A Theory of Medicine Effectiveness, Differential Mortality, Income Inequality and Growth for Pre-Industrial England

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May 15, 2006

Abstract

We study how mortality reductions and income growth interact, looking at their relationship prior to the Industrial Revolution, when income per capita was stagnant. We first present a model of individual medical spending giving a rationale for individual health expenditures even when medicine was not effective in postponing death. We then explain the rise of effective medicine by a learning process function of expenditures in health. The rise in effective medicine can then be linked to the take-off of the eighteenth century through life expectancy increases, and fostered capital accumulation. The rise of effective medicine has also an impact on the relation between growth and inequality and on the intergenerational persistence of differences in income. These channels are operative through differential mortality induced by medicine effectiveness that turns out to determines a differential in the propensity to save among income groups.
Keywords: Differential mortality, Life expectancy, Propensity to save, Health expenditures.
JEL Classification Numbers: J10, I12, D91, E13, N33.

CORE DISCUSSION PAPER 2006/45

[1]We acknowledge financial support from the Belgian French speaking community (Grant ARC 03/08-235 "New macroeconomic approaches to the development problem") and the Belgian Federal Government (Grant PAI P5/21, "Equilibrium theory and optimization for public policy and industry regulation").
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We are grateful to Raouf Boucekkine for useful comments on an earlier draft.

Introduction

In the last centuries, after years of Malthusian stagnation, income per-capita has shown a remarkably increasing trend. Better material condition and important advances in medical science have changed the quality and the length of life for millions of people. A question that naturally emerges, concerns the relation between medical knowledge and income. The causality between these two variables seems to be bidirectional: higher income allows higher health expenditures, which contributes to build medical knowledge; advances in medical knowledge increase longevity which, modifying the incentives to invest in physical and human capital, has an impact on the growth rate and on the distribution of income. The link from growth to longevity is reviewed for example in Fogel (1994) and the subsequent literature. The effect of longevity on growth is quantitatively assessed by Boucekkine, de la Croix, and Licandro (2003) and Nicolini (2004) who show that small improvements in adult life expectancy in the eighteenth century can cause big changes in economic decisions, leading to an acceleration in income growth.

In order to disentangle the causation links between medicine and income, it is enlightening to look at the period prior to the Industrial Revolution, when income was stagnant but medical knowledge improving.

From the available data for England, discussed in details in section 2, we can decompose the evolution of medical knowledge and of life expectancy into three periods. Before the seventeenth century the effectiveness of medicine was very low. Truly speaking, medicine was probably not effective at all. Given its low effectiveness of medical services, doctors hardly managed to increase the life expectancy of their patients. As a consequence, differential mortality between the rich adult who can afford the services of a doctor and the common was very low, or even non existent: income was of little importance in determining life expectancy. Next comes a time where medicine becomes more effective. The rich is the first to benefit from these improvements, and their life expectancy rose, as it is witnessed in the study of British peerage families carried over by Hollingsworth (1977) on the basis of genealogical data. Differential mortality starts increasing, because the improvements in longevity still do not benefit to the whole population. In the third period there is a global improvement in health. Medicine become more and more effective, and the advantage of upper classes in terms of longevity is partially made up. Differential mortality decreases.

In this paper we first build a model of medical spending that is suitable to be applied in a period, like pre-industrial Europe, where medicine effectiveness was very low. In this framework we give a rationale for individual health expenditures even when medicine was not effective in postponing death. Then we combine this result with a simple learning process based on health expenditures to model the rise of effective medicine: the continuous demand for health services allow to accumulate knowledge, paving the way for advances in medical science. We show that these ingredients are sufficient to build a consistent explanation of the path of differential mortality in England that we have presented above.

Then we use our model economy to look at the implications of the evolution of medical science on income. In particular we discuss the effect on the growth rate and we also study how differential mortality, inducing a differential in the propensity to save and bequeath across income groups, plays a role in shaping the path of inequality and of the relationship between inequality and growth.

The structure of the paper is the following. In Section 1 we present a brief review of the literature linking health and growth. In Section 2 we show mortality data per social class in order to assess the role of income in shaping health. We also provide a brief discussion and some references concerning the evolution of medical science. Section 3 presents the model. Section 4 focuses on the solution to the household optimization problem. Section 5 is devoted to the study of dynamic properties of the model. Finally Section 6 summarizes the main conclusions.

1 Literature Review

The study of the relations between longevity and economic variables is receiving a growing attention. In particular it is finding specific applications in development economics and in the literature that, following the contribution of Galor and Weil (1999) and Galor and Weil (2000), aims to explain the take off of European economies during the eighteenth and the nineteenth centuries after years of Malthusian stagnation. A general and informal discussion on the possible links between health (and in particular life expectancy) and economic variables is for example presented in Sala-I-Martin (2002).

More specifically de la Croix and Licandro (1999) explore the possibility that exogenous improvements in longevity have positive effects on education decisions. Anal-

ogous ideas have been used by Boucekkine, de la Croix, and Licandro (2003) and Nicolini (2004) to shed light on the historically observed relation between rising life expectancy and acceleration of growth in the period before the industrial revolution. The effects of an exogenous reduction of mortality on growth are also proved to be shaped by social security systems, in a framework where fertility decisions are endogenous and parents face a trade-off between the quality and the quantity of their children (Zhang, Zhang, and Lee (2001)).

Not only life expectancy has an impact on economic variables and in particular on growth; the opposite is also true. For these reasons other authors endogenize life expectancy to study the bidirectional link between longevity and growth. Morand (2002) argues that investment in health plays a crucial role in the transition from a growth regime fueled by physical capital to a sustained growth regime fueled by human capital. In Chakraborty (2004) low income and high mortality reinforce each other, making possible the existence of poverty traps. A similar result is found in Cervellati and Sunde (2005) and it is used to model an endogenous transition from a long period of stagnant growth to a period of sustained growth through a process that can be interpreted as industrial revolution. Kalemli-Ozcan (2002), Blackburn and Cipriani (2002) and Lagerloef (2003) combine endogenous life expectancy and endogenous fertility decisions with the purpose to give a complete account of the interrelation between demographic and economic variables. Finally Castello-Climent and Domenech (2004) and Chakraborty and Das (2005) show that the endogenous determination of life expectancy could significantly affect the intergenerational transmission of inequality, contributing to explain the persistent disparities between individual wealth or income levels.

Usually growth models that endogenize life expectancy relate either directly or indirectly life expectancy to medical knowledge. A direct link can be established by the explicit introduction of investment in health. An indirect link is often modeled linking life expectancy to human capital. This is also true for those models (for example Morand (2002), Cervellati and Sunde (2005), Lagerloef (2003)) that are more directly involved in the explanation of the take off European economies during the eighteenth and nineteenth centuries: life expectancy increase as a consequence of an higher investment in health (in Morand (2002)) or an higher level of human capital, that could be interpreted as higher medical knowledge (in Cervellati and Sunde (2005) and Lagerloef (2003)). Galor and Moav (2005) develop a complementary theory (that does not rely on the role of medical technology) of the time path of life expectancy over time in the $10,000$ years between the Agricultural Revolution in the

Neolithic period and the Industrial Revolution at the end of the eighteenth century. The theory relies on the idea that mortality risk associated with environmental factors rose in the transition from hunter-gatherer tribes to sedentary agricultural community and ultimately to urban societies, triggering a process of natural selection that finally produce a population with an higher genetic potential for longer life expectancy.

However to the best of our knowledge, though many aspects of the relation between longevity, growth and inequality have been explored in the literature, no paper focuses on the specific role of the effectiveness of medical science, explaining its evolution and the related implications for growth and inequality.

2 Income and Mortality in Pre-Modern Europe

It is difficult to find data on mortality rate or life expectancy by income groups for the pre-industrial period. We gather here some evidence for England as a whole, combining sources from Hollingsworth (1977) and Wrigley et al. (1997), and for two cities from continental Europe, based on surveys by Perrenoud (1975) and Bardet (1983).

Starting with England, we compare in Figure 1 life expectancy at birth of the average person (from parish reconstitution, see Wrigley et al. (1997)) with the one of the British aristocracy (from genealogical data, see Hollingsworth (1977)). Before 1700, there is not much differences in adult mortality across social classes. Surprisingly the elites have lower life expectancy; this unexpected result is attributed by Johansson (1999) to an urban penalty paid by the aristocracy for the pleasures and opportunities of city life. The quasi-egalitarian mortality regime began to change by 1700. Life expectancy rose for all groups but faster and further for the elite.

Two other data sets can give further hints on mortality per social class before the Industrial Revolution. They cover the population of two cities in Continental Europe, Geneva (Perrenoud 1975) and Rouen (Bardet 1983). Age specific survival probabilities are presented in Table 1 for three social classes. In both data sets, infant mortality rates are much lower in the elite groups. This reflects the better living conditions of this group. We also find that in Geneva (XVIII century) and in Rouen (XVIII century) there is not much differences in adult mortality across social classes. In Rouen, there is no difference in the survival probabilities from age 15 to age 30 and from age 30 to

Figure 1: Life expectancy at birth in England

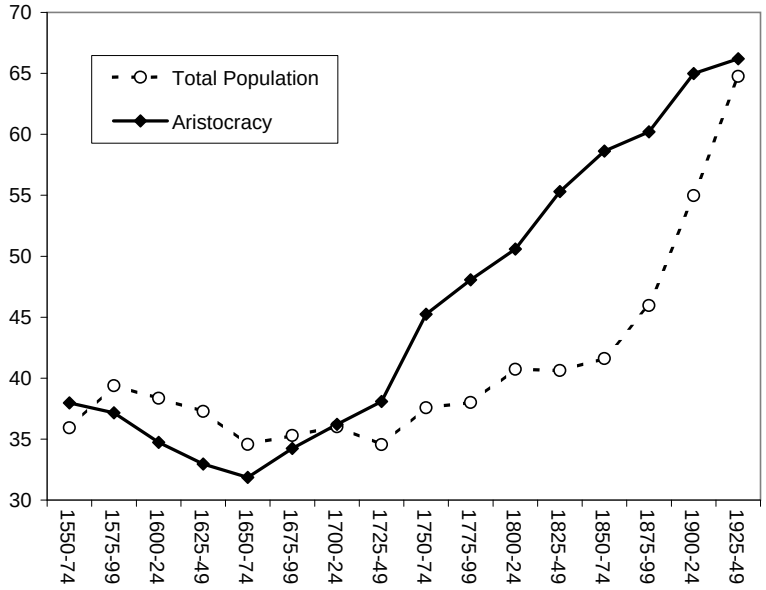


Table 1: Survival rates in Geneva and Rouen

	social class	Survival probabilities 0→15	15→30	30→45
Geneva XVII	workers	0.34	0.80	0.70
	merchants	0.45	0.84	0.74
	notables	0.61	0.89	0.81
Rouen XVIII	workers	0.33	0.85	0.87
	merchants	0.49	0.87	0.86
	notables	0.47	0.86	0.84

age 45 of notables and simple workers. This is in line with the English data presented above. In Geneva, the upper social class has a slight advantage.

The determinants of the evolution of life expectancy during the the past centuries have been lively discussed in medical history and mortality history. One of the issue concerns the role of medical science.

Siraisi (1990) provides a survey of medical learning and practice in Europe between 1100 and 1500. There was little to distinguish from one healer to one another in terms of effectiveness, with the possible exception of surgeons. The techniques employed and the general agreement on what they could or could not achieved remained remarkably stable over the period. Lindemann (1999) covers the period 1500-1800 and shows how ancient ideas persisted well into the eighteenth century.

The weakness of ancient medicine was well known by the consumers of the seventeenth century. According to Porter (1995), critics denied that medicine as taught in the universities did much good. The pompous physician, spouting Greek aphorisms, was an easy satirical target; and the experiences of decimating waves of epidemics did nothing to enhance doctors reputation. Popular proverbs endorsed this distrust: one doctor makes work for another.

As a consequence some authors claim that the rise of life expectancy in early modern Europe relied more on changes in immunology and/or improvement in the climate than on human factors (like medical advances). Johansson (1999) argues against the traditional therapeutic nihilism - that tend to denies any effectiveness of medicine before the end of the nineteenth century - and suggested an increase of medicine effectiveness as a possible explanation for the change, documented in Figure 1, from an egalitarian mortality regime to a regime characterized by mortality differential between rich and poor people. Indeed in the period 1500-1800, medicine showed an increasing experimental attitude. Though no improvement was done on the ground of the disease theory (which was still mainly based on traditional ideas), significant advances based on practice and empirical observations were done.

Many valuable observations on diseases were made before 1800 but was not accompanied by systematic research programs, as described in Porter (1996). For example, Dobson demonstrated in 1776 that the sweetness of urine in diabetes was due to sugar; Lettsom published in 1786 a detailed account on alcoholism; Beddoes and others conducted investigations into tuberculosis. But no decisive breakthroughs came from disease theory.

The effectiveness of the treatment of some important diseases was improved thanks to the discovery of new drugs coming from the new world: ipecacuanha was used for severe dysentery, guaiacum for Syphilis, and the bark of the cinchona tree for Ague (malaria). Advances in the treatment of Syphilis were also done due to improvements in the development of condoms. Citrus fruits started to be used in the prevention and in the treatment of scurvy. All these drugs allowed to make advances. The understanding of how drugs works only came progressively in the nineteenth century, with the development of chemistry (Weatherall 1996).

Finally Table 2, which shows the number of books containing lifestyle advice written in the period $1600 - 1800$ by sub-periods, provides some indirect evidence of the fact that lifestyle advices (regarding for example personal and domestic cleanliness) became popular [1]. These new medical advances, mainly due to their high cost, were available for a long period only to rich people.

In the eighteenth century bubonic plague also disappeared from England. However the reasons of this disappearance of the bubonic plague are still lively debated and some authors explain it in exogenous terms. In any case, independently of this debate, it could be noticed that a "warning system" was developed by the seventeenth century in London. Weekly bills of mortality, containing the cause of death, were published: when the number of plague deaths reached a worrying value, foreshadowing the outbreak of the epidemic, people having enough money to move leaved the city. This system, though not effective in the treatment of the plague itself, can be interpreted as a public health measure, that had an impact on the health of rich people.

As suggested by Johansson (1999) the cumulative effects of the above described improvements could have determined a net efficacy of medicine in the eighteenth century. "As early as 1829 Dr.F.B. Hawkins wrote a book entitled *Elements of medical statistics*, in which lie described what could be called an early modern epidemiological transition. Several centuries before his own time leprosy, plague, sweating sickness, ague, typhus, smallpox, syphilis and scurvy had been leading causes of death. Now all of these diseases had disappeared, could be cured, or treated effectively. At the present scarlet fever, consumption, gout, dropsy, palsy, apoplexy (including heart attacks and strokes), mania, and diseases of the brain were the most prevalent causes of death. The last six of the eight diseases listed were not contagious; they were

[1] We are aware that, to draw conclusions about the increasing diffusion of lifestyle advices, it would be better to look at the ratios between medical books and the total number of books. However, to the best of our knowledge, data about the total number of book written in the period are not available

chronic diseases most likely to strike older adults"(Johansson (1999), pag. 48)

Table 2: Titles in health in England, 1600-1800

Period	Number of books
1600-24	9
1625-49	16
1650-74	17
1675-99	25
1700-24	28
1725-49	34
1750-74	53
1775-1800	81

We conclude this section with an important remark. In the remaining part of this paper we focus on the role of medicine (as in the analysis of the evidence presented above, we use this term in a broad meaning, including also lifestyle advices). This does not mean that the increase in the level of life expectancy can be explained relying only on advances in medical science. Many factors may have contributed to the reduction in mortality since the eighteenth century (nutrition, medicine, immunology) and attributing a specific weight to each of them is very difficult (Fogel 2004). The reason for which we restrict our analysis to medicine effectiveness is that the focus of the paper is more on the differences in the increase of life expectancy between the rich and the poor than on the evolution of the level of life expectancy in absolute terms. Among the factors that may have affected life expectancy in early modern Europe, access to health care seems to be particularly relevant for explaining this differential mortality between social classes.

3 A Model of Medical Spending

The model is set up in discrete time, with time going from 0 to infinity. In each period one physical good is produced using labor, capital and land. All households are endowed with one unit of labor. The total supply of land is fixed and it is normalized to 1. We assume as initial condition that the ownership of land is equally distributed among some households (called "landlords" or "aristocrats" that we will denote with the index "L") and that other households (called "commons" that we will denote with the index "W") do not have any property right on land. Moreover we also assume

for simplicity that there is no market for land and, as a consequence, this ownership structure perpetuates over time. In this sense we have in the economy two social classes or groups. The size N^i of the newborn generation in each population group is assumed constant over time (any other stationary dynamic process for the population would qualitatively give the same result, without adding any crucial element).

Households can live for two periods, adulthood and old-age. They are alive with certainty during the first period and at the end of adulthood, they die with a probability $\beta(h_t^i)$ which depends on their health status h_t^i.

Individual budget constraints

In the first period of life, agents receive a bequest b_t^i from their parents and obtain income from land and/or labor:

$$y_t^L = b_t^L + w_t + \rho_t x_t \quad (1)$$

$$y_t^W = b_t^W + w_t \quad (2)$$

where y_t^i denotes income, ρ_t is the return from holding one unit of land, $x_t = 1/N^L$ is the amount of land per person of group L, and w_t is the wage per person that is the same for both groups.

Income finances consumption c_t^i, investment in health d_t^i and savings s_t^i. If an agent survives in the second period he do not consume and he uses his savings to finance the bequest he leaves to his offspring. Thus budget constraints of the first and second period are respectively:

$$y_t^i = c_t^i + d_t^i + s_t^i \quad (3)$$

$$R_{t+1} s_t^i = b_{t+1}^i \quad (4)$$

The inter-temporal budget constraint is

$$y_t^i = c_t^i + d_t^i + \frac{b_{t+1}^i}{R_{t+1}} \quad (5)$$

Here we do not assume the existence of a perfect annuities market. If an agent dies before entering in the old-age his resources (i.e. $R_{t+1} s_t^i$) pass on to his offspring. As a consequence we have in any case that the bequest received by an agent at the beginning of its adulthood does not depend on the survival of his father.

Medicine as an experience good

We distinguish the stock of health h_t^i from the investment in health d_t^i. The reason is that in the model the health status is determined by health expenditure, but in a non deterministic way: indeed we explicitly take into account that medical treatment may turn bad, in particular if medicine is inefficient. More precisely, we assume that, when allocating an amount d_t^i of resources to health expenditures, the household can reach a high level of health $h^+(d_t^i)$ with probability p_t, and a worsened level of health $h^-(d_t^i)$ with probability $1 - p_t$; in particular:

$$h(d_t^i) = \begin{cases} h^+(d_t^i) = 1 + v(d_t^i) & \text{with probability } p_t \\ \\ h^-(d_t^i) = 1 - v(d_t^i) & \text{with probability } 1 - p_t \end{cases} \tag{6}$$

with:

$$v(0) = 0; v'(d_t^i) > 0$$

The idea underling this formulation is that a) medical expenditures can be good or bad for health and b) each agent is not able to know the quality of medical cares before having experienced them, but he only knows the probability p_t that can be interpreted as a measure of the effectiveness of medicine.

The possibility that medicine could harm patients could seem at a first glance strange given today's standards, but it has been always recognized by physicians. One of the most basic precepts all medical students - also nowadays- are taught is

primum non nocere

(First of all, do not harm!): this aphorism is meant to remind that there is a possibility for any medical intervention to damage patients.[2]. When building economic models of the demand of *modern* health services, it is considered reasonable for analytical simplicity to omit this feature of medical treatments, since one may think that the probability of medicine to damage the health of its patients is very low.[3] However when analyzing the demand of medical services in *pre-modern* Europe, we think that it is crucial to explicitly take into account the potential damage that this medical

[2]This aphorism is usually considered a Latin paraphrase by Galen of an Hippocratic aphorism, but it seems more plausible to attribute it to the English physician Thomas Sydenham (1624-1689) (for an history of this aphorism and a discussion of its applicability as an ethical guide for modern medicine see Smith (2005).)

[3]However saying that this probability is perhaps negligible from an economic point of view, does not mean that it can be disregarded from the concrete point of view of medical science and its patients. In medical literature the problem of adverse reactions to drugs is for example widely discussed.

services might cause: a theory that abstract from this feature of health expenditure should be considered of difficult applicability in a period where the development of medicine and its effectiveness were very far from the present levels.

The other assumption implicit in (6) is that medicine is an experience good, i.e. people do not know the quality of medical services before having bought them Once again this is also true nowadays and it was even more true in the past[4].

We stress that by using these assumptions we put ourselves in the worst position for justifying our theory of the rise of effective medicine, which relies on the learning by spending mechanism described later on. Indeed if we use a standard formulation in which medicine is always a good it is easy to explain why the demand of medical services was positive in early modern Europe. This explanation is much less obvious under our formulation that allows the probability of medicine to damage patients to be equal to $1/2$.

Preferences

The preferences of an individual born at time t and belonging to class i are defined over bundles $(c_t^i, b_{t+1}^i, h_t^i)$. Preferences are represented by a utility function $U(c_t^i, b_{t+1}^i, h_t^i)$ which is increasing in the three arguments.

Health affects the utility through two channels: it influences the probability to survive in the old-age and it has a direct effect on the utility function when old. Assuming time separability, $U(c_t^i, b, h_t^i)$ can be written as:

$$u(c_t^i) + \beta(h_t^i)\tilde{u}(b_{t+1}^i, h_t^i) \tag{7}$$

where u and $\tilde{u}$ are well-behaved increasing instantaneous utility functions.

The survival probability $\beta(h_t^i)$ takes the following form:

$$\beta(h^+(d_t^i)) = \beta + f(d_t^i) \tag{8}$$

$$\beta(h^-(d_t^i)) = \beta - f(d_t^i) \tag{9}$$

where $f'(.) > 0$, $f(0) = 0$ and $f(\infty) \leq \beta \leq 1 - f(\infty)$. From (6) we have that the survival probability is determined by (8) and (9) respectively with probability p_t and $1 - p_t$.

[4]For a description of the uncertainty a consumer faced in the selection of a good medical service and of the way medical technology diffused among households see Mokyr (1993)

If we finally assume that $\tilde{u}(b^i_{t+1}, h^i_t) = u(b^i_{t+1})h^i_t$, utility can be written in expected terms as:

$$\begin{aligned}
\mathrm{E}\, U(c^i_t, b^i_{t+1}, h^i_t) =& u(c^i_t) + p_t(\beta + f(d^i_t))u(b^i_{t+1})(1 + v(d^i_t)) \qquad (10)\\
&+ (1 - p_t)(\beta - f(d^i_t))u(b^i_{t+1})(1 - v(d^i_t))\\
=& u(c^i_t) + u(b^i_{t+1})[\beta + f(d^i_t)v(d^i_t) + (2p_t - 1)(f(d^i_t) + \beta v(d^i_t))]\\
=& u(c^i_t) + \phi(d^i_t, p_t)u(b^i_{t+1}) \qquad (11)
\end{aligned}$$

$$\text{with } \phi(d^i_t, p_t) \equiv \beta + f(d^i_t)v(d^i_t) + (2p_t - 1)(f(d^i_t) + \beta v(d^i_t))$$

Notice that this utility function is formally similar to the one of Chakraborty and Das (2005); the difference is that in our case the function $\phi(d, p)$ is not a survival probability: it is a more complex expression that depends on the survival probability (and thus on the probability p_t) and on the utility of health and is derived from our previous assumptions.

Medical technology

The probability p_t of getting benefits from medicine depends on the current state of medical knowledge:

$$p_t = G(M_t) \qquad (12)$$

where M_t is the stock of medical knowledge available at period t. We assume that there is a threshold $\bar{M}$ below which medicine is inefficient:

$$G(M_t) = \begin{cases} \frac{1}{2} \text{ if } M_t < \bar{M} \\ \frac{1}{2} + g(M_t) \text{ if } M_t \geq \bar{M} \end{cases} \qquad (13)$$

where $g(M_t) > 0$ and $g'(M_t) > 0$.

The stock of medical knowledge M_t accumulates with expenditures in health:

$$M_t = (1 - \delta)M_{t-1} + D_t \qquad (14)$$

where aggregate expenditure in health are given by

$$D_t = N^W d^W_t + N^L d^L_t$$

and δ is the depreciation rate of the stock of medical knowledge, possibly zero.

The view behind the above formulation is that medical knowledge evolves through

a learning process based on experiments with treatments. Learning is thus related to the amount of medical expenditures. Moreover while standard model of learning by doing (explaining improvements in labor productivity) are continuous, we assume in (13) a threshold effect. This assumption depends on the specific nature of science and reflects the fact that "physicians slowly learned how to learn from empirical observations". The idea is that it took many observations (and thus time) before the importance of observations themselves was fully understood and more effective methods for drawing conclusions from experience were developed. As we have already said in section 2, it was only after the seventeenth century that medicine started to rely on systematic and organized methods of observation [5].

We remark that in our model, there is no exogenous technical progress, neither in the medical sector nor in the final good sector. However this does not mean that we do not believe that there cannot be improvements in medical knowledge without experimentation (after all, fundamental research may play a role) but it implies that our results do not rely on the presence of such exogenous trends.

Final good technology

The final good is produced from a technology combining land, labor and capital. We assume that capital fully depreciates after one period. The production function is of the AK type, and includes a positive externality of the aggregate stock of physical capital $\bar{K}_t$ in such a way that social marginal returns to capital are constant:

$$Y_t = AL_t^{1-\alpha-\theta} K_t^{\alpha} X_t^{\theta} \bar{K}_t^{1-\alpha}$$

with A denoting total factor productivity, L_t labor input, K_t capital input, and X_t land. The profits of the representative firm are given by

$$Y_t - w_t L_t - R_t K_t - \rho_t X_t$$

The equilibrium

Given an initial stock of medical knowledge M_0, an initial stock of capital K_0, and an initial asset distribution (s_{-1}^W, s_{-1}^L) such that $K_0 = N^W s_{-1}^W + N^L s_{-1}^L$, an equilibrium is

[5] In particular two english personages played an important role: the philosopher Francis Bacon (1561-1626) (who is very well known for his theory of induction and wrote "The historie of life and death with observations naturall and experimentall for the prolonging of life") and the physician Thomas Sydenhan (1642-1689) (who is known as the "father of clinical medicine").

- a vector of individual variables maximizing utility subject to the budget constraint:

$$(c_t^i, d_t^i, b_{t+1}^i) = \arg\max u(c_t^i) + \phi(d_t^i, p_t)u(b_{t+1}^i) \text{ subject to } y_t^i = c_t^i + d_t^i + \frac{b_{t+1}^i}{R_{t+1}}$$

with income given by (1)- (2) and with savings s_t^i related to bequests b_{t+1}^i through equation (4);

- a vector of factor input maximizing profits

$$(L_t, K_t, X_t) = \arg\max AL_t^{1-\alpha-\theta}K_t^{\alpha}X_t^{\theta}\bar{K}_t^{1-\alpha} - w_tL_t - R_tK_t - \rho_tX_t$$

- a vector of medical effectiveness (p_t) and medical knowledge (M_t) satisfying (12), (13) and

$$M_t = (1-\delta)M_{t-1} + N^L d_t^L + N^W d_t^W$$

- a vector of prices (w_t, R_t, ρ_t) such that all markets clears

$$\begin{aligned} L_t &= N^W + N^L && (15)\\ K_{t+1} &= N^W s_t^W + N^L s_t^L && (16)\\ X_t &= 1, && (17) \end{aligned}$$

- an aggregate stock of capital $\bar{K}_t = K_t$.

4 The consumer problem

In this section we study the solution to consumer optimization problem. We first propose analytical results under some simplifying assumptions. In a second step we study the sensitivity of these results in a more general case, through numerical experiments. A specific objective is to prove that, though medicine is not effective in the sense that $p = 1/2$, people with a sufficiently high income level have an incentive to invest in health. To alleviate notation, we abstract for time and class indexes in this section.

4.1 A simplified model

We consider a simple version of the model in which we assume that there is no first period consumption; this implies that the only arbitrage is between leaving bequest and spending on health. We also assume the following specific functional forms:

$$u(b) = \frac{\sqrt{b}}{2}, \qquad v(d) = \frac{d}{1+d} \qquad f(d) = \tau\frac{d}{1+d} \tag{18}$$

where we assume $\tau \leq \beta \leq 1 - \tau$ to have a well defined life expectancy. Indeed this condition is necessary and sufficient to have, according to (8) and (9), $0 \leq \beta(h(d)) \leq 1$ when d tends to infinity.

Under the above simplifying assumptions, the individual optimization problem becomes:

$$\max_{d,b} \frac{\sqrt{b}}{2}\left(\beta + \tau\frac{d^2}{(1+d)^2} + (2p-1)(\beta+\tau)\frac{d}{1+d}\right) \tag{19}$$

s.t.

$$y = \frac{b}{R} + d \tag{20}$$

$$d,\ b \geq 0 \tag{21}$$

Substituting the budget constraint (20) into the objective, the problem can be stated as:

$$\max_{d\in[0,y]} W(d,y,p)$$

where:

$$W(d,y,p) = \frac{\sqrt{R(y-d)}}{2}\left[\beta + \tau\frac{d^2}{(1+d)^2} + (2p-1)(\beta+\tau)\frac{d}{1+d}\right] \tag{22}$$

We define the set of optimal levels of d as:

$$D^\star = D^\star(y,p) = \left\{d : d = argmax_{d\in[0,y]} W(d,y,p)\right\}.$$

$D^\star$ is not empty since $W(d,y,p)$ is a continuous function and $[0,y]$ is a closed and bounded set. An element of $D^\star$ is denoted by $d^\star = d^\star(y,p)$. In general $D^\star$ could contain interior solutions (i.e. $d^\star \in (0,y)$), corner solutions (i.e. $d^\star = 0$ or $d^\star = y$), or both.

To study the properties of $D^\star$ we compute:

$$W_d(d,y,p) = \frac{\sqrt{R}}{2}\left\{\sqrt{(y-d)}\left[\tau\frac{2d}{(1+d)^3} + (2p-1)(\beta+\tau)\frac{1}{(1+d)^2}\right]\right.$$
$$\left. -\frac{1}{2\sqrt{(y-d)}}\left[\beta + \tau\frac{d^2}{(1+d)^2} + (2p-1)(\beta+\tau)\frac{d}{1+d}\right]\right\} \tag{23}$$

We define:

$$\Gamma(d,y,p) \equiv W_d(d,y,p) \tag{24}$$

and list its main properties:

P1 $\Gamma(d,y,p)$ is continuous in its three arguments.

P2 When $d = 0$ we have:

$$\Gamma(0,y,p) = \frac{\sqrt{R}}{2}\left\{\sqrt{y}(2p-1)(\beta+\tau) - \frac{\beta}{2\sqrt{y}}\right\} \tag{25}$$

In particular we have that: if $p > 1/2$ and y is sufficiently high then $\Gamma(0,y,p) > 0$; if $p = 1/2$ then $\Gamma(0,y,p) < 0$.

P3 When $d = y$ we have

$$\Gamma(y,y,p) = \Gamma(d,d,p) = -\infty \tag{26}$$

P4 The effect of income is given by:

$$\Gamma_y(d,y,p) > 0 \tag{27}$$

P5 The value $\Gamma(d,y,p)$ when y goes to ∞ depends on the value of p and on the fact that $d = 0$ or $d \in (0,y)$:

$$\begin{aligned}
&\Gamma(d,\infty,p) = \infty \quad \forall\, d \in [0,y) \quad \text{if } p > 1/2 \\
&\Gamma(d,\infty,p) = \infty \quad \forall\, d \in (0,y) \quad \text{if } p = 1/2 \\
&\Gamma(0,\infty,p) = 0 \quad \text{if } p = 1/2
\end{aligned} \tag{28}$$

P6

$$\Gamma_p(d,y,p) > 0 \text{ for } d \leq d^\star(y,p) \tag{29}$$

See Appendix A for the proof.

We notice that **P3** implies $d^\star \neq y$: leaving nothing for bequest is never optimal; as a consequence $d^\star \in [0, y)$. We now prove in Proposition 1 that the solutions can be corner ($d^\star = 0$) or interior ($d^\star \in (0, y)$) depending on the value of y. For this purpose we define the minimum income such that we can have an interior maximum:

$$\begin{aligned} &\hat{y} = \hat{y}(p) = \\ &\min \{y > 0 : \exists \text{ at least one } d \in (0, y) \text{ such that } W(d, y, p) - W(0, y, p) \geq 0\} \end{aligned} \tag{30}$$

Proposition 1
(i) $\hat{y}(p)$ is finite $\forall p$.
(ii) $\forall y > \hat{y}(p)$ we have $d^\star \in (0, y)$, i.e. the optimal choices of d are all interior.
(iii) $\forall y < \hat{y}(p)$ we have $d^\star = 0$, i.e. $d = 0$ is the unique optimal choice.

Proof
(i) A necessary and sufficient condition for a $d^\star = 0$ is

$$W(d, y, p) - W(0, y, p) = \int_0^d \Gamma(s, y, p) ds \leq 0$$

$\forall\, d \in (0, y)$. According to **P5**, when income tends to infinity, $\Gamma(s, y, p)$ is ≥ 0 for $s = 0$ and it tends to infinite for $s > 0$. By continuity **P1**, this also holds true when income is finite but large enough. Hence, $W(d, y, p) - W(0, y, p)$ is positive for a finite large enough income.
(ii) From definition (30) we know that $W(d, \hat{y}, p) - W(0, \hat{y}, p) = \int_0^d \Gamma(s, \hat{y}, p) ds \geq 0$ for at least one $d \in (0, y)$. From **P4** we know that $\Gamma_y(s, \hat{y}, p) > 0$. Thus if $y > \hat{y}$ then $W(d, y, p) - W(0, \hat{y}, p) > 0$, implying that $d = 0$ is not an optimal choice.
(iii) It follows directly from the definition (30). ■

Proposition 1 implies that people with a sufficiently high level of income invest in health also when medicine is not effective, i.e. $p = 1/2$. This is our first result and it is the basic ingredient of our explanation of the rise of effective medicine.

To understand the idea behind Proposition 1, we can start with the simple remark that $d^\star$ is chosen comparing the cost of health investment (represented by the foregone amount of bequest), with its gain. This comparison is obviously affected by the income level. What could seem less obvious is the fact that the gain of investing in health is positive when $p = 1/2$. The intuition is the following. Consumers that invest in health face two possible states of the world: medicine could be a good or a bad. However, since the health status has an impact both on life expectancy and on

the quality of life of the second period, they do not assign to these states of the world the same utility. The expected utility of investing in health is given by the sum of the two following expressions:

$$p\beta u(b)v(d) + pf(d)u(b) + pf(d)u(b)v(d) \tag{31}$$

$$-(1-p)\beta u(b)v(d) - (1-p)f(d)u(b) + (1-p)f(d)u(b)v(d) \tag{32}$$

where (31) is p times the utility derived from investing in health when medicine is a good and 32 is $1-p$ times the disutility derived from investing in health when medicine is a bad. The first term in each expression is the quality of life effect (applied to the life expectancy computed without investment in health). The second term is the life expectancy effect (evaluated with the utility of second period computed without investment in health). The third one is cross effect, i.e. the positive (or negative) quality of life effect applied to the increase (or the reduction) in life expectancy. The first two terms may be positive or negative, depending of the fact that medicine is good or bad. However, the third term is always positive: if medicine is good it represents an increase in the number of years during which a better life effect is enjoyed; if medicine is bad, it represents a reduction in the number of years during which a low quality of life effect is experienced. It is clear that, due to presence of the cross effect, (31) is greater than (32) even when $p = 1/2$.

Given $d^\star$, the optimal level of bequest $b^\star$ is determined using the budget constraint (20). As a consequence we have that $b^\star \in (0, Ry]$ and in particular: $\forall y \geq \hat{y}(p)$ we have $b^\star \in (0, Ry)$, whereas $\forall y < \hat{y}(p)$ we have $b^\star = Ry$.

We now study the effects on $d^\star$ of changes in y and prove that health expenditure is a normal good.

Proposition 2

For $y \geq \hat{y}(p)$, $d^\star$ is strictly increasing in y.

Proof

The proof is a simple application of supemodularity to our one-dimensional choice problem (see Edlin and Shannon (1998)). Let us consider $d_1^\star = d^\star(y_1, p)$. If y_1 increases to y_2 then $d_2^\star = d^\star(y_2, p)$ cannot be below $d_1^\star$. Indeed , by definition of $d_1^\star$ and $d_2^\star$, we always have $W(d_1^\star, y_1, p) \geq W(d_2^\star, y_1, p)$ and $W(d_1^\star, y_2, p) \leq W(d_2^\star, y_2, p)$

and as a consequence $W(d_1^\star, y_2, p) - W(d_1^\star, y_1, p) \leq W(d_2^\star, y_2, p) - W(d_2^\star, y_1, p)$ (i.e. the effect of y on $W(d, y, p)$ is higher at $d_2^\star$ than at $d_1^\star$). If $d_1^\star \geq d_2^\star$ the last inequality contradicts **P4**, which is equivalent to $W_{yd}(d, y, p) > 0$. Therefore we have $d_1^\star \leq d_2^\star$. Moreover, since $W_d(d_1^\star, y_1, p) = 0$ for the first order condition, **P4** also implies that $W_d(d_1^\star, y_2, p) > 0$ and thus we can conclude that $d_1^\star < d_2^\star$. ■

Then we focus on the behavior of the propensity to save when y changes. The propensity to save is defined as:

$$\frac{s^\star}{y} = \frac{b^\star}{Ry} = 1 - \frac{d^\star}{y} \tag{33}$$

Proposition 3
(i) If $y < \hat{y}(p)$ *then* $\frac{s^\star}{y} = 1$.
(ii) If $y \geq \hat{y}(p)$ *then* $\frac{s^\star}{y} < 1$.
(iii) $\lim_{y\to\infty} \frac{s^\star}{y} \to 1$.

Proof
Using Proposition (1), we have that if $y < \hat{y}(p)$ then $\frac{s^\star}{y} = 1$ and if $y \geq \hat{y}(p)$ then $\frac{s^\star}{y} < 1$.

Then we define $x = d^\star/y$ and write the first order condition of the optimization problem in this way:

$$\begin{aligned}(1-x)\left[\tau\frac{2d^\star}{(1+d^\star)^2} + (2p-1)(\beta+\tau)\frac{1}{1+d^\star}\right] - \\ -\frac{1}{2}\left[\frac{1+d^\star}{d^\star}\beta + \tau\frac{d^\star}{1+d^\star} + (2p-1)(\beta+\tau)\right]x = 0\end{aligned} \tag{34}$$

By Proposition 2, $d^\star$ is an increasing function of income y. When $y \to \infty$, $d^\star$ goes either to some limit $\bar{d} < \infty$, or to $+\infty$. If it goes to $\bar{d}$, x goes to zero and $s^\star/y$ goes to 1. If $d^\star$ goes to $+\infty$ equation (34) becomes

$$2p(\beta+\tau)x = 0$$

which implies $x = 0$ and $s^\star/y = 1$. ■

Proposition 3 establishes that the relation between propensity to save and income has a U-shape: very rich people and poor people has an higher propensity to save than people with an average income.

We finally analyze the effects of changes in p, which is a measure of medicine effectiveness.

Proposition 4
(i) For $y \geq \hat{y}(p)$:

$d^\star$ is strictly increasing in p

$b^\star$ and $s^\star/y$ are strictly decreasing in p.

(ii) $\hat{y}(p)$ is strictly decreasing in p.

Proof See Appendix B. ■

According to Proposition (4) an increase in medicine effectiveness has a positive impact on investment in health and a negative one on bequests and propensity to save out of income. Moreover the threshold level above which people start investing in health decreases. This means that poor agents that do not invest in health when medicine is not efficient, may decide to invest in health once medicine effectiveness increases.

As we explain in the next section, Proposition 3 and part (i) of 4 turn out to be different in the general model where first period consumption is present.

4.2 The model with first period consumption

In the simplified model of Section 4.1, we prove a result that is crucial for the way in which we model the rise of effective medicine: people with a sufficiently high income invest in medicine also when medicine is not effective (i.e. $p = 1/2$). However there are two properties of the model of the previous section that are undesirable. Firstly, the propensity to save is not monotonically increasing in income. Secondly, better medicine will increase the propensity to save only by reducing the share of health expenditures on income. These two properties are a direct consequence of having abstracted from first period consumption. Indeed, in such a case, savings are negatively related to health expenditures through the budget constraint (20). This is no longer true if first period consumption is introduced, since the relation between savings and health expenditures turns out to depend on first period consumption through the budget constraint (3).

This last consideration and more generally the need to assess the robustness of the results derived in the previous section, motivate the inclusion of first period consumption. We perform such a task by way of numerical simulations.

With regard to the utility of bequests $u(b)$ and to the effect of health investment on the health status $v(d)$ and on the survival probability $f(d)$, we keep assumption (18). The functional form for utility of first period consumption is the same as the one of bequests: $u(c) = \sqrt{c}/2$. Thus agents maximize the following utility function with respect to b and d:

$$W(b,d) = \frac{\sqrt{y - \frac{b}{R} - d}}{2} + \frac{\sqrt{b}}{2}\left(\underbrace{\beta + \tau\frac{d^2}{(1+d)^2} + (2p-1)(\tau+\beta)\frac{d}{1+d}}_{\equiv\phi(d,p)}\right) \tag{35}$$

where we have used the budget constraints (5) to express the first period consumption c as a function of b and d. Finally for the numerical simulation of this section we put $\beta = 0.25$, $\tau = 0.1$ (this is the parametrization that we use in the simulation of the model that we perform in Section 5) and $R = 1$.

Figure 2 displays $\hat{y}(p)$. It is clear that $\hat{y}(p)$ exists and is finite for all level of p and in particular for $p = 1/2$ (the intuition of this result is provided in the previous section). Moreover $\hat{y}(p)$ turns out to be decreasing with p. Thus Proposition 1 and Proposition4 (ii) still hold true.

Simulations also show that investment in health increases in income and thus Proposition 2 is robust to the inclusion of first period consumption. Moreover bequests and first period consumption turn out to be normal goods too.

Then we look at the share of health expenditures on income and at the propensity to save and to consume. Figure 3 shows $d^\star/y$ and $s^\star/y$ as a function of y. $c^\star/y$ is in Figure 4 . The behavior of $d^\star/y$ is similar to the one of Section 4.1: the share of health expenditures on income is zero for people with an income below $\hat{y}(p)$, and then it follows an inverse U-shape. However this figure for $d^\star/y$ does not imply a behavior of $b^\star/y$ analogous to the one of the previous section. Indeed the propensity to save turns out to be a constant below 1 for income levels up to $\hat{y}(p)$ and then it is increasing. This is due to the fact that $c^\star/y$ decreases for $y > \hat{y}(p)$. The intuition can be understood, taking into account that $d^\star$ positively depends on y and that a higher $d^\star$ induces agents to increase the weight they put on the utility of the second period.

The fact that the propensity to save turns out to be increasing with income has two important implications. First it introduces a mechanism of intergenerational transmission of inequality: rich people devote to bequests a larger fraction of their income than poor people. Secondly it determines a positive relation between growth and in-

Figure 2: $\hat{y}(p)$

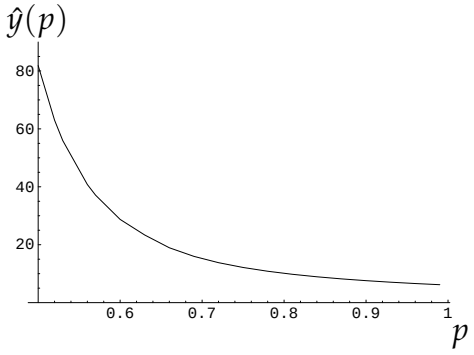


Figure 3: Health spending and savings income shares

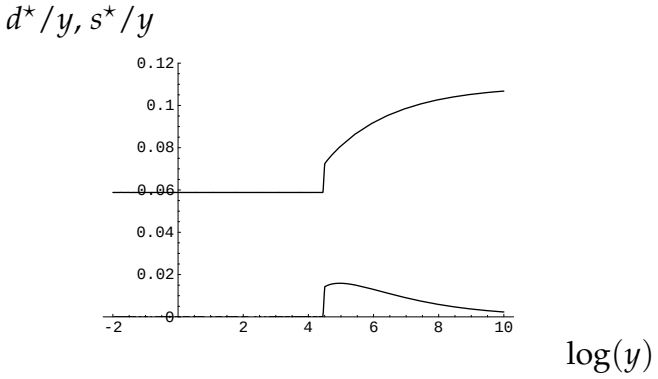


Figure 4: Consumption income shares

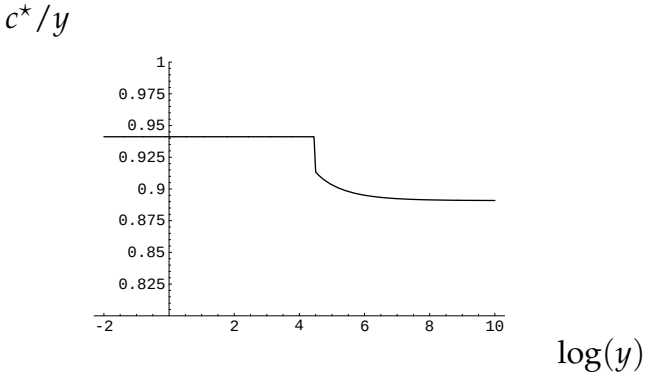
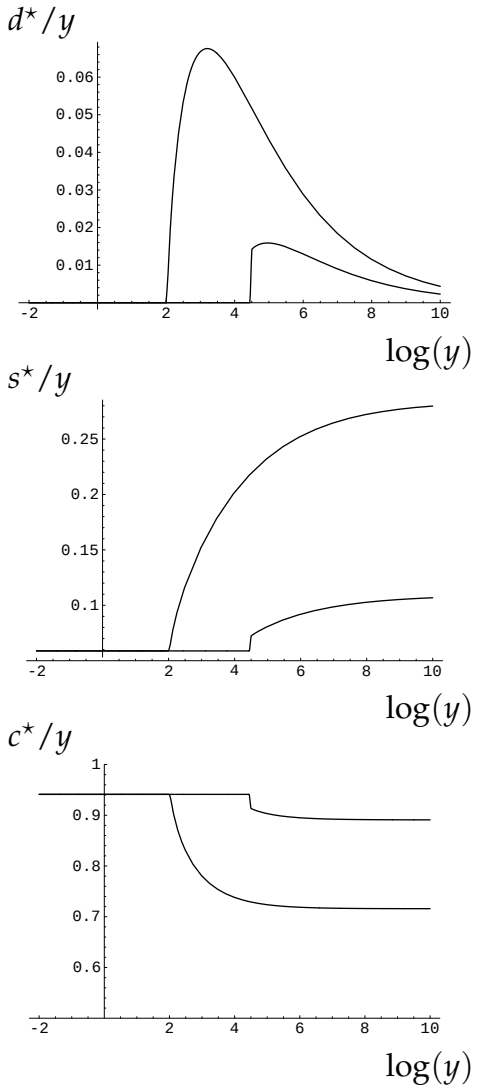


Figure 5: Effects of an increase in medicine effectiveness



equality: the higher is the share of income distributed to the rich, the higher is the the total amount of saving in the economy. This kind of relationship between growth and inequality is an assumption which is sometimes made in the growth literature for which we have proposed here a foundation based on the role of medicine. Such an assumption can be found in Galor and Moav (2004) who present a general discussion of all the possible channels through which inequality and growth could be related.

Finally in Figure 5 we display the effect of an increase in medicine effectiveness (i.e. a change from p to $p' > p$) on $d^\star/y$, $s^\star/y$ and $c^\star/y$. For income levels below $y(p')$, this effect is nought. For income levels above $y(p')$ and contrary to what is claimed in Proposition 4, both the share of health expenditure on income and the propensity to save increase in response to an increase in medicine effectiveness. Once again this is now possible since a rise in p causes $c^\star/y$ to drop for $y > y(p')$.

5 Dynamics

In the previous sections we present the building blocks of our model economy and we discuss in details the features of the individual optimization problem. In this section we discuss the dynamic implications of the results derived above.

Our aim is twofold. First we show that the model we have constructed is a consistent explanation of the empirical evidence on life expectancy presented in Fig. 1. Then we look at the implication of the model for growth, inequality and the relation between them. For this purpose it is crucial to study the behavior over time of the propensity to save. *Ceteris paribus*, an increase in the propensity to save of at least one of the two classes implies an increase in aggregate savings and as a consequence an increase in the growth rate of income. Moreover, as explained in the previous section, the higher is the propensity to save of the rich compared to the one of the poor, the higher is inequality and the stronger is the positive relation between inequality and growth.

For these purposes, we use a numerical simulation of the model. We do not have the ambition to calibrate our two-period overlapping generation model to actual data. We only give a numerical example of the dynamics generated by the model. We keep the same assumption on the utility function of section 4.2 ($\beta = 0.25$, $\tau = 0.1$). One period is 30 years. The values for the parameters of the production function are: $A00.525$, $\alpha = 1/3$, $\theta = 1/10$. As far as the evolution of medical knowledge is concerned, we initially want $p = 1/2$ and thus, according to (14), we need to choose

$M_0 < \bar{M}$. In particular we put $M_0 = 0$, $\bar{M} = 15$ and $\delta = 0.12$. For the function $g(M_t)$ we choose the following specification: $0.3\frac{M_t/10}{1+M_t/10}$. Thus $g'(M_t) > 0$, $g''(M_t) < 0$ and $\lim_{M_t \to \infty} g(M_t) = 0.3$. Finally we set the initial condition for the income of the two social classes: in period zero the income of the landlord is assumed to be higher than $\hat{y}(1/2)$, while the income of the commons is fixed below $\hat{y}(1/2)$.

Panels A and B of Figure 6 show the evolution over time of the propensity to save and of life expectancy for the landlords and the commons. The behavior of inequality (defined here as the ratio between the income of the landlords and the income of the commons) and of the growth rate of aggregate income are in panel C and D.

Looking at these figures we can distinguish three phases.

In the first phase medicine is ineffective (i.e. $p = 1/2$) and has no effect in terms of life expectancy. Only the landlords invest in health. As a consequence their propensity to save is slightly higher than the one of the commons.

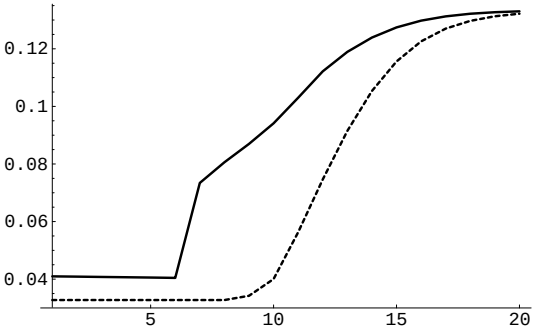
According to (14), medical experimental knowledge accumulate. At one point, medical experimentations are numerous enough to generate an improvement in medicine effectiveness. *A second phase* starts. The landlords goes on investing in health, while the commons still choose to not buy medical services. Life expectancy increases for the landlords and mortality differential widens. Stimulated by longer lives, the propensity to save of landlords strongly increases over time. This boosts the growth rate of income and inequality. Moreover the positive relation between inequality and growth is strengthened.

Thanks to growth in capital, and thanks to the increase in medicine effectiveness, the income of the commons reaches the threshold above which they also start to invest in health. In this *third phase* their life expectancy increases. Medical knowledge, stimulated by the growth in health expenditures, progresses rapidly. Asymptotically, the life expectancy of the poor catches up the one of the rich and they converge to a constant value. The propensity to save of the two groups follows the same path. As a consequence inequality decreases over time and the positive relation between growth and inequality disappears. In the long run, the growth rate of income tends to its balanced growth value.

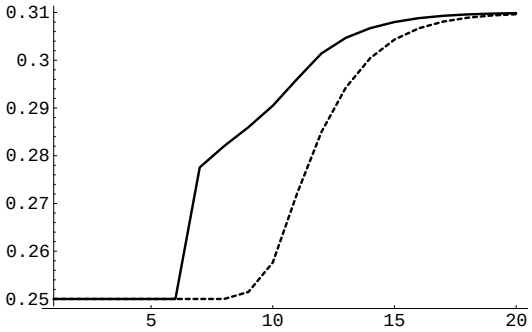
¿From the simulation above we can draw two main conclusions, concerning the implication for economic variables of our model economy. First, the evolution of medicine effectiveness may have contributed in England to the acceleration in the growth rate during the eighteenth century. Moreover it may also have played an important role

Figure 6: Example of dynamics

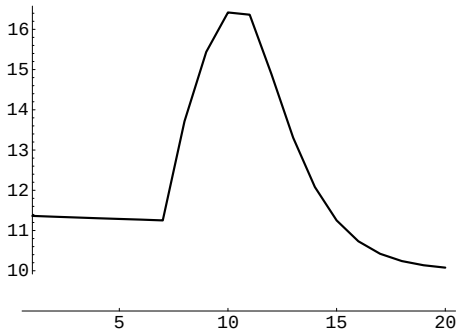
A. Propensity to save
Landlords (solid), commons (dots)



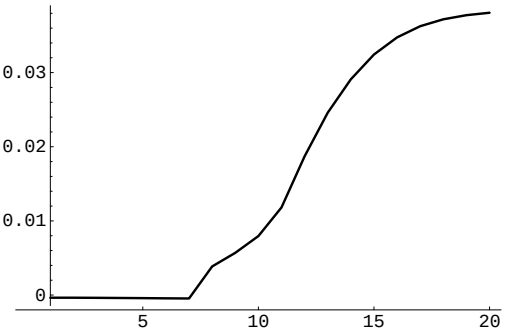
B. Life expectancy
Landlords (solid), commons (dots)



C. Inequality



D. Growth



in shaping the evolution of inequality and of the relation between inequality and growth. They both follow an inverted U. Initially, when the development process starts, inequality increases and the positive relation between inequality and growth is strong. Then, when there is a further increase in output, there is a trend towards a more equal distribution of income and the positive relation between inequality and growth disappears. This relation between the level of development and inequality qualitatively resembles the well known Kuznets' curve, for which our framework provides a theoretical justification [6]. As far as the empirical evidence is concerned, the existence of the Kuznets curve in England between the eighteenth and the nineteenth century seems to be corroborated by the evidence on income distribution (see Lindert (2000)) [7].

6 Conclusion

As discussed in section 1 many aspects of the relation between longevity, growth and inequality have been explored in the literature. However, to the best of our knowledge, no paper formally discuss the specific role played by the evolution of the effectiveness of medical science, which has been viewed by some historians (see section 2) as an important factor for understanding the evolution of life expectancy in England between the eighteenth and the nineteenth century.

Our first contribution in this paper is to build a model of medical spending that is suitable to be applied in a period, like pre-industrial Europe, where medicine effectiveness was very low. For this purpose we model medicine as an experience good: medicine can be a good with probability p_t and a bad with probability $1 - p_t$, and agents simply knows this probability distribution.

We give a rationale for individual health spending even when $p_t = 1/2$, i.e. when medicine was on average not effective. Combining this result with a simple learning process based on health expenditures, we are able to give a consistent explanation of the path of life expectancy in England for the different income groups: initially there are no difference in life expectancy of the rich and of the poor; then life expectancy

[6] For other explanations of the Kuznets curve see for example Acemoglou and Robinson (2002) and the reference therein

[7] However, given the limited reliability of available data, some caveats have to be added. Indeed to best guess the evolution of income inequality, the available information on income distribution should be also integrated with the analysis of movements in factor-price ratios and in inequality of wealth and of earnings (see Lindert (2000))

of the rich increases while life expectancy of the poor remains stagnant; finally life expectancy of the poor starts increasing and it catches up the one of the rich.

Then we use our model economy to look at the implication for economic variables. First the rise of effective medicine, producing an increase in life expectancy and thus in savings, positively affects the growth rate of income. Secondly we show that the evolution over time of medical effectiveness plays a role in shaping inequality and the strength of the relation between inequality and growth; inequality follows the same inverted U shape as the differential in mortality between the rich and the poor.

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A Proof of Property P6

¿From (23) we can compute:

$$\Gamma_p(d,y,p)\frac{\sqrt{R}}{2}(\beta+\tau)\left\{2\sqrt{(y-d)}\frac{1}{(1+d)^2}-\frac{1}{\sqrt{(y-d)}}\frac{d}{(1+d)}\right\} \tag{36}$$

Thus:

$$\Gamma_p(d,y,p)>0 \Leftrightarrow 2(y-d)-d(1+d)>0 \tag{37}$$

Using the first order condition derived from (23), inequality (37) at $d = d^\star$ can be written as:

$$\begin{aligned}\Gamma_p(d^\star, y, p) > 0 \Leftrightarrow & \frac{\beta + \tau\frac{d^{\star 2}}{(1+d^\star)^2} + (2p-1)(\beta+\tau)\frac{d^\star}{1+d^\star}}{\tau\frac{2d^\star}{(1+d^\star)^3} + (2p-1)(\beta+\tau)\frac{1}{(1+d^\star)^2}} - d^\star(1+d^\star) > 0 \\ \Leftrightarrow & [\beta-\tau]d^2 + 2\beta d + \beta > 0\end{aligned} \tag{38}$$

which always holds true since we have assumed $\beta \geq \tau$. Moreover from (36) we see that $\Gamma_p(d, y, p)$ is decreasing in d. Thus we can conclude that $\Gamma_p(d, y, p) > 0 \; \forall d < d^\star$.

B Proof of Proposition 4

(i) The proof is similar to the one used in Proposition 2. Let us consider $d_1^\star = d^\star(y, p_1)$. If p_1 increases to p_2, then $d_2^\star = d^\star(y, p_2)$ cannot be below $d_1^\star$. Indeed, by definition of $d_1^\star$ and $d_2^\star$, we always have $W(d_1^\star, y, p_1) \geq W(d_2^\star, y, p_1)$ and $W(d_1^\star, y, p_2) \leq W(d_2^\star, y, p_2)$ and as a consequence $W(d_1^\star, y, p_2) - W(d_1^\star, y, p_1) \leq W(d_2^\star, y, p_2) - W(d_2^\star, y, p_1)$ (i.e. the effect of p on $W(d, y, p)$ is higher at $d_2^\star$ than at $d_1^\star$). If $d_1^\star \geq d_2^\star$ the last inequality contradicts **P6**, which is equivalent to $W_{pd}(d, y, p_1) > 0 \; \forall d < d_1^\star$. Therefore $d_1^\star \leq d_2^\star$. Moreover since $W_d(d_1^\star, y, p_1) = 0$ for the first order condition, **P6** also implies that $W_d(d_1^\star, y, p_2) > 0$ and thus $d_1^\star < d_2^\star$. Finally using the budget constraint (20) and equation (33), we can also determine how $b^\star$ and $\frac{s^\star}{y}$ react to a change in p.

(ii) By definition (30), $W(d, \hat{y}(p), p) - W(0, \hat{y}(p), p) \geq 0$ for at least one $d \in (0, y)$.

Using (22) to compute:

$$\begin{aligned}W(d, y, p) - W(0, y, p) = & \frac{\sqrt{R(y-d)}}{2}\left[\beta + \tau\frac{d^2}{(1+d)^2} + (2p-1)(\beta+\tau)\frac{d}{1+d}\right] \\ & - \frac{\sqrt{Ry}}{2}\beta\end{aligned} \tag{39}$$

we can notice that $W(d, y, p) - W(0, y, p)$ is strictly increasing in y and p.

Thus if p increases to $p\prime$ then $W(d, \hat{y}(p), p\prime) - W(0, \hat{y}(p), p\prime) > 0$. Since $W(d, y, p)$ is continuous in y can choose $y < \hat{y}(p)$ and have $W(d, y, p\prime) - W(0, y, p\prime) \geq 0$. As consequence, we can conclude that $\hat{y}(p\prime) < \hat{y}(p)$.