π_1 to account for the fact 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 12 displays the model's fit for the 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. .

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 (17). We then represent world GDP growth as we did for Sweden. The obsolete and modern technologies are the same as Sweden's. The initial age distribution is
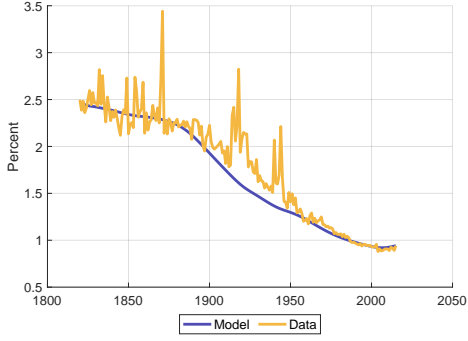
A – CDR, Denmark (1835-2015) B – LEB and LE$_{15}$ Denmark (1835-2015)
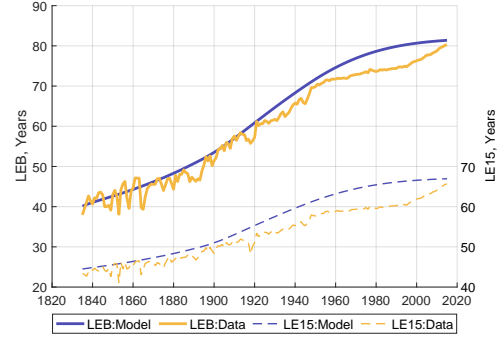
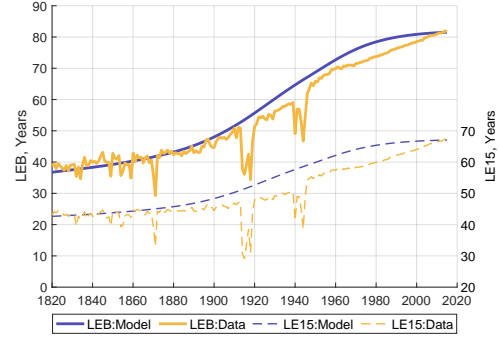C – CDR, France (1820-2015) D – LEB and LE$_{15}$ France (1820-2015)

Figure 12: 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).

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 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 $= 0.99$.
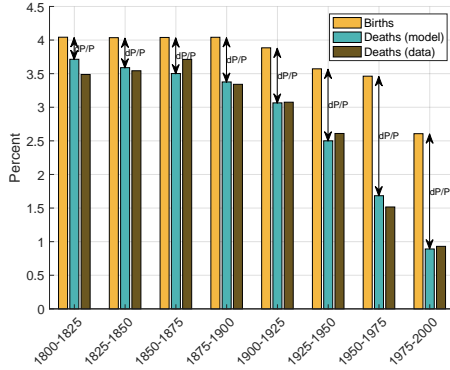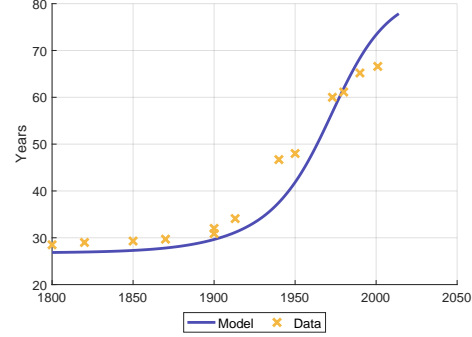
Figure 13: World population growth and life expectancy at birth, 1800-2000

Source: Panel A: Authors' calculations and https://www.gapminder.org/data/. Panel B: Authors' calculations, Riley (2005b, Table 1), and Acemoglu and Johnson (2007, Table 1).

Panel A of Figure 13 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 13 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).

Non-mortality moment   So far we have discussed our model's performance using untargeted mortality and age distribution statistics. Is there any suggestive evidence of adoption of individual-level practices, such as washing hands and using soap? Recall that individual decisions on adoption are the key to higher survival rates in our model. Table F.1 illustrates soap consumption per capita over time in Great Britain. As is evident, the practice became more prevalent: The soap consumption more than doubled between the middle of the 18th century and World War I.

6 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. 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.

A – GDP elasticity of CDR B – GDP elasticity of LEB
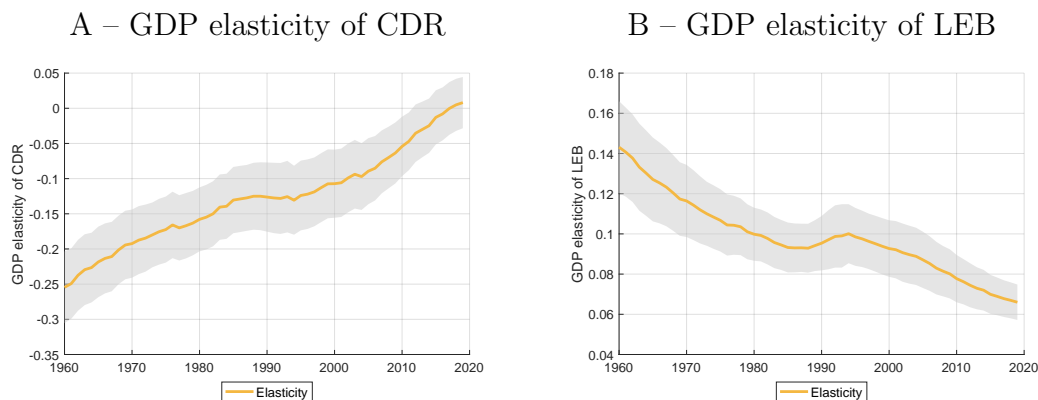

Figure 14: Convergence in mortality

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 Development Indicators (2024) 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 cross-country convergence of CDR and life expectancies despite the lack of income convergence—see Figure 14; and (iii) the large contribution of poor countries to world population growth, despite the decline in fertility in poor countries.

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, as in Eckstein et al. (1999), Doepke (2005), Bar and Leukhina (2010), and Albanesi and Olivetti (2016), 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.[10]

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 of economic transition from Malthusian stagnation to modern growth.

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[10]Such a model could account for demographic transition in the sense of Lee (2003), who describes it in terms of dynamics of mortality, fertility, population, and age distribution.

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A   POPULATION STATISTICS

Recall that the crude death rate at time t is

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

and that life expectancy at age j is the expected additional years of life for age-j individuals if they experience the age-specific mortality observed at t:

$$\text{LE}_{t,j} = 1 - q_{t-j,j} + (1 - q_{t-j,j}) \, \text{LE}_{t,j+1}. \tag{A.2}$$

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 (A.1), we see that $\text{CDR}_t = q_t$ and does not depend on the age distribution. Using the recursive formula (A.2),

$$\text{LEB}_t = (1 - q_t) + (1 - q_t)\text{LE}_{t,1} = (1 - q_t) + (1 - q_t)\left[(1 - q_t) + (1 - q_t)\text{LE}_{t,2}\right].$$

Iterating forward yields

$$\text{LEB}_t = \frac{1 - q_t}{q_t}.$$

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 both CDR and life expectancies.

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,

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

Note that, at the optimum,

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

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

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

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 Age distribution decomposed over time

In this appendix, we enrich Figure 9: The blue dots in Figure C.1 represent the years 1755 to 1900, and the orange dots represent 1901 to 2015. The model's performance is not systematically worse for the last 115 years compared with the first 145 years.
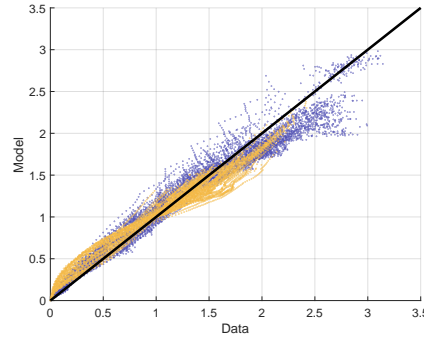
Figure C.1: Age distribution in Sweden, model and data, 1755-2015

Note: The blue dots represent the years 1755 to 1900 and the orange dots represent 1901 to 2015.
Source: Human Mortality Database (2022) and authors' calculations.

Going beyond Figure C.1, Figure C.2 illustrates the age distribution for four different years: 1825, 1850, 1925, and 1950 (two in the first 150 years and two in the last 118 years.) First, overall, the model's implications are consistent with the data. Second, the fit in 1825 and 1850 is about the same as the fit in 1925 and 1950.
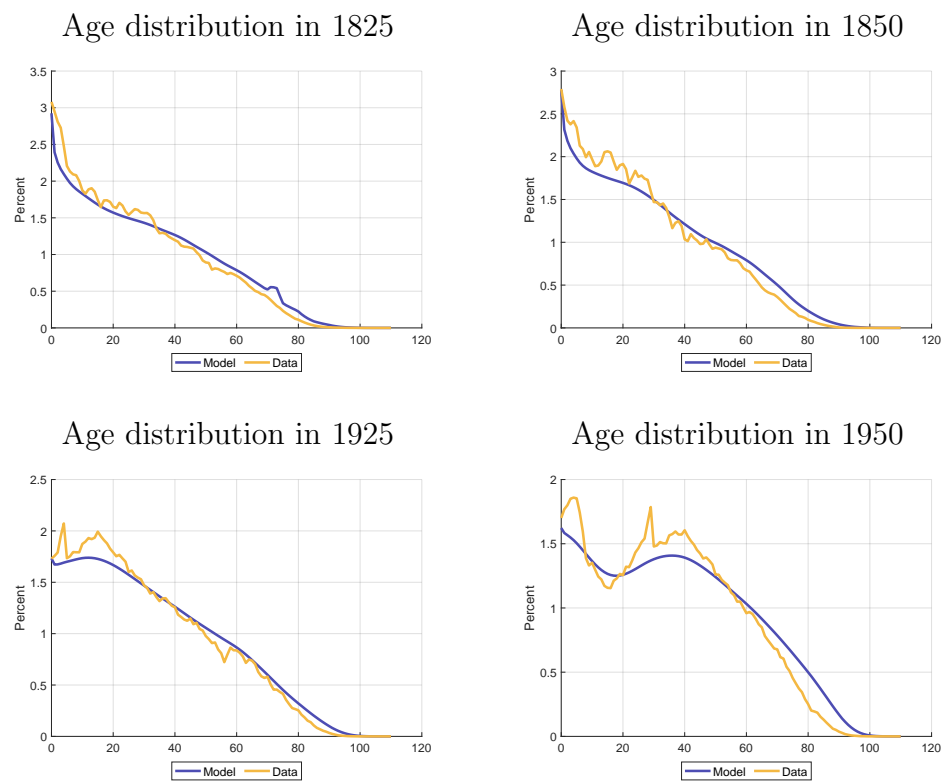
Age distribution in 1825

Age distribution in 1850

Age distribution in 1925

Age distribution in 1950

Figure C.2: Age distribution, model and data

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

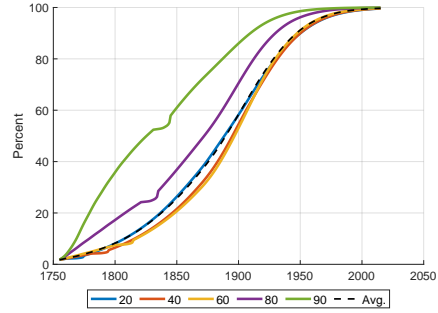# D THE PROPORTION OF MODERN BY AGE AND BY YEAR

Figure D.1: Proportion of modern in the baseline–Age-specific and average

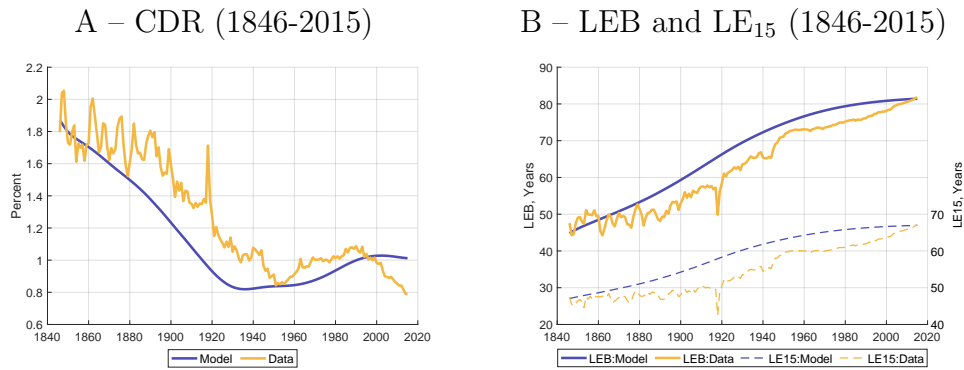*Source*: Authors' calculations.

E WESTERN EUROPE

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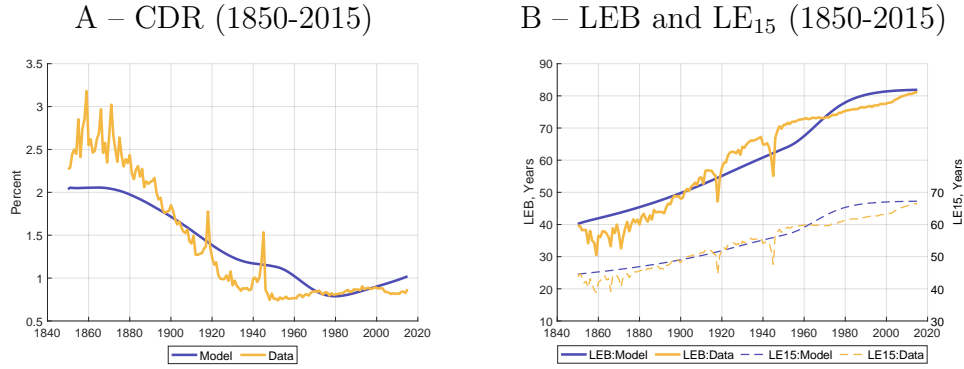*Source*: Authors' calculations and Human Mortality Database (2022).

A – CDR (1850-2015)

B – LEB and LE$_{15}$ (1850-2015)

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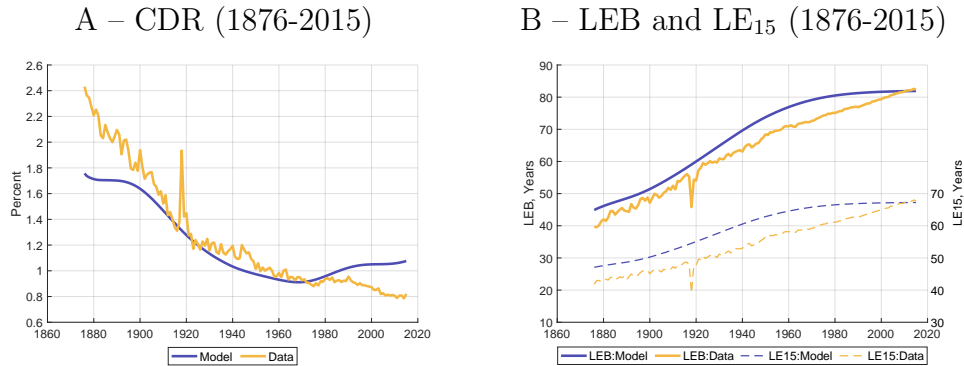*Source*: Authors' calculations and Human Mortality Database (2022).

A – CDR (1876-2015)

B – LEB and LE$_{15}$ (1876-2015)

Figure E.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).

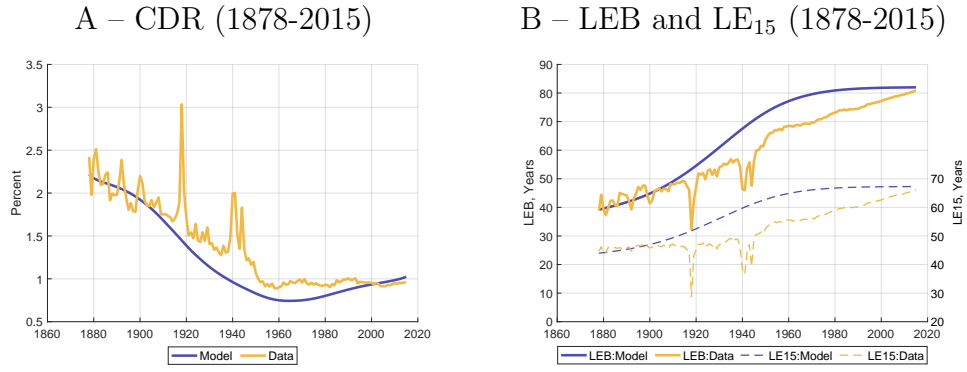A – CDR (1878-2015)   B – LEB and LE₁₅ (1878-2015)

Figure E.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).

F   The consumption of soap

Table F.1: Annual consumption of soap per capita in Great Britain

Year	1851	1861	1871	1881	1891	1901	1912
Per-capita soap consumption, pounds	7.1	8.0	10.7	14.0	15.4	17.4	18.0

Source: Mokyr and Stein (1997, Table 4.1)