

# The Role of Mortality in the Transmission of Knowledge\*

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We investigate, both theoretically and quantitatively, a previously unexplored link between gains in adult mortality and productivity growth. Our mechanism allocates a central role to individuals as carriers of useful ideas and to personal contact as an important means of transferring these ideas. It thus implies that disrupting a human life impedes the process of knowledge transmission across time. We derive a simple and intuitive form of the dependence of aggregate knowledge transfer on adult mortality and incorporate it into a model of endogenous growth. We then quantitatively examine the relevance of the proposed link in application to the long-run growth experience of England. Our main finding is that the reduction in adult mortality, by improving knowledge transmission across time and encouraging more innovation, accounted for one third of the takeoff in output per capita.

**Keywords:** Economic growth, total factor productivity, adult mortality, longevity, knowledge transmission, ideas, human capital

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# 1. Introduction

Motivated by the strong positive association between gains in longevity and growth, found both in time series and cross-sectional studies (e.g. Kelley and Schmidt (1995), Shastry and Weil (2003), Lorentzen et al. (2005), Aghion et al (2009)), we investigate a previously unexplored link between adult mortality and productivity growth.<sup>1</sup>

Our mechanism is rooted in the premise that in preindustrial and traditional societies, a large body of useful knowledge was tacit, uncodifiable and embodied collectively in individuals. An effective transfer of tacit knowledge requires extensive personal contact. In fact, we observe that most learning in traditional societies was accomplished through master-apprentice and parent-child relationships. In a society where knowledge is embodied in individuals and personal contact is essential for transferring knowledge, the premature death of a parent (master) has two negative implications. (1) It interrupts the skill transfer to their children (apprentices), and (2) it implies a loss of their own contribution to aggregate productivity, comprised of their knowledge and expertise.<sup>2</sup> These factors represent the direct impact of mortality on productivity growth. In addition, the anticipation of the low rate of knowledge transmission across time characterizing high mortality regimes discourages investment in productivity, representing an indirect impact of mortality. Our main hypothesis is that the reduction in adult mortality facilitated the takeoff in productivity growth by improving knowledge transmission across time and by encouraging more innovation.

Episodes of high mortality are historically known to disrupt the process of knowledge transmission. Recorded episodes of such losses of useful knowledge due to mortality are numerous. Archaeological findings reveal that the bubonic plague and the scarlet fever epidemic that greatly depopulated the Senecas in the 1630s eliminated much of their knowledge of ceramic craftsmanship (Halverson 2007). Even though HIV, unlike preindustrial diseases and epidemics, kills people slowly, the AIDs epidemic in Sub-Saharan Africa also appears to have disrupted the transmission of indigenous farming practices to children.<sup>3</sup> In fact, Waterhouse (2005) documents a large negative impact of HIV/AIDS on farmers' knowledge in the identification of seed through conducting farmer surveys in several provinces of Mozambique. The negative impact of mortality on local productivity in preindustrial times in Europe is evidenced by the well-documented guilds' practice of sending their skilled craftsmen to regions that had recently experienced an epidemic. At a macro level, there are several preindustrial records of large-scale mortality episodes interfering with productivity growth spurts: for example, Black Death in the case of 14th c. Europe, wars with the Ottoman empire in the case of the Italian Golden Age and Jurchen and Mongol invasions in the case of Sung China (Monteiro and Pereira 2007). Several more historical episodes of technological regress following a population decline are discussed in Aiyar et al. (2008), who also attempt to understand why high mortality episodes often lead to the loss of knowledge.<sup>4</sup>

<sup>1</sup>Acemoglu and Johnson (2007) find no evidence that increases in output per capita levels are associated with increases in life expectancy. However, as shown by Aghion et al (2009), if one includes the level effect of lifetime expectancy in addition to its growth effect, then the same dataset and methodology yield a positive dependence of output per capita growth on life expectancy and its growth. Consistently with these findings, our model incorporates both the level and the growth effects.

<sup>2</sup>Our explicit modeling of mortality interfering with the process of knowledge transfer across individuals by diminishing the amount of personal contact in learning relationships, i.e. the first effect, is closely related to the idea developed in Lucas (2009). However, while in Lucas (2009), everyone gets a productivity draw, and high mortality limits the number of personal contacts with others that can help raise one's productivity, we assume that one learns from their parents and high mortality limits the amount of personal contact with the parents.

<sup>3</sup>This information was collected from [www.un.org](http://www.un.org) and [www.fao.org](http://www.fao.org).

<sup>4</sup>While we focus on the disruption of knowledge transfer as a direct consequence of an epidemic, this paper analyzes a different reason for why some knowledge may be forgotten forever. A smaller population size, and hence a lower aggregate demand, imply that it is not profitable to produce some of the varieties. These production techniques are not passed on to the future generations. Note that this mechanism is also built on a premise of embodied knowledge.

We first derive the impact of adult mortality on productivity, starting with reasonable microeconomic assumptions regarding the process according to which embodied ideas are transferred across time and regarding the mapping of these ideas into aggregate productivity. We obtain a simple and intuitive form of the adverse impact of adult mortality on productivity growth that captures the ideas described above. We derive that the destruction rate of productivity is an increasing and convex function of adult mortality. This convexity arises due to the assumption of diminishing marginal returns to experience associated with a given idea and to the number of carriers of this idea. We then embed this impact of adult mortality on productivity transmission into a general equilibrium model of endogenous technological change.<sup>5</sup> We allow for knowledge diffusion across regions in order to moderate the adverse impact of a high realization of a region-specific mortality shock. Innovation and improvement of existing techniques is carried out by young adults, whose sole motivation for doing so is greater productivity in the future, should they remain alive.

To assess the quantitative relevance of our hypothesis, we apply our model to study the case of the English takeoff of output per capita, focusing on the historical period from 1680 to 1880, the midpoint of which dates the beginning of the Industrial Revolution.<sup>6</sup> We first parameterize the model using the method of moments and the assumption of balanced growth to capture the available economic and demographic observations for England around 1600-1700. We employ the historical data on real wage dynamics following the Black Death epidemic in England to identify the key parameter in the knowledge destruction function. The Black Death is the perfect case study, as it entailed an unexpected disease, with a short period of illness, followed by death – exactly the situation that should lead to low rates of knowledge transfer over time. Taking as given fertility rates and the time varying parameters of the mortality shock process, estimated using the actual time series data on age-specific mortality, we quantitatively study the role of gains in adult longevity in the takeoff from stagnation to growth. Figure 1 reports the actual time series of adult mortality (ages 25-50) alongside output per capita, revealing the positive relation between gains in adult longevity and growth.

We find that the decline in adult mortality generates about 3/4 of the rise in the log of output per capita observed during the two hundred year period that we study. Moreover, the influence it exerted through the knowledge transmission mechanism proposed here, accounts for one third of the empirical rise in the log of output per capita, working through both, the direct effect and the indirect effect on productivity investments. Our findings thus suggest that, in addition to their more conventional effects, reductions in adult mortality contribute to the process of development by extending the amount of personal contact in learning relationships and thereby improving the process of knowledge transmission across generations.

We believe that our model, built on the notion of embodied knowledge, is suitable for examining the period up to 1880. Mokyr (2002) argues that “most practical knowledge in the 18th c. was informal, often uncoded and passed on vertically from master to apprentice or horizontally across agents... Printed text might have remained secondary to personal contact throughout the 19th c....”, David Mitch, summarizing a number of historical sources, concludes that informal human capital accumulation was at the heart of the Industrial Revolution: “technological advance [in the 19th c.] was primarily due to the practical experience by men of little or no formal scientific training.” He further

<sup>5</sup>Bar and Leukhina (forthcoming), using price data, estimates large productivity changes during 1600-1910 and finds that these changes were largely responsible for the economic transformation of England. Hence, we focus on changes in productivity, abstracting from physical capital here.

<sup>6</sup>It is important that we are able to use age-specific mortality data. The reason that we focus on England is that such data is not available for other countries.

documents very small gains in literacy rates in 19th c. England (Mitch 1998).

19th c. England is often associated with a number of developments, other than gains in adult mortality, that could potentially improve the speed and efficiency of knowledge transfers, such as the rise in informal communication and learning networks and scientific communities, the rise in population density, which would make idea exchange easier, the decline in transportation and post costs, and a movement away from small family farms towards larger-scale production (Mokyr 2002). In the context of our framework, these developments would (1) further reduce the negative impact of adult mortality on knowledge transfer across time and hence reinforce the mechanism examined here and (2) increase the diffusion of knowledge across regions. We explore the potential effects of increasing knowledge diffusion across regions in Section 3.4.1, by modeling the rate of absorption of the gap between the local and frontier technology as positively dependent on population density.

Finally, we place our work in the context of existing literature on takeoffs. By modeling the impact of mortality on knowledge transmission, we contribute to the previous work that emphasized the important role of gains in life expectancy in the process of output per capita takeoff. Such papers include Ehrlich and Lui (1991), Kalemli-Ozcan et al. (2000), Kalemli-Ozcan (2002), Boucekkine et al. (2002, 2003), Lagerlöf (2003a, 2003b), Cervellati and Sunde (2005), Soares (2005) and Tamura (2006) among others.<sup>7</sup>

Two mechanisms linking declines in mortality to increases in growth are commonly used. First, a decline in child mortality and/or its uncertainty reduces parental costs of educating each surviving child, inducing greater investments in children's human capital, and in turn, leading to a takeoff in output per capita and a drop in fertility through the quantity-quality trade-off. The timing of reductions in adult mortality, however, is better aligned with the period of takeoff than the timing of reductions in child mortality. For the case of England, for example, output per capita accelerates around 1800, while child mortality declines at the very end of the 19th c. (Figure 1).

The second mechanism encountered in the literature is one whose logic dates back to Ben-Porath (1967); it has become the conventional wisdom regarding the causal effect from gains in life expectancy to growth. Gains in life expectancy increase the expected period over which investments in human capital are paid off, consequently encouraging more human capital accumulation and growth. This particular link has been recently criticized by Hazan and Zoaby (2006) and Hazan (2009). The latter paper documents that for cohorts of American men born in 1840-1970, the labor input declined despite the dramatic gains in life expectancy. The author then argues that because a rise in the lifetime labor supply is a necessary implication of the Ben-Porath type model that he examines, gains in longevity could not have caused human capital accumulation and hence growth via the Ben-Porath mechanism. Although a closer look into more general Ben-Porath type frameworks is warranted to determine whether or not they can maintain the causal link from gains in longevity to growth in the face of a declining labor supply, the model proposed here, which also incorporates the Ben-Porath channel, certainly can; i.e., it reconciles Hazan's findings with the possibility of causation from gains in longevity to growth. First, the direct impact of the decline in adult mortality on knowledge retention over time and consequently on growth, is independent of the lifetime labor supply. Second, the indirect impact of the decline in adult mortality on growth is through increasing innovation time. This effect can exist even in the presence of a declining lifetime labor supply, because falling mortality directly raises the return to human capital accumulation by improving knowledge transmission over time, i.e. by raising the productivity of time spent innovating.<sup>8</sup>

<sup>7</sup>Servellati and Sunde (2005), Lagerlöf (2003a, 2003b), Tamura (2006) endogenize mortality.

<sup>8</sup>We should point out that there exist other mechanisms that can raise returns to human capital accumulation even in the presence of a declining lifetime labor input. For example, the idea that productivity of human capital production increases in population density, developed

Because we show that the existence of a high adult mortality regime can contribute to long periods of stagnation, our mechanism is also complementary to the Malthusian mechanism, the conventional explanation for long periods of stagnation.<sup>9</sup> Recently, this effect has been subjected to quantitative analysis by Crafts and Mills (2007) and Ashraf and Galor (2008). The effect of a temporary and unexpected high mortality shock introduced in the model proposed here is consistent with a positive Malthusian check. Despite its negative impact on productivity, it raises output per capita, because it raises per capita land holdings.

Finally, because we endogenize gains in productivity, our work also complements existing work that emphasizes the importance of technological progress in driving the economic transformation (e.g. Galor and Weil (2000), Fernandez-Villaverde (2001), Jones (2001), Hansen and Prescott (2002), Greenwood and Seshadri (2002), Doepke (2004), Bar and Leukhina (forthcoming)).

The rest of the paper is organized as follows. In Section 2, we present the environment, including the microfoundations of the knowledge destruction rate, and equilibrium analysis. In Section 3, we present our quantitative analysis. This includes estimating the time-dependent parameters of the mortality shock distribution, calibrating the model, performing the experiments and sensitivity analysis. We conclude in Section 4.

## 2. The Benchmark Model

Time is discrete and indexed by  $t = 0, 1, 2, \dots$ . There are  $J$  regions within the country, indexed by  $j \in \{1, 2, \dots, J\}$ , each endowed with  $\Lambda_j$  acres of land, total land is given by  $\Lambda = \sum_j \Lambda_j$ . We model several regions to moderate the adverse impact of a region-specific epidemic. We represent total population in region  $j$  at time  $t$  by  $N_{t,j}$ , and the total country population by  $N_t = \sum_j N_{t,j}$ . In each region, there is a representative dynasty.<sup>10</sup> Every generation lives for at most three periods, as a child, a young adult and an old adult, indicated by the superscripts  $c$ ,  $y$  and  $o$ . The populations of children, and young and old adults in region  $j$  at time  $t$  are denoted by  $N_{t,j}^c$ ,  $N_{t,j}^y$  and  $N_{t,j}^o$ .

### 2.1. Laws of Motion for Population and Land

We intentionally abstract from modeling endogenous fertility and focus on the period prior to the fall in birth rates, the beginning of which dates to 1880. The main reason for doing so is to ensure that the model closely matches the population dynamics and hence the evolution of land per capita, which is a crucial factor behind growth in output per capita.<sup>11</sup>

In each period, random fractions of children and young adults in region  $j$ , denoted by  $m_{t,j}^c$  and  $m_{t,j}^y$ , do not survive to the next period. Similarly to Lagerlöf (2003a, 2003b), we model mortality rates as functions of a log-normally distributed random variable,  $\ln(\omega_t) \sim N(\mu_t^\omega, \sigma_t^\omega)$ , drawn at the end of each period. More precisely, we assume that

in Lagerlöf (2003a, 2003b), could be adopted to generate a causal link from gains in adult longevity to growth.

<sup>9</sup>See Galor (2005) for the description of the Malthusian epoch.

<sup>10</sup>Alternatively, we could reinterpret the setup as consisting of  $J$  representative dynasties within a single location.

<sup>11</sup>In addition, since the probability of a 25 year old surviving to 50 was already as large as 95% by 1880, there is no reason to believe that changes in adult mortality could contribute to the fall in birth rates. We find it more plausible that the decline in birth rates in the late 19th c. can be attributed to factors such as compulsory education and child labor reforms (Hazan and Berdugo 2002, Doepke and Zilibotti 2005) or the change in the nature of the technological progress to being more skill-biased (Galor 2005). Previous versions of this paper did incorporate endogenous fertility and the time cost of raising children, but we found that their inclusion added little to the main message of the paper.

the realized mortality rates are given by

$$m_{t,j}^y = m^y(\omega_{t,j}) = \frac{\omega_{t,j}}{1 + \omega_{t,j}} \text{ and } m_{t,j}^c = \zeta_t m^y(\omega_{t,j}), \quad (1)$$

where  $\omega_{t,j}$  denotes region- and time-specific realization of mortality shock  $\omega_t$ , and  $\zeta_t$  captures the difference in trends of child and adult mortality. Thus, the survival rate in region  $j$  is a strictly decreasing function of  $\omega_{t,j}$ . Note that the distribution parameters for  $\omega_t$  are indexed by time, and will be estimated, along with the time series for  $\zeta_t$ , to match age-specific mortality data.<sup>12</sup> While all regions possess identical  $\mu_t^o, \sigma_t^o, \zeta_t$ , they will differ in mortality shock realizations.

The following relationships are implied

$$N_{t+1,j}^y = (1 - m_{t,j}^c) N_{t,j}^c, \quad (2)$$

$$N_{t+1,j}^o = (1 - m_{t,j}^y) N_{t,j}^y, \quad (3)$$

$$N_{t,j}^c = n_t N_{t,j}^y, \quad (4)$$

where  $n_t$  denotes fertility, assumed to be exogenous and the same across regions.

The above three relations imply the following evolution of old and young adult populations:

$$N_{t+1,j}^y = (1 - m_{t,j}^c) n_{t,j} N_{t,j}^y, \quad (5)$$

$$N_{t+1,j}^o = (1 - m_{t,j}^y) N_{t,j}^o (1 - m_{t-1,j}^c) n_{t-1,j} / (1 - m_{t-1,j}^y). \quad (6)$$

We assume that land is jointly and equally owned by young and old adult cohorts:

$$\lambda_{t,j}^y = \frac{0.5\Lambda_j}{N_{t,j}^y}, \quad (7)$$

$$\lambda_{t,j}^o = \frac{0.5\Lambda_j}{N_{t,j}^o}. \quad (8)$$

This assumption, together with (3), implies that the land holdings of the young adults surviving to old adulthood is incremented with those of non-surviving members of their cohort, thus capturing the link from high mortality realization to an increase in resources:

$$\lambda_{t+1,j}^o(\omega_{t,j}) = \frac{\lambda_{t,j}^y}{1 - m^y(\omega_{t,j})}. \quad (9)$$

## 2.2. Technology and the Law of Motion of Total Factor Productivity

The total factor productivity (TFP) in region  $j$  at time  $t$  is denoted by  $A_{t,j}$ . The production technology is given by  $A_{t,j} L_{t,j}^\theta \Lambda_j^{1-\theta}$ , where  $L_{t,j}$  is the labor input,  $\Lambda_j$  is land input, and  $\theta \in (0, 1)$ .

<sup>12</sup>Lagerlöf (2003) instead keeps the distribution parameters for  $\omega$  fixed, but allows the constant in the denominator to decrease as the amount of human capital in the economy increases. In that model, only two periods of life are assumed, and hence it is the mortality in the first period of life (eliminating those who do not make decisions) that is modeled. By contrast, we model both child and young adult mortality, focusing on the causal link from young adult mortality to growth.

We assume that during the period, TFP can be augmented with diffused knowledge from other regions  $d_{t,j}$  and an increment due to a time consuming process of innovating  $A_{t,j}i_{t,j}^\eta$ , where  $i_{t,j}$  denotes the average time spent innovating:  $A_{t,j}^{end} = A_{t,j} + A_{t,j}i_{t,j}^\eta + d_{t,j}$ ,  $\eta \in (0, 1)$ .<sup>13</sup> Note that it is assumed that innovation results from building on what is already known.<sup>14</sup> There is extensive evidence that in preindustrial time, landlords and tenant farmers actively engaged in the costly process of improving their methods of production: Macdonald (1979), using postal records, uncovers a great variety of methods farmers used for that purpose, such as traveling and corresponding with other farmers, sending their children to another farm for a year to master a more advanced technique, and attempting new techniques on their own farms. In fact, Macdonald (1979) reports that at the end of the 18th c., farmers were willing to pay as much as 60 pounds, or roughly twice the annual farm wages, to spend a season and work on a more progressive farm learning a better technique of production.

While the innovation increment and diffusion terms can be borrowed from the growth literature, the challenge is to motivate a reasonable form of future TFP dependence on the realization of young adult mortality  $m_{t,j}^y$ , i.e.  $A_{t+1,j} = g(A_{t,j}^{end}, m_{t,j}^y)$ . We aim to capture the idea that premature death interferes with the intergenerational knowledge transmission, and hence adversely impacts future TFP. To do so, we follow Lucas (2009) and start with the premise that all ideas and techniques reside in individual heads. These embodied ideas, through complex interactions among their carriers, determine TFP level at any point in time. In what follows, we lay out the microfoundations of idea transmission from parents to children and of the TFP dependence on the stock of ideas and their carriers. The advantage of our approach, relative to directly assuming a particular functional form  $g(\cdot)$ , is that it allows us to be explicit about the assumptions that imply it.

Suppose that useful ideas (or production techniques) known collectively at the end of the period are indexed by  $i = 1, 2, \dots, I_{t,j}$  and vary by quality  $q_{t,j}(i)$ . A person can embody any number from 0 to  $I_{t,j}$  of useful ideas. The number of young and old adult carriers of idea  $i$  are denoted by  $N_{t,j}^y(i)$  and  $N_{t,j}^o(i)$ . Assume that TFP level at any point of time is a Dixit-Stiglitz aggregate of individual idea contributions  $a_{t,j}(i)$ . We assume that although ideas and knowledge are embodied in private individuals, they aggregate to yield a publicly available (non-rival) TFP. Thus, the end-of-period TFP is given by

$$A_{t,j}^{end} = \left[ \sum_{i=1}^{I_{t,j}} a_{t,j}(i)^\rho \right]^{1/\rho}.$$

An idea-specific contribution  $a_{t,j}(i)$  depends positively on its quality  $q_{t,j}(i)$ , the fraction of productive population that embodies this idea,  $\frac{N_{t,j}^y(i) + N_{t,j}^o(i)}{N_{t,j}^y + N_{t,j}^o}$ , and a factor reflecting the available experience associated with using this idea in production, which, for simplicity, we assume to be the fraction of the old adult carriers of idea  $i$  in total productive population. The later dependence aims to capture the idea that the older generation's expertise facilitates the application of a particular technique by its current carriers. Having the experience of the elder to draw upon is particularly important in farming economies (Rozenzweig and Wolpin, 1996). Precisely,

$$a_{t,j}(i) = q_{t,j}(i)^{1-\phi} \left[ \left( \frac{N_{t,j}^o(i)}{N_{t,j}^y + N_{t,j}^o} \right)^\mu \left( \frac{N_{t,j}^y(i) + N_{t,j}^o(i)}{N_{t,j}^y + N_{t,j}^o} \right)^{1-\mu} \right]^\phi,$$

<sup>13</sup>We will discuss the form of  $d_{t,j}$  later.

<sup>14</sup>The motivation for this assumption can be found in papers by Weitzman (1998) and Olsson (2000) that model the process of new idea creation.

with  $\mu, \phi \in (0, 1)$ . The diminishing returns to experience and the number of idea carriers are the key assumptions.

By the law of large numbers, fraction  $1 - m_{t,j}^y$  of young adult carriers of idea  $i$  survive to the next period and become old adult carriers of idea  $i$  :

$$N_{t+1,j}^o(i) = (1 - m_{t,j}^y) N_{t,j}^y(i).$$

Further assume that the young adults who live through the entire period and survive to old adulthood (i.e. fraction  $1 - m_{t,j}^y$  of their cohort) pass all of their ideas to their surviving children, while those who do not survive do not pass on their ideas to their children. A simple interpretation of this assumption is that a child observing and helping a father produce throughout the entire period, will master the technique, while a child whose father dies prematurely, will not receive the amount of personal contact needed to acquire his father's skills. Hence, the law of motion of the young adult carriers of idea  $i$  is given by<sup>15</sup>

$$N_{t+1,j}^y(i) = (1 - m_{t,j}^y) N_{t,j}^y(i) (1 - m_{t,j}^c) n_{t,j}. \quad (10)$$

The above two relations imply  $N_{t,j}^o(i) (1 - m_{t-1}^c) n_{t-1} = N_{t,j}^y(i)$ , and hence, the number of old adult carriers of idea  $i$  evolves according to

$$N_{t+1,j}^o(i) = (1 - m_{t,j}^y) N_{t,j}^o(i) (1 - m_{t-1}^c) n_{t-1}. \quad (11)$$

Productivity in the beginning of period  $t + 1$  is then

$$A_{t+1,j} = \left[ \sum_{i=1}^{I_{t,j}} \left[ \left( \frac{N_{t+1,j}^o(i)^\mu [N_{t+1,j}^y(i) + N_{t+1,j}^o(i)]^{1-\mu}}{N_{t+1,j}^y + N_{t+1,j}^o} \right) q_{t,j}(i)^{1-\phi} \right]^\rho \right]^{\frac{1}{\rho}} = \left( (1 - m_{t,j}^y)^\mu (1 - m_{t,j}^c)^{1-\mu} \right)^\phi \cdot \left[ \sum_{i=1}^{I_{t,j}} \left[ \left( \frac{[(1 - m_{t-1,j}^c) n_{t-1,j} N_{t,j}^o(i)]^\mu [(1 - m_{t,j}^c) n_{t,j} N_{t,j}^y(i) + (1 - m_{t-1,j}^c) n_{t-1,j} N_{t,j}^o(i)]^{1-\mu}}{(1 - m_{t,j}^c) n_{t,j} N_{t,j}^y + \frac{1 - m_{t,j}^y}{1 - m_{t-1,j}^c} (1 - m_{t-1,j}^c) n_{t-1,j} N_{t,j}^o} \right) q_{t,j}(i)^{1-\phi} \right]^\rho \right]^{\frac{1}{\rho}},$$

where we used equations (5), (6), (10) and (11).

If the mortality and fertility rates are the same in periods  $t - 1$  and  $t$ , then the above simplifies exactly to  $A_{t+1,j} = \left( (1 - m_{t,j}^y)^\mu (1 - m_{t,j}^c)^{1-\mu} \right)^\phi A_{t,j}^{end}$ , i.e.<sup>16</sup>

$$A_{t+1,j} = (1 - m_{t,j}^y)^\phi \left[ A_{t,j} + A_{t,j} i_{t,j}^\eta + d_{t,j} \right], \quad (12)$$

which implies that the fraction of potential TFP in  $t + 1$  lost due to premature death interfering with knowledge

<sup>15</sup>To see this, note that the number of surviving young adult carriers at time  $t$  is  $(1 - m_{t,j}^y) N_{t,j}^y(i)$ , and each of those carriers can transmit this idea to  $n_{t,j}$  children,  $1 - m_{t,j}^c$  of which survive to  $t + 1$ .

<sup>16</sup>If the economy is not on a balanced growth path, but the mortality distribution parameters change slowly, the knowledge destruction function (12) serves as an approximation. Using this approximation simplifies our analysis tremendously and eliminates the need to identify  $\rho$  and  $\mu$ .

transmission across time is an increasing and convex function of the adult mortality rate:

$$\frac{A_{t,j}^{end} - A_{t+1,j}}{A_{t,j}^{end}} = 1 - (1 - m^y(\omega_{t,j}))^\phi. \quad (13)$$

We refer to the influence of mortality through factor  $(1 - m_{t,j}^y)^\phi$  in the law of motion (12) as its *direct effect* on knowledge transmission across time.

There are several important messages to take. First, for reasonable micro assumptions on idea aggregation and the process of their intergenerational transfer, we derived the adverse effect of adult mortality on the evolution of TFP: the knowledge transmission factor  $(1 - m_{t,j}^y)^\phi$  is less than one and decreases in  $m_{t,j}^y$ . Second, the adverse impact of adult mortality on knowledge transmission reflects two main forces at play: (1) A premature death of the parent (master) eliminates his own contribution to future productivity and interrupts the technique transfer to his children (apprentices), eliminating their contribution as well; (2) A premature death of the parent also implies a loss of their experience to draw upon in the next period. We see that the transmission rate of knowledge across time is a product of two factors corresponding to these two effects,  $(1 - m_{t,j}^y)^{(1-\mu)\phi}$  and  $(1 - m_{t,j}^y)^{\mu\phi}$ . Third, the destruction rate  $\frac{A_{t,j}^{end} - A_{t+1,j}}{A_{t,j}^{end}}$  is an increasing and convex function of adult mortality.

The above derivations explicitly reveal the assumptions that give rise to this convexity. It arises due to the assumption of diminishing marginal returns to the level of experience associated with idea  $i$  and to the number of carriers of idea  $i$ . Intuitively, if the adult mortality rate is low, a young adult survivor has a very low marginal contribution to TFP, as ideas embodied by this person are embodied by many other survivors and their surviving children, and as many other survivor carriers are available to provide their expertise. Thus, the negative impact of one death on TFP is low. However, if mortality is high, the marginal contribution of an adult survivor is large, as both the experience and the number of idea carriers are scarce in the next period. The negative impact of one death on TFP thus increases in adult mortality.

Note that although we model the increments to TFP due to innovation and knowledge diffusion directly by borrowing from the growth literature, our assumptions regarding these terms could be made consistent with the mathematical link from embodied knowledge to TFP, used to derive the dependence of future TFP on mortality. For example, consider the innovation increment. We assumed that  $A$  increases to  $A + Ai^\eta$ , provided that young adults each spend  $i$  units of time improving the existing production process. This result could be derived through the Dixit-Stiglitz aggregator used above if the time spent innovating resulted in raising the quality of each existing idea by a factor of  $(1 + i_{t,j}^\eta)^{\frac{1}{1-\phi}}$  without changing the number of ideas or the relevant fractions of idea carriers.

### 2.3. Preferences, Constraints, Young Adults' Problem

The time  $t$  decisions are made prior to the end-of-period realization of mortality shock  $\omega_t$ . For a given realization  $\omega_{t,j}$ , a young adult may or may not survive to old adulthood. We assume that the utility of a young adult surviving to the next period is given by

$$U_{t,j}^S(\omega_{t,j}) = \frac{(c_{t,j}^y)^{1-\sigma}}{1-\sigma} + \beta \frac{(c_{t+1,j}^o(\omega_{t,j}))^{1-\sigma}}{1-\sigma},$$

where  $c_{t,j}^y$  and  $c_{t+1,j}^o(\omega_{t,j})$  denote consumption when young and old, respectively. Parameter  $\sigma > 0$  governs substitutability of the two consumption goods. Note the explicit dependence of survivor's utility on the realization of a mortality shock: although it does not influence the decision-making at time  $t$ , it will influence future consumption through resource and TFP evolution constraints. We further assume that the utility of the young adult not surviving to old adulthood is given by  $U_{t,j}^{NS} = \frac{(c_{t,j}^y)^{1-\sigma}}{1-\sigma}$ . The young adult's expected utility associated with a particular realization  $\omega_{t,j}$  is therefore

$$\begin{aligned} E(U_{t,j}|\omega_{t,j}) &= (1 - m^y(\omega_{t,j})) U_{t,j}^S(\omega_{t,j}) + m^y(\omega_{t,j}) U_{t,j}^{NS} = \\ &= \frac{(c_{t,j}^y)^{1-\sigma}}{1-\sigma} + (1 - m^y(\omega_{t,j})) \beta \frac{(c_{t+1,j}^o)^{1-\sigma}}{1-\sigma}. \end{aligned}$$

When young, adults produce, consume and innovate. When old, adults produce and consume. We abstract from physical capital accumulation in order to maintain tractability and focus on understanding the TFP acceleration, which characterized the economic transformation of England (See Bar and Leukhina, forthcoming).

The young adult cohort, taking  $A_{t,j}$ ,  $\lambda_{t,j}^y$  and frontier productivity  $\bar{A}_t := \max\{A_{t,j}\}_{j \in \{1, \dots, J\}}$  as given, chooses consumption  $c_{t,j}^y$ , time allocated to production  $l_{t,j}$ , time allocated to innovation  $i_{t,j}$  to solve

$$\max_{c_{t,j}^y, l_{t,j}, i_{t,j}} E_{\omega_{t,j}} E(U_{t,j}|\omega_{t,j}) = \frac{(c_{t,j}^y)^{1-\sigma}}{1-\sigma} + \frac{\beta}{1-\sigma} E_{\omega_{t,j}} \left[ (1 - m^y(\omega_{t,j})) (c_{t+1,j}^o(\omega_{t,j}))^{1-\sigma} \right] \text{ s.t.} \quad (\text{OP})$$

$$c_{t,j}^y = A_{t,j} l_{t,j}^\theta (\lambda_{t,j}^y)^{1-\theta}, \quad (14)$$

$$A_{t+1,j}(\omega_{t,j}) = (1 - m^y(\omega_{t,j}))^\phi \left[ A_{t,j} + A_{t,j} i_{t,j}^\eta + \tau_t (\bar{A}_t - A_{t,j}) \right], \quad (15)$$

$$l_{t,j} + i_{t,j} = 1, \quad (16)$$

$$c_{t+1,j}^o(\omega_{t,j}) = A_{t+1,j}(\omega_{t,j}) \left( \frac{\lambda_{t,j}^y}{1 - m^y(\omega_{t,j})} \right)^{1-\theta}, \quad (17)$$

where in the last constraint we substituted for  $\lambda_{t+1,j}^o(\omega_{t,j})$  from (9) and used the optimal choice to allocate the entire unit of time to production when old. Constraint (14) states that all output produced when young is used as consumption. Constraint (15) is the law of motion of TFP (12) derived in Section 2.2, in which we further assumed, following Acemoglu (2009), that  $d_{t,j} = \tau_t (\bar{A}_t - A_{t,j})$ , i.e. the increment to TFP due to diffusion of knowledge from other regions, increases in the gap between the frontier and local technology. The rate of absorption of this gap is denoted by  $\tau_t \in (0, 1)$ ; it dictates the ease of knowledge flow across regions. It is possible that this parameter positively depends on population density and factors emphasized in Mokyr (2002), such as accessibility to communication devices (railroads, post) and the extent of the availability of scientific communities. To derive the main results, we assume  $\tau_t = \tau$ ; we explore the implications of a rising  $\tau$  in Section 3.4.1.

## 2.4. Optimal Time Allocation

After substituting from the constraints into the objective function, the time  $t$  problem simplifies to

$$\begin{aligned} & \max_{l_{t,j}} \frac{1}{1-\sigma} \left[ A_{t,j} l_{t,j}^\theta \left( \lambda_{t,j}^y \right)^{1-\theta} \right]^{1-\sigma} + \\ & \frac{\beta}{1-\sigma} E_{\omega_{t,j}} \left[ \left( 1 - m^y(\omega_{t,j}) \right) \left( \left( 1 - m^y(\omega_{t,j}) \right)^\phi \left[ \tilde{A}_{t,j} + A_{t,j} (1 - l_{t,j})^\eta \right] \left( \frac{\lambda_{t,j}^y}{1 - m^y(\omega_{t,j})} \right)^{1-\theta} \right)^{(1-\sigma)} \right], \end{aligned} \quad (18)$$

where  $\tilde{A}_{t,j} = A_{t,j} + \tau (\bar{A}_t - A_{t,j})$ . Simplifying further gives

$$\max_{l_{t,j}} \frac{l_{t,j}^{\theta(1-\sigma)}}{1-\sigma} + B_t \frac{\left[ 1 + \tau (\bar{A}_t - A_{t,j}) / A_{t,j} + (1 - l_{t,j})^\eta \right]^{(1-\sigma)}}{1-\sigma}, \quad (19)$$

where  $B_t = \beta E_{\omega_{t,j}} \left[ \left( 1 - m^y(\omega_{t,j}) \right) \left( 1 - m^y(\omega_{t,j}) \right)^{\phi(1-\sigma)} \left( 1 - m^y(\omega_{t,j}) \right)^{(\theta-1)(1-\sigma)} \right]$  is the relative weight on future utility, which is the same across regions because mortality shocks are i.i.d.

Because labor is supplied inelastically in the last period of life, the decisions of the time  $t$  cohort are independent of the time  $t$  realization of the mortality shock. So the only way the time  $t$  parameters of the mortality shock distribution affect the time  $t$  decision making is through the expectation term  $E_{\omega_{t,j}} [\cdot]$  that enters  $B_t$ . The time  $t - 1$  realization of mortality, however, does affect the state variables  $A_{t,j}$  and  $\bar{A}_t$  and hence the time  $t$  decision-making. Note that the state variables  $\lambda_{t,j}^y$  and  $N_{t,j}^y$  have no bearing on the optimal time allocation.

$E_{\omega_{t,j}} [\cdot]$  appearing inside  $B_t$  is taken over the product of three factors. Formulation (18) elucidates their origin. The first,  $D(\omega_{t,j}) := (1 - m^y(\omega_{t,j}))$ , represents the discount factor on future consumption due to lifespan uncertainty. As mortality declines and people adjust their expectations accordingly, the weight on future utility increases, which encourages more innovation. The influence of the mortality decline exerted through this factor can be interpreted as the standard Ben-Porath channel, in our formulation appearing as independent of the elasticity of substitution between current and future consumption. The second factor is the knowledge transmission factor  $K(\omega_{t,j}) := (1 - m^y(\omega_{t,j}))^{\phi(1-\sigma)}$ , a function of the rate of TFP transmission across time. Declining mortality increases the term  $(1 - m^y(\omega_{t,j}))^\phi$ , which raises the amount of total knowledge transmitted to the next period, and hence increases the returns to innovation. The impact of declining mortality on time allocation through the knowledge channel represents the *indirect effect* of the knowledge transmission mechanism proposed here. It depends on the substitutability of the two consumption goods. If the goods are substitutes, an increase in returns to innovation leads to labor reallocation towards innovation, and substitution of future for present consumption. However, if the goods are complements, an increase in returns to innovation raises the demand for both present and future consumption, and consequently induces labor reallocation towards current consumption and away from future consumption, the sector experiencing productivity gains. The last factor in the expectation term is the resource dilution channel  $M(\omega_{t,j}) := (1 - m^y(\omega_{t,j}))^{(\theta-1)(1-\sigma)}$ , appearing due to the assumed land redistribution among the surviving cohort members, and branded Malthusian because of the active role that resources per capita played in driving the Malthusian population dynamics (see Malthus, 1798). As mortality declines, people adjust to expect less augmentation of their land holding over the lifecycle in case of survival, which lowers the returns to innovation and, if the consumption goods are substitutes, encourages labor reallocation away from innovating.

The objective function in (19) is strictly concave, which guarantees uniqueness of the solution. The solution is

interior, as setting the labor input to zero in either sector implies infinitely large gains from time reallocation. The first order condition characterizing the optimal  $l_{t,j}$  is given by

$$\theta l_{t,j}^{\theta(1-\sigma)-1} = B_t \frac{\eta (1 - l_{t,j})^{\eta-1}}{\left[ \tilde{A}_{t,j}/A_{t,j} + (1 - l_{t,j})^\eta \right]^\sigma}. \quad (20)$$

The first term gives the marginal benefit from increasing  $l_{t,j}$ , i.e. marginal utility from the resulting increase in consumption when young. The second term is the marginal cost of doing so, i.e. the loss, in terms of utility, of future productivity, resulting from the decline in the time spent innovating.

## 2.5. Equilibrium Dynamics

**Definition 1** For given parameter values, sequence  $\{\zeta_t\}$ , time-dependent parameters of the mortality shock distribution  $\{\mu_t^\omega, \sigma_t^\omega\}$ , mortality shock realizations  $\{\omega_{t,j}\}$  that imply a history of mortality rates according to (1), birth rates  $\{n_{t,j}\}$  and initial conditions  $\{N_{0,j}^y, N_{0,j}^o, A_{0,j}\}$ , an equilibrium consists of sequences  $\{N_{t+1,j}^y, N_{t+1,j}^o, A_{t+1,j}, \lambda_{t+1,j}^y, \lambda_{t+1,j}^o, c_{t,j}^y, c_{t,j}^o, l_{t,j}, i_{t,j}\}$  such that  $\forall t, j$ , the young adult's problem (OP) is solved and the laws of motion (2), (3), (7) and (15) hold.

The economy starts off with the initial conditions  $\{N_{0,j}^y, N_{0,j}^o, A_{0,j}\}$ . Next, we will show how given the state variables in period  $t$ , the remaining variables in period  $t$  and the state variables in period  $t + 1$  are determined. Given  $\{N_{t,j}^y, N_{t,j}^o, A_{t,j}\}$ , equations (7) and (8) give  $\lambda_{t,j}^y, \lambda_{t,j}^o$ . Equation (17) gives consumption of the current old adults  $\{c_{t,j}^o\}$ . Then (4) pins down  $N_{t,j}^c$ . The optimization problem (OP) is solved for  $c_{t,j}^y$  and  $l_{t,j}$  in each region. The mortality shocks  $\{\omega_{t,j}\}$  are realized, so the region-specific mortality rates  $m^c(\omega_{t,j})$  and  $m^y(\omega_{t,j})$  are determined for each  $j$  according to (1). Given the realizations  $\{\omega_{t,j}\}$ , equations (2), (3) and (15) determine the state variables in period  $t + 1$ :  $\{N_{t+1,j}^y, N_{t+1,j}^o$  and  $A_{t+1,j}\}$ .

## 2.6. Balanced Growth

Consider a deterministic version of our model economy in which  $\zeta, n, \mu^\omega, \sigma^\omega$  are fixed, and mortality shocks are identical across time and regions and equal their expectation value:  $m^y = E_\omega m^y(\omega)$ . Assuming further that all regions are identical in their initial conditions implies the lack of knowledge diffusion across regions, and hence regions remaining identical. It suffices to drop the subscript  $j$ . It is useful to discuss a balanced growth (BG) equilibrium of this deterministic economy, as it represents the trend for the behavior of the model with uncertainty, in which parameters of the mortality shock distribution are held fixed.<sup>17</sup>

**Proposition 1** Denote the solution to equations (21) and (22) by  $l^{BG}$  and  $\gamma_A^{BG}$ :

$$\gamma_A = (1 - m^y)^\phi [1 + (1 - l)^\eta], \quad (21)$$

$$\theta l^{\theta(1-\sigma)-1} = \frac{\beta E \eta (1 - l)^{\eta-1}}{[1 + (1 - l)^\eta]^\sigma}, \quad (22)$$

<sup>17</sup>Adding mortality shocks to this deterministic model would amount to adding shocks to the stable dynamical system characterizing it.

where  $E = E_{\omega_t} [(1 - m^y (\omega_t))^{1+(\phi+\theta-1)(1-\sigma)}]$ . Assume an arbitrary number  $J$  of regions. Suppose the initial conditions in each region are identical and satisfy  $N_0^c = n^{BG} N_0^y$ ,  $\frac{N_0^o}{N_0^y} = \frac{(1-m^y)}{(1-\zeta m^y)n}$ ,  $\lambda_0^y = \frac{0.5\Lambda}{N_0^y}$ ,  $\lambda_0^o = \frac{0.5\Lambda}{N_0^o}$ . Then the equilibrium paths exhibit balanced growth behavior from period 0 and onward, that is,  $l_t = l^{BG}$  and  $\frac{A_{t+1}}{A_t} = \gamma_A^{BG}$ ,  $\forall t$ . Moreover, the growth rates of all population subgroups, output per capita, and land holdings are then given by

$$\gamma_{pop}^{BG} = \frac{N_{t+1}^y}{N_t^y} = \frac{N_{t+1}^o}{N_t^o} = \frac{N_{t+1}^c}{N_t^c} = (1 - \zeta m^y) n, \quad (23)$$

$$\gamma_y^{BG} = \gamma_A^{BG} \left( \frac{1}{\gamma_{pop}^{BG}} \right)^{1-\theta}, \quad (24)$$

$$\gamma_{\lambda^y}^{BG} = \gamma_{\lambda^o}^{BG} = \frac{1}{\gamma_{pop}^{BG}}. \quad (25)$$

**Proof.** See the appendix. ■

As seen from (21), we obtain that the rate of TFP growth positively depends on the rate of knowledge transmission across generations,  $(1 - m^y)^\phi$ , and the time spent on innovation.<sup>18</sup> Relation (24) elucidates that TFP growth positively contributes to the growth of output per capita, while population growth inhibits it, the latter effect capturing the negative impact of the dilution of the non-reproducible factor. Population growth is given by the product of the child survival rate and fertility.

### 3. Quantitative Analysis

#### 3.1. Estimating the parameters of the mortality shock distribution

Because the main question that we ask is how changes in the frequency and severity of epidemics affected the process of output takeoff in England, we need to estimate  $\{\mu_t^\omega, \sigma_t^\omega, \zeta_t\}_{t=1600}^{2000}$ . We take a stand that each period in the model is 25 years. We gather the data on country-level mortality rates for the age groups 0-25 and 25-50 from Wrigley et al. (1997) and the human mortality database. For the list of all data sources used in this paper, refer to the appendix. The data is available at 10-year frequency; the appropriate adjustments to accommodate the 25-year period length in the model were made.

From (1), we solve for  $\omega_{t,j}$  obtaining  $\omega_{t,j} = \frac{m_{t,j}^y}{(1-m_{t,j}^y)}$ , i.e.,  $\ln \omega_{t,j} = \ln \left( \frac{m_{t,j}^y}{1-m_{t,j}^y} \right)$ . We then set  $\mu_t^\omega = \ln \frac{m_t^{25-50}}{1-m_t^{25-50}}$  for each  $t = 1600, 1625, \dots, 2000$ , that is, we set the mean of the log mortality shock to its data counterpart. We find that  $\mu_t^\omega$  fell from -0.40 to -4.98, the trajectory of the fall closely resembling the trajectory of  $m_t^{25-50}$  plotted in Figure 1.

To obtain the variance of the log mortality shock,  $\ln \omega_{t,j}$ , we linearly interpolate between its data counterpart,  $\ln \frac{m_t^{25-50}}{1-m_t^{25-50}}$ , for 1600-1650 and 1950-2000.<sup>19</sup> We obtain that the ratio of its standard deviation to the absolute value of the mean (i.e. coefficient of variation) declined by 90%.

We estimate  $\{\zeta_t\}$  by taking the ratio of the observed mortality rates for the age groups 0-25 and 25-50, reported in Figure 1, obtaining a hump-shaped series. The ratio starts at 0.82, grows initially because the mortality rate of the

<sup>18</sup>Note the growth rate of productivity is independent of the population size, i.e. there are no scale effects, criticized in Jones (1995).

<sup>19</sup>To adjust for the number of locations and convert 10 year frequency into 25 year frequency, the obtained time series is adjusted by a factor of  $10J/25$ .

age group 0-25 is roughly fixed in the data, while the adult mortality declines. It eventually decreases because in this period child mortality falls faster than the adult mortality.

### 3.2. Calibration of the Benchmark Model

One goal of this study is to quantitatively assess the importance of our hypothesis. In order to accomplish this goal, we parameterize the model to match several key features of preindustrial England assuming that the behavior of the 17th c. England can be accurately captured by the balanced growth behavior of the model economy. We thus employ equations from Proposition 1, where recall  $m^y = E_\omega m^y(\omega)$  is the mortality shock. Because all the regions are identical ex-ante and hit with an identical shock, there will be no knowledge flows across regions, and the regions will remain identical. It suffices to ignore the subscript  $j$ .

From the estimation of the mortality process parameters, we have  $m^y = E_\omega m^y(\omega) = 0.4$  and  $\zeta = 0.82$ . We also construct the general fertility rate<sup>20</sup> (GFR) series using the demographic data in Wrigley et al. (1997), Mitchell (1978) and the human mortality database. GFR is around 134 in the 17th c. England, which implies  $n = (134/1000) 25/2 = 1.675$ , where the adjustment is made to the 25-year frequency and the fact that our model is unisex. Then, according to (23), the model implies population growth to be  $\gamma_{pop} = 1.125$ , or 0.47% annual growth rate, which is roughly consistent with the population growth estimates in Wrigley et al. (1997).

Clark (2001b) reports that the land share in total income during 1600-1700 is around 0.3, so we set  $\theta = 0.7$ . From Clark (2001b), output per capita in the 17th century rose at about 0.176% per year, which implies the growth rate of 1.045 per 25 years. Then (24) implies the model must predict TFP growth along the calibrated BG path to be  $\gamma_A^{BG} = 1.082$ , or 0.3% annual growth.

Wrigley et al. (1997) documents the total population size around 1600 to be 4, 109, 981. Hence,  $N_0 = 4, 109, 981/J$  per region. Since we do not have the data on the population age composition in the 17th c., we employ the BG implication of the constant age structure, i.e.  $N_0^c/N_0^y = n$  and  $N_0^o/N_0^y = \frac{1-m^y}{(1-\zeta m^y)n}$ . We already determined the mortality and fertility rates, which allows us to determine the population structure along the calibrated BG. We must set  $N_0^y = 4, 109, 981/J / (1 + n + \frac{1-m^y}{(1-\zeta m^y)n}) = 4, 109, 981/J / (1 + 1.675 + \frac{1-0.4}{(1-0.7(0.4))1.675}) = 1281223/J$ , which implies 31% of the total population are of ages 25-50. It also follows that  $N_0^o = 682, 710/J$  per region.

Next we calibrate  $\phi$ , an important parameter that governs the rate of knowledge destruction due to mortality. Along the calibrated BG, realizations of mortality shocks equal their expected value every period, and marginal (and average) products of labor of the young and the old grow at the rate of  $(A_{t+1}\lambda_{t+1}^{1-\theta}) / (A_t\lambda_t^{1-\theta}) = \gamma_A^{BG} ((1 - \zeta m^y) n)^{\theta-1} = 1.045$ . However, a surprise mortality shock, higher than its expected value, will cause the marginal (and average) products of labor to rise, as it will raise the per capita land holdings among the survivors. Our knowledge destruction hypothesis implies that wages should rise less than what is implied by the simple arithmetic of recalculating the marginal product in the case of perfect knowledge transmission across time. How much less depends on  $\phi$ . To identify this parameter, we use the documented response of real agricultural wages to the Black Death epidemic in England. The Black Death epidemic appears to be the most useful case study for this purpose. The epidemic was not expected; people of prime age were infected and died quickly after; physical capital remained intact.<sup>21</sup>

<sup>20</sup>The general fertility rate is the number of births in a given year per 1000 females of ages 15-44.

<sup>21</sup>Wars would not be as appropriate for identifying the key parameter of the knowledge transmission mechanism, as they tend to destroy physical capital too, which we do not model here. Epidemics that involved a long and perhaps not as unpleasant a period of infection before death, would not be as appropriate either, because there would be time for the infected parent to expedite their skill transmission to the children.

Suppose a period  $t^*$  (unexpected) mortality rate,  $m_{t^*}$ , affects children and young adults indiscriminately. Clark (2001a) estimates that 25 years after the Black Death, population of England was reduced by approximately 45%. We compute the (unexpected) mortality rate  $m_{t^*}$ , that would imply a comparable destruction of the population in the calibrated model economy, by solving

$$\frac{N_{t^*+1}}{N_{t^*}} = \frac{n(1-m_{t^*})nN_{t^*}^y + (1-m_{t^*})nN_{t^*}^y + (1-m_{t^*})N_{t^*}^y}{nN_{t^*}^y + N_{t^*}^y + (1-m^y)N_{t^*}^y/(1-\zeta m^y)n} = 0.55,$$

where we used the population age structure implied by BG. The above implies  $m_{t^*} = 0.678$ . Because of the unexpected nature of the shock and because the shock equally affects all regions (so there is no knowledge diffusion), the optimal time allocation is unaffected, i.e.  $l_{t^*} = l_{t^*+1} = l^{BG}$ . It follows that, in response to  $m_{t^*}$ , the calibrated model implies the following rise in the marginal (and average) product of labor of the young adults:

$$\begin{aligned} \frac{A_{t^*+1}\lambda_{t^*+1}^{y1-\theta}}{A_{t^*}\lambda_{t^*}^{y1-\theta}} &= \left(\frac{1-m_{t^*}}{1-m^y}\right)^\phi \gamma_A \left(\frac{N_{t^*+1}^y}{N_{t^*}^y}\right)^{\theta-1} = \\ &= \left(\frac{1-m_{t^*}}{1-m^y}\right)^\phi \gamma_A ((1-m_{t^*})n)^{\theta-1}, \end{aligned} \quad (26)$$

where we used (21). Clark (2001a) presents data on real agricultural wages during the 14th c., revealing that 25 years after the Black Death, real wages were 20% higher than their preplague level. Setting (26) to 1.2 gives  $\phi = 0.132$ .

Our procedure of identifying  $\phi$  implies a very low rate of knowledge destruction: with  $m^y = 0.4$ , the expected young adult mortality of the 17th c., only 6.55% of productivity fails to be transmitted from one 25 year period to the next. Even the Black Death episode implied only a 13.6% level of productivity destruction. The smaller the value of  $\phi$ , the smaller will be the effect of gains in life expectancy. What we aim to identify is the influence of these quantitatively small gains in knowledge transmission on the course of development.

It is difficult to map and measure in the data the time spent on innovation  $i^{BG}$ . We set it to a low value of 0.05 for the 17th c. England, which means that  $l^{BG} = 0.95$ . Then the BG path equation (21) can be solved to determine its remaining unknown:  $\eta = 0.616$ . This estimate is consistent with most available estimates of labor elasticities in human capital production functions. Erosa et al (2009), for example, estimate it to be 0.6.

Setting  $\beta = \left(\frac{1}{1.02}\right)^{25} = 0.6$ , we can solve the final BG equation (22) for  $\sigma$  :

$$\theta l^{\theta(1-\sigma)-1} = \frac{\beta E \eta^{n-1}}{[1+i^\eta]^\sigma},$$

where  $E = E_{\omega_t} [(1-m^y(\omega_t))^{1+(\phi+\theta-1)(1-\sigma)}]$ . Note that  $E$  depends on  $\sigma$  and, given the distributional assumptions on  $\omega_t$ ,  $E$  does not have a closed form solution. We solve the above equation numerically and obtain  $\sigma = 0.33$  as a unique solution.<sup>22</sup>

or make suitable alternative learning arrangements.

<sup>22</sup>A word of caution is needed to warn the reader about comparing the implied intertemporal elasticity of substitution (IES)  $\frac{1}{\sigma}$  to the estimates found in the real business cycles literature. First of all, the existing estimates of the IES are mixed, although most papers tend to use  $\frac{1}{\sigma} < 1$ . Second, recall that the existing macro estimates of the IES are based on an Euler equation from the neoclassical growth model, which is obviously very different from our model, in the setup and length of period. Finally, Gruber (2008) gives a strong case for the IES to be greater than 1. He claims that the large literature that estimates IES has produced very mixed results, and most estimates suffer from the endogeneity problem. He uses an exogenous variation in tax rates across individuals to identify IES = 2. Finally, in defense of the implication of our

Finally, we normalize  $\Lambda = 10000$  and  $A_{0,j} = 1$ . Table 1 summarizes the calibrated parameters.

Table 1: Calibrated Parameter Values

Consumption Technology:	$A_0 = 1, \Lambda = 1, \theta = 0.70$
Idea Production Technology:	$\eta = 0.616, \phi = 0.132$
Preferences:	$\sigma = 0.33$
Mortality	$m^y = 0.4, \zeta = 0.82$

### 3.3. Results

We now use the calibrated model to examine the impact of the adult mortality decline on output per capita takeoff in England, and in particular, the impact due to the link proposed here.

It should be pointed out that in the calibrated model the impact on future output per capita of a temporary unexpected rise in mortality (such as the Black Death epidemic used to identify  $\phi$  earlier) and an expected permanent rise in mortality are very different. With an unexpected shock, time allocation is not affected, but the growth rate of per capita land holdings temporarily increases because the fixed resource is now shared among fewer survivors, the so called positive Malthusian check. Even though the growth rate of TFP temporarily declines (direct effect), it does not reverse the positive Malthusian check, output per capita growth rising temporarily.

A permanent expected rise in mortality, just prior to the shock realization, also has a positive effect on output per capita as, in anticipation of the low transmission of knowledge and low probability of own survival to the next period, labor moves into production of output and away from innovation. However, upon the realization of the shock, TFP growth suffers more relative to the case of the temporary rise in mortality, because in addition to the decline in the knowledge transmission  $(1 - m^y)^\phi$  (direct effect), less time is allocated to innovation (indirect effect). Although the positive Malthusian check (i.e. the rise in  $(\lambda^y)^{1-\theta}$ ) is just as large, the negative impact on TFP growth due to the permanent rise in mortality is strong enough to reverse the positive Malthusian check.

Hence, before the onset of permanent mortality changes, a surprise high mortality shock would increase output per capita growth, and would appear as "a positive check" to an observer like Thomas Malthus. The opposite would be true for a surprise low mortality rate. However, as we will show next, permanent declines in mortality, by increasing knowledge transmission across time and by encouraging innovation, will increase output per capita growth.

#### 3.3.1. The Overall Effect of Mortality Decline on Output Takeoff

In this main experiment, we simulate the model subject to the exogenously changing parameters of the mortality shock process, obtained in Section 3.1, and GFR. In other words, we vary  $\{\mu_t^\omega, \sigma_t^\omega, \zeta_t, n_t\}$  according to our estimation and track the magnitude of the resulting change in per capita output and other quantities.<sup>23</sup> While these inputs are identical across regions, regions will differ in their history of mortality shock realizations.

calibration, note that it is standard in Ben-Porath type models to maximize income over lifetime, i.e. assume infinity IES= $\infty$ . We explore the implications of setting  $\sigma > 1$  in Section 3.4.2.

<sup>23</sup>We set  $n_t = 25GFR_t/2000$  to adjust for the 25-year frequency and the fact that the model is unisex.

Although we solve the model for the time period corresponding to [1600, 2000], we report the results by zooming in on the two-hundred year period 1680-1880, the midpoint of which dates the beginning of the industrial revolution and the endpoint of which dates the beginning of the fertility transition.

In the calibrated deterministic version of the model, all regions were identical, and hence no knowledge diffused across regions. With differential mortality shocks across regions, introduced by this simulation, regions become different within one period, and hence knowledge begins to flow across regions according to (15). There are two effects that allowing for knowledge diffusion introduces in our model economy. First, it moderates the negative impact of a high mortality episode on knowledge transmission across generations, i.e. working against the hypothesis we aim to test here. More precisely, it makes the negative impact of a high location-specific mortality episode only short-lived. Second, because a knowledge inflow increases productivity of labor when old, it lowers the marginal utility of future consumption, consequently discouraging innovation.

To simulate the economy, we need to choose  $J$  and  $\tau$ . We find that the volatility of the growth rate of total population in the country is sensitive to the number of regions assumed, decreasing with the number of regions. Although the number of regions has little influence on our quantitative results, we choose  $J = 5$  to match the coefficient of variation of population growth of 0.048 around 1600. We set the absorption rate to a low level of 0.3, which allows to have a low level of variation of region-specific time investments in innovation.<sup>24</sup> In Section 3.4.1, we will investigate the quantitative implications of a rising rate of absorption.

Figure 2 reports the results from one such simulation. Panel *a* plots the log of output per capita index in each of the five regions, together with the log of output per capita index in the data. The presence of differential time paths of per capita output across regions indicates the influence of differential histories of mortality shock realizations.

Column 3 of Table 2 summarizes the results from averaging over 1000 simulations. The main experiment generates about 75% of the increase in the log of output per capita observed in the data ( $0.6839/0.9172 = 0.75$ ). Panel *c* of Figure 2 plots the population dynamics in the data and its model counterpart. Since we directly fed in our estimates of GFR and we modeled mortality dynamics in an attempt to reflect the actual mortality experience, it is not surprising that the model population dynamics matches its empirical counterpart very well. Nonetheless, it validates the model performance, in particular, our estimation of the mortality shock process.

Panel *b* of Figure 2 reports the time spent on innovation in each location. There is little difference in innovation activity across region because of the low rates of knowledge diffusion. Panel *b* reveals that the mortality decline generates a substantial rise in the time spent on innovation (from around 5% to 12%) due to the rise in the expectation term, reflected in the plot of the relative weight on future utility  $B_t = \beta E_{\omega_{t,j}} \left[ (1 - m^y(\omega_{t,j}))^{1+(\theta+\phi-1)(1-\sigma)} \right]$  reported in panel *d*. Recall the expectation term consists of three factors that we referred to as  $D(\omega_{t,j})$ ,  $K(\omega_{t,j})$  and  $M(\omega_{t,j})$ . The declining mortality trend raises the discount and knowledge factors but lowers the Malthusian channel. The first two effects are stronger than the last one, which leads to the rise in the expectation term.

Panels *e* and *f* reveal the forces behind the takeoff in per capita output produced by this experiment. All the quantities reported are averages over the regions. Panel *e* reports the growth rates of the components of output per young adult  $y_t^y$ , that is, the growth rates of  $A_t$ ,  $l_t^o$  and  $(\lambda_t^y)^{1-\theta}$ . Recall that the old adults enjoy the same productivity  $A_t$ , while the growth rate of their labor input is 1 by construction, and the growth rate of  $\lambda_t^o$  closely resembles the growth rate of  $\lambda_t^y$ , and therefore omitted from the graph. This panel reveals that gains in TFP were the main force

<sup>24</sup>Keeping the leader productivity fixed,  $\tau = 0.3$  implies that even after 100 years of knowledge diffusion, the gap between the leader and any follower still remains at 24% of the original difference.

behind the output per capita takeoff, while the dilution of land among the growing population worked in the opposite direction. Column 3 of Table 2 decomposes the change in output per young adult during the 200-year period into changes of its components: resource dilution implied that in the absence of other changes, output per young adult should shrink by 40% ( $(\lambda_{1880}/\lambda_{1680})^{1-\theta} = 0.5945$ ), the declining labor input implied a further 5% drop in  $y_t^y$ , while TFP grew by the factor of 3.44, more than offsetting the negative impact. Together these changes implied that  $y_t^y$  rose by a factor of 1.93.

Column 3 of Table 2 further decomposes the growth rate of  $A_t$  into its components:  $(1 - m_t^y)^\phi$  and  $[1 + i_t^\eta + \tau (\bar{A}_t/A_t - 1)]$ , the two terms inside the brackets reported separately. The growth rate of  $A_t$  increased from 8.6% to 26%. This increase came about due to both, the increase in the rate of knowledge transmission,  $(1 - m_t^y)^\phi$ , from 0.93 to 0.99 (a 6.6% rise), and an increase in the innovation increment  $i_t^\eta$  from 0.16 to 0.27 (a 72% rise), with the innovation increment exerting a larger influence. Panel *f* of Figure 2 plots the changes of these two components. The contribution of the knowledge inflow from other regions to TFP growth was small in 1680 at 0.0046 and was further reduced to 0.0024 in 1880 as the relative TFP gap declined.

Table 2: Results

	Data	Main Exp.	Main with Destruction of $A$ of 1600	Main with Increasing Diffusion
$\ln y_{1880} - \ln y_{1680}$	0.9172	0.6839	0.3941	0.6954
$\frac{y_{1880}^y}{y_{1680}^y}$		1.9309	1.4493	1.9530
$\frac{A_{1880}}{A_{1680}}$		3.4427	2.5577	3.4617
$\left(\frac{l_{1880}}{l_{1680}}\right)^\theta$		0.9478	0.9549	0.9478
$\left(\frac{\lambda_{1880}}{\lambda_{1680}}\right)^{1-\theta}$		0.5945	0.5957	0.5981
$\frac{A_{1705}}{A_{1680}}$		1.0862	1.0824	1.0864
$(1 - m_{1680}^y)^\phi$		0.9323	0.9345	0.9338
$1 + i_{1680}^\eta$		1.1580	1.1583	1.1580
$\tau (\bar{A}_{1680}/A_{1680} - 1)$		0.0046	0	0.0051
$\frac{A_{1905}}{A_{1880}}$		1.2647	1.1757	1.2652
$(1 - m_{1880}^y)^\phi$		0.9934	0.9345	0.9924
$1 + i_{1880}^\eta$		1.2714	1.2581	1.2713
$\tau (\bar{A}_{1880}/A_{1880} - 1)$		0.0024	0	0.0028

### 3.3.2. The Importance of the Knowledge Transmission Mechanism

In order to assess the importance of our hypothesis, that is, to single out the impact of adult mortality on takeoff through the knowledge transmission mechanism proposed here, we perform an additional experiment. The impact of gains in adult longevity on takeoff through the knowledge transmission mechanism transpires through two different channels: its direct effect through the knowledge transmission factor  $(1 - m^y(\omega_{t,j}))^\phi$  in the law of motion of TFP (15) and its indirect effect on time allocation through its influence on factor  $K(\omega_{t,j}) = (1 - m^y(\omega_{t,j}))^{\phi(1-\sigma)}$  in the expectation term. How much of the total influence of gains in adult mortality established by the main experiment transpired through these two channels? To answer this question, we simulate the economy again, except this time we

fix the channels in question fixed at their 1600 level. We then examine how much of the overall influence of mortality on takeoff established by the main experiment is eliminated.

Operationally, we still vary  $\{\mu_t^\omega, \sigma_t^\omega, \zeta_t, n_t\}$  according to our estimation, however, we do not allow changes in  $\mu_t^\omega, \sigma_t^\omega$  to affect the factor  $K(\omega_{t,j})$  in the expectation term and the factor  $(1 - m^y(\omega_{t,j}))^\phi$  in the law of motion of TFP. Instead, we set these factors to their levels implied by the mortality rate at its expected value in 1600. The remaining effects of mortality changes are those on the expectation terms through factors  $D(\omega_{t,j})$  and  $M(\omega_{t,j})$  and on population (and hence land) dynamics.

The difference in mortality histories across regions, generated in this simulation, translate into differences in population, and therefore land, dynamics, but not into differences in TFP dynamics. The reason for the identical TFP dynamics across regions is the following. Initially, TFP is identical across regions, so none of the regions experience a knowledge inflow. Time allocation is identical across locations as it can differ only due to differences in  $E$  or knowledge inflows. Since the direct effect of mortality on TFP evolution is fixed at the same level for all regions, TFP remains identical to the next period, and so on.

The results for one arbitrary simulation are reported in Figure 3, while column 4 of Table 2 reports important statistics obtained by averaging over 1000 simulations. Panel *a* of Figure 3 reveals that the influence of mortality changes on output takeoff is weakened. The rise in the log of output per capita obtained in this experiment accounts for a much lower part, only 43%, of its rise in the data, indicating that the influence exerted through the two knowledge transmission channels accounts for 32% (approximately one third) of the empirical takeoff.

Panel *d* of Figure 3 reveals that, relative to the main experiment, the rise in the relative weight on future utility is only slightly smaller; this is because  $\phi$  (and hence the influence of mortality through factor  $K(\omega_{t,j})$ ) present in the main experiment but eliminated in this experiment) is very small in our calibration. Consequently, the rise in the time spent innovating is only slightly smaller:  $i_t$  increases from 5% to 11% (as opposed to 5% to 12% rise observed in the main experiment).

Note that because the effect of mortality changes on population dynamics is identical to that in the main experiment, population and land plot dynamics are unchanged: resource dilution again implies that output should shrink by 40%. Again, it is the gains in TFP growth that drive the output per capita takeoff (panel *e*). The output takeoff obtained in this experiment is weaker essentially because TFP rises by a smaller factor (2.6 instead of its 3.44 counterpart in the main experiment), its growth rate rising only to 1.18 (instead of 1.26 observed in the main experiment). In turn, the growth rate of  $A$  exhibits a smaller rise because the direct contribution of the knowledge transmission channel is entirely shut down (direct effect) and because the innovation increment rises less (by 63% instead of 72% obtained in the main experiment) as we shut down the influence of mortality changes on  $K(\omega_{t,j})$  (indirect effect).<sup>25</sup>

In other words, we found that shutting down the influence of mortality through the two knowledge transmission channels substantially inhibits potential economic growth. Much of the growth generated in the main experiment is lost, almost entirely due to inhibited growth of TFP. In turn, the TFP acceleration was weakened mainly due to the lack of influence of mortality reductions through their direct effect.

Overall, we learn that despite our calibration procedure identifying a very low level of  $\phi$ , gains in adult mortality transpiring through the knowledge transmission mechanism account for approximately one third of the rise in the log of output per capita index. These findings suggest that, in addition to their more conventional effects, reductions in

<sup>25</sup>None of the TFP growth is due to the knowledge diffusion across regions, because TFP dynamics is identical across regions, as explained above.

adult mortality substantially contribute to the process of development by improving the process of knowledge transfer across generations.

### 3.4. Robustness Analysis

#### 3.4.1. Population Density and Knowledge Diffusion

It is reasonable that in our context, adult mortality may have yet another channel through which it can influence evolution of TFP. This potentially important influence of mortality decline is through its influence on population density and consequently the rate of knowledge absorption across regions. To capture the idea that higher population density facilitates idea exchange across regions, we set

$$\tau_t := \frac{(N_t^y + N_t^o) / \Lambda}{\psi + (N_t^y + N_t^o) / \Lambda}, \quad (27)$$

i.e., an increase in the population density would imply an increase in the speed and efficiency of knowledge transmission across regions. Parameter  $\psi$  captures other relevant factors for idea flows across regions, such as the extent of the availability of scientific communities, accessibility to communication devices, railroads, post, and others emphasized in Mokyr (2002). A decline in  $\psi$  would also increase the rate of knowledge absorption.

To maintain  $\tau_{1600} = 0.3$ , given the initial population age structure, we set  $\psi = 500$ . Next, we perform the main experiment again, except that now we allow for  $\tau_t$  to evolve according to (27). Over the time period examined, as population density rises,  $\tau_t$  increases from 0.3 to 0.76. The last column of Table 2 reveals that this large change in the rate of absorption, however, has an almost negligible effect on the dynamics generated by the model, attributing only a slightly larger role to changes in mortality. Intuitively, the rate of knowledge absorption increases the rate of catching up to the frontier TFP and hence reduces the productivity gap across regions, thus lowering potential gains from increasing the rate of absorption in the future. Moreover, mortality shocks also become less severe over time, also offsetting the potential gains from increasing the rate of knowledge absorption.<sup>26</sup>

Note that because there are no TFP differences in the second experiment that we performed, as explained in Section 3.3.2, this change in  $\tau$  would not introduce any change to that experiment. Thus, our conclusions regarding the importance of the knowledge transmission mechanism would remain the same.

#### 3.4.2. Sensitivity Analysis with respect to $\sigma$

We already discussed how the choice variables are affected by  $\sigma$ . Next, we study how our quantitative results are affected if we adjust the calibration procedure to deliver  $\sigma > 1$ . If we set the time spent innovating to 0.035 instead of 0.05 as in the benchmark model, then our calibration procedure yields  $\eta = 0.55$  (perhaps unreasonably low, but nonetheless necessary to produce a larger  $\sigma$ ) and  $\sigma = 1.3$ . Increasing  $\sigma$  actually increases the rise in the expectation term implied by declining mortality for the following reasons. In the benchmark model, the influence of gains in longevity exerted through the knowledge transmission channel  $K(\omega_{t,j}) := (1 - m^y(\omega_{t,j}))^{\phi(1-\sigma)}$  inside  $E$  was positive, while the influence exerted through the resource dilution channel  $M(\omega_{t,j}) := (1 - m^y(\omega_{t,j}))^{(\theta-1)(1-\sigma)}$  was negative. As the present and future consumption goods become complements, both effects are reversed, but the

<sup>26</sup>We do find that this experiment generates a lower variation in experiences of different regions.

overall rise in  $E$  is actually greater, because the influence of mortality through the resource dilution channel is larger in magnitude. Intuitively, an increase (decrease) in returns to innovation due to the increase in knowledge transmission (resource dilution) raises (lowers) the demand for both present and future consumption, and consequently induces labor reallocation towards (away) current consumption and away from innovation (towards).

Table 3 reports the results. Even though the main experiment generates a larger increase in the relative weight on future utility, the innovation increment  $i^\eta$  rises to a slightly lower level: to 0.24 (instead of 0.27 implied by the benchmark model). The reason for this is the lower labor elasticity in the innovation sector.

Overall, the main experiment generates a slightly smaller rise in the log of output per capita (0.6125), accounting for 67% of the increase in the log of output per capita. Shutting down the knowledge transmission channel reduces this quantity to 39.5%. This calibration then implies that gains in adult longevity working through the knowledge transmission channels accounted for 27% of the takeoff. This number is only slightly smaller than 1/3 implied by the benchmark model.

Essentially, with  $\sigma > 1$ , the decline in mortality positively influences growth only through the direct effect, while the direction of its influence through the indirect channel is reversed. However, because its influence through the direct channel is overwhelmingly stronger (just as was the case for the benchmark model), the impact of the decline in adult mortality on development through the knowledge transmission mechanism remains important.

Table 3: Results,  $\sigma = 1.3$ ,  $\eta = 0.55$

	Data	Main Exp.	Main with Destruction of $A$ of 1600
$\ln y_{1880} - \ln y_{1680}$	0.9172	0.6125	0.3627
$\frac{y_{1880}^y}{y_{1680}^y}$		1.8164	1.4141
$\frac{A_{1880}}{A_{1680}}$		3.1487	2.4619
$\left(\frac{l_{1880}}{l_{1680}}\right)^\theta$		0.9709	0.9695
$\left(\frac{\lambda_{1880}}{\lambda_{1680}}\right)^{1-\theta}$		0.5971	0.5950
$\frac{A_{1705}}{A_{1680}}$		1.0863	1.0824
$(1 - m_{1680}^y)^\phi$		0.9424	0.9345
$1 + i_{1680}^\eta$		1.1575	1.1583
$\tau (\bar{A}_{1680}/A_{1680} - 1)$		0.005	0
$\frac{A_{1905}}{A_{1880}}$		1.2339	1.1625
$(1 - m_{1880}^y)^\phi$		0.9934	0.9345
$1 + i_{1880}^\eta$		1.2399	1.2440
$\tau (\bar{A}_{1880}/A_{1880} - 1)$		0.0028	0

## 4. Conclusions

We proposed a new causal link from gains in longevity to TFP growth, based on the notion of embodied knowledge and personal contact being crucial in its transfer. According to this link, a decline in mortality can contribute to

the increased growth by (1) directly improving knowledge transmission across time and (2) indirectly encouraging innovation, which can transpire even in the face of a declining labor supply.

For reasonable micro assumptions on idea aggregation and their intergenerational transfer, we derived a simple and intuitive form of the adverse impact of adult mortality on knowledge transmission across generations. The destruction rate of TFP we obtained is an increasing and convex function of adult mortality. We then incorporated this form into an overlapping generations model of endogenous growth, which allowed us to identify its key parameter by taking advantage of the available historical and the Black Death epidemic data for England.

In order to assess the quantitative relevance of the proposed link between adult mortality and growth, we applied the model to investigate the long-run growth experience of England. When we vary the time-dependent parameters of the mortality shock distribution and fertility rates according to our estimates, so that the model accurately captures the population and land plot dynamics, the parameterized model generates both an early stagnation and the later rise in output per capita, explaining three fourths of the increase in output per capita between 1680 and 1880. Moreover, the influence of the decline in adult mortality, exerted through the two knowledge transmission channels (by directly improving knowledge transmission across time and by indirectly encouraging investment in productivity), accounts for one third of the empirical rise in the log of output per capita.

These findings suggest that, in addition to their more conventional effects, reductions in adult mortality substantially contribute to the process of development by extending the amount of personal contact in learning relationships and thereby improving the process of knowledge transmission across generations. The causal link between mortality and growth, proposed here, thus deserves further theoretical and empirical investigation.

## 5. Appendix

### *Data Sources*<sup>27</sup>

*Index of Real GDP per capita* ( $y$ ): [1565-1865] - Clark (2001a), Table 7, p. 30, rescaled to equal 100 in 1565 (England and Wales); [1820-1990] - Maddison (1995), p. 194, rescaled to match Clark's index in 1850 (UK).

*Crude Birth and Crude Death Rates*: [1541 - 1871] - Wrigley et al. (1997) (England); [1871 - 1986] - Mitchell, 1978 (England and Wales).

*General Fertility Rate*: Computed using CBR and the fraction of females in the total population, taken from Wrigley et al. (1997) for [1541 - 1841] (England) and Human Mortality Database for [1841 - 1999] (England and Wales).

*Population Growth Rate*: [1541 - 1836] - Wrigley et al. (1997) (England); [1841 - 1999] - Human Mortality Database (England and Wales).

*Age-specific survival probabilities*: [1580-1837] - Wrigley et al. (1997) (England); [1841 - 1999] - Human Mortality Database (England and Wales).

### *Proof to Proposition 1*

We want to show that the BG equilibrium described in Proposition 1, i.e.  $\{A_t = (\gamma_A^{BG})^t A_0, N_t^y = (n(1 - \zeta m^y))^t N_0^y, N_t^c = (n(1 - \zeta m^y))^t N_0^c, N_t^o = (n(1 - \zeta m^y))^t N_0^o, l_t = l^{BG}, \lambda_t^y = \lambda_0^y / (n(1 - \zeta m^y))^t, \lambda_t^o = \lambda_0^o / (n(1 - \zeta m^y))^t\}$ , indeed comprises the equilibrium of the model economy with the parametric restrictions and initial conditions specified in the supposition. We proceed by showing that the candidate solution satisfies all conditions sufficient for the equilibrium. First, the candidate solution also satisfies  $N_0^c = nN_0^y, \frac{N_0^o}{N_0^y} = \frac{(1-m^y)}{(1-\zeta m^y)n}$  and (23) and hence it satisfies the equilibrium relationships (4) – (6). The candidate solution satisfies  $\lambda_0^y = \frac{0.5\Lambda}{N_0^y}, \lambda_0^o = \frac{0.5\Lambda}{N_0^o}$  and (25), and hence it satisfies the equilibrium law of motion for land holdings (7) and (8). Because the candidate solution satisfies (21) and because all regions are ex-ante and ex-post identical along the candidate path, i.e.  $\bar{A}_t = A_{t,j}$  for all  $t$  and  $j$ , the candidate solution also satisfies the equilibrium relationship (15). Finally, since the candidate solution satisfies (22), it also satisfies the first order condition (20). Hence, we showed that the candidate BG solution satisfies all of the sufficient conditions for the equilibrium. Since there is a unique equilibrium solution, the candidate BG solution is the equilibrium solution. *Q.E.D.*

<sup>27</sup>Due to data limitations for England, we were forced to draw on the data sources available for England and Wales and UK. Although this inconsistency introduces some degree of error, we believe that it is small for the following reasons. (1) We do not consider level variables, such as GDP or population size, but instead growth rates, indices, and fractions of level variables. (2) For the period under consideration, the population of Wales is less than 6% of that of England. (3) Scotland's population size relative to that of England and Wales falls from 17% in 1820 (the earliest date for which we are forced to use UK data sources) to less than 10% today. (4) Appropriate rescaling was made in all cases.

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FIGURE 1: MORTALITY RATES AND OUTPUT PER CAPITA IN ENGLAND

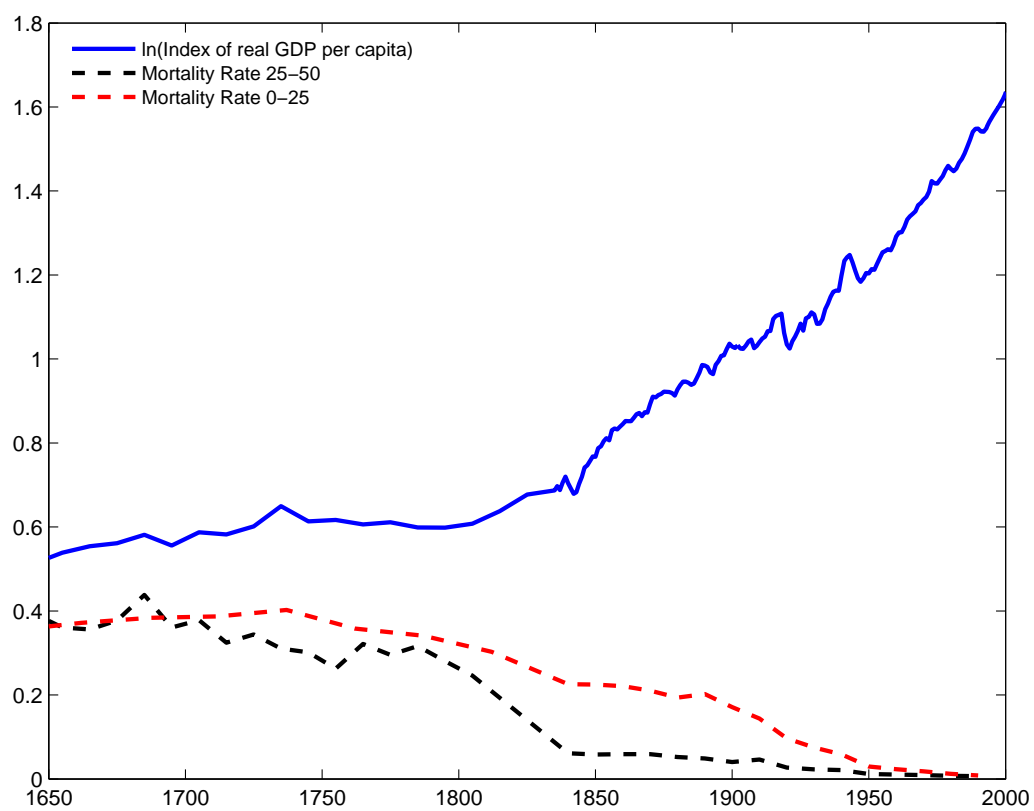


FIGURE 2: MAIN EXPERIMENT

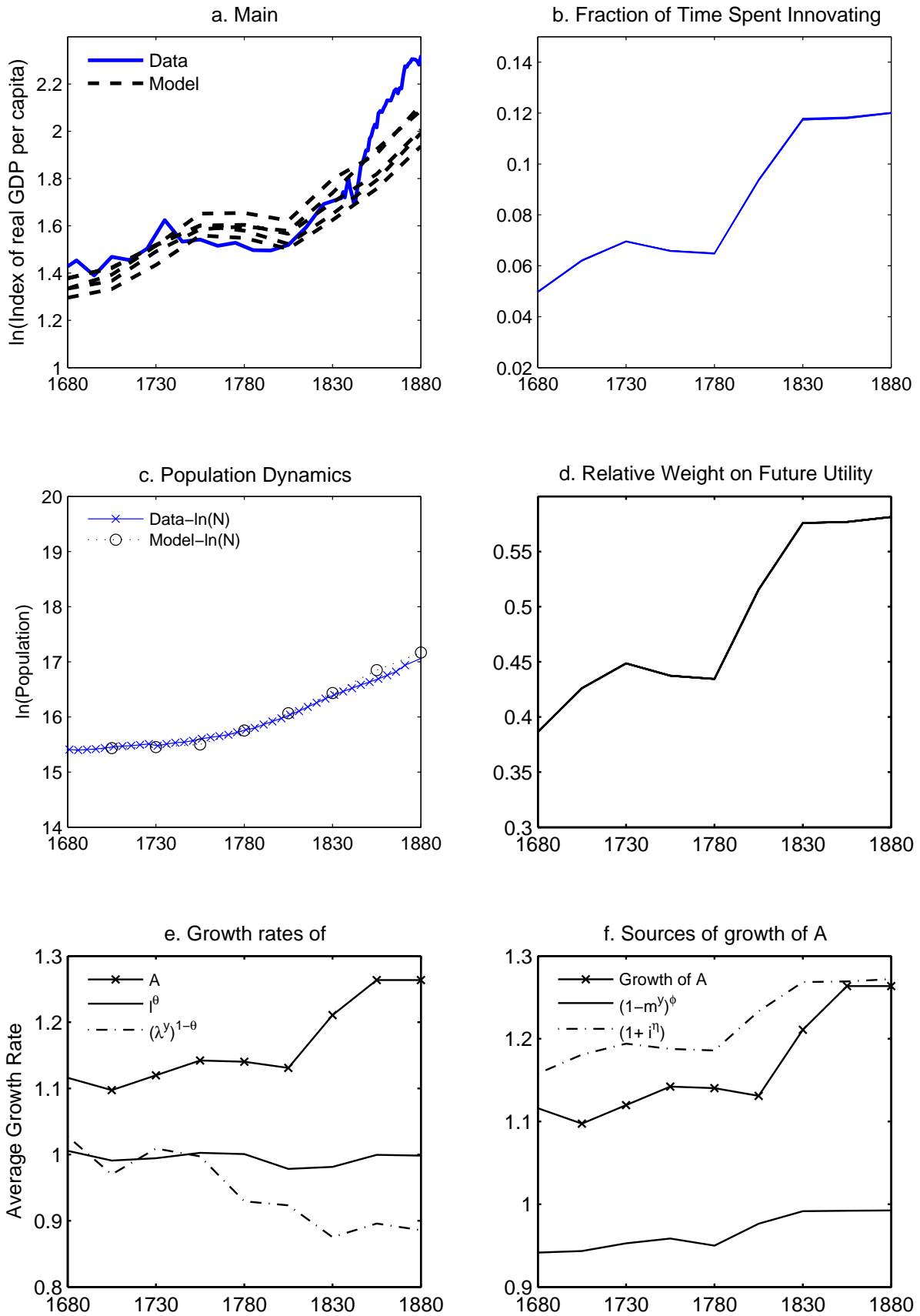


FIGURE 3: MAIN EXPERIMENT WITH KNOWLEDGE DESTRUCTION OF 1600

