Some Further Notes on "Was Malthus Right? Economic Growth and Population Dynamics"*

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November 5, 2001

Abstract

These notes present further discussion and details of several aspects of "Was Malthus Right? Economic Growth and Population Dynamics". They should be readed following each particular section of the main paper.

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"The problems of population, that is to say the question what it is that determines the size of human population and what the consequences are that attend the increase or decrease in the number of a country's inhabitants, might well the first to occur to a perfectly detached observer as soon as he looks at those societies in a spirit of scientific curiosity."

Joseph A. Schumpeter.

1. The general case of demographic transition

The demographic transition is well documented in literature. Indeed, Guillard (1855), the inventor of the word 'demography', already noticed the close relationship between levels (and trends) of mortality and fertility. Thompson (1929) developed the concept of demographic transition, generally accepted today as an accurate description of historical and cross-sectional data. Basically, this term names the process of change from a regime of high fertility-high mortality, predominant in most of the history of humanity, to the actual situation (at least in developed countries) of low fertility-low mortality. This change is characterized by a period of time where the mortality fall begins several decades before than the fertility fall, generating a huge surge in population. This increase in population is maintained for a while after the actual decline in fertility *per women* is achieved because of the so-called *momentum* of the population: even if each fertile woman is having less children, the big size of fertile cohorts keeps the fertility rate *per inhabitant* relatively high for several years[1].

The effects of the transition have been dramatic. Worldwide population has jumped from less than one billion to around six billions and it will probably only level off around ten or eleven billion (the exact figure is under strong dispute).

Mortality rates began to fall during the middle of the eighteen century in England, France, North America and Scandinavian countries and continued that process for about 75 years, including progressively more European countries. After a mini crisis in the middle decades of the nineteenth century, the decline reappeared again at the end of the century and it is still going on worldwide[2].

Understanding this fall was the main reason behind the birth of the scientific study of mortality in the second decade of the twentieth century. The scientific study of mortality began and relied extensively for some time on the use of parish record of burials (Gooner (1913)). For decades, it seemed that the main conclusion of the study of those records was that the first decline in mortality during the eighteenth century corresponded basically to the elimination of traditional mortality crisis (as first pointed out by Meuvret (1946) and pushed through by Goubert (1960) in a famous study of the french village of Beauvais). However, more extensive documentation (Wrigley and Schofield (1981) or Dupâquier (1989)) compiled at national levels for most European countries suggested otherwise: about ninety percent of the reduction in mortality was due to falls in normal causes of death (although the

[1] This is, for instance, the case today of several asian countries as China, where fertility is around replacement levels but population is still growing.

[2] Actually, strictly speaking, the observed mortality rate has gone up a little in developed countries because of the change in the poblational structure. It is the mortality rate among any given demographic group what keeps falling.

disappearance of mortality crises is a major cause in the reduction of variance in mortality). Indeed the existing evidence supports the idea that big famines were at a lower order of magnitude in comparison with chronic malnutrition as a death cause[3].

Precisely the increment of average caloric consumption and the subsequent remise of malnutrition were suggested by McKeown (1976) as the most likely candidates to explain this first decline in mortality although he could only proved it inferentially.. Finding supporting evidence has shown itself rather complicated given the available historical data. An important report is in Fogel and Floud (1994). They produce a series for *per capita* calories intaking from the estimates of English food production compiled by Holderness (1989) and Allen (1994) to show an increment from 1802 calories at the beginning of the eighteen century to 2346 a century after. But this emphasis in consumption raised the controversial issue of the effect of medical improvements over those years. McKeown also argued that health measures and medical advances did not become effective until late in the nineteenth century, leaving alone increments in consumption as the major cause of decline in mortality. This assertion created a huge controversy. Lee (1981) showed little correlation between short-term variations in death rates and wheat price although this absence of a short-term relation is not a definite proof as the effects of malnutrition, in comparison with famines, can take years to show up and failures in distribution networks can imply that the use of variations in prices grossly overestimates variations in food supply. Razzel (1974) pointed out a more interesting finding: mortality rates of nobility rapidly fell after 1725 although there is no sign of change in the diet of the peerage[4]. Finally Preston (1991) documented extensively the development of scientific knowledge of causes of death and the subsequent improvement in public health over those decades, specially after the quick diffusion of the smallpox vaccine, discovered by Jenner in 1796.

The central decades of the nineteenth century show a pause in the decline of mortality. For instance, that pause began in England around 1830 (Floud and Harris (1996)) and a decade later in United States around 1840 (Costa and Steckel (1995)). Those year also coincide grossly with the great famines in Ireland. Several explanations has been proposed to explain that detour: industrial revolution, overpopulation or the growth and crowding of inner cities.

The industrial revolution could cause the stop in the fall as a consequence of deteriorating conditions of the working class or because of an increment in inequality. However, even if evidence is controversial, it seems (Crafts (1997)) that if a fall in standard of livings ever happened, it was neither general nor drastic. However as data on height show a decline in low class males stature but none in upper classes one, this difference could explain a small amount of the aggregate movement on death rates.

Overpopulation has been proposed as an explanation by Sandberg and Steckel (1988) for the Swedish case. They show how the fall in stature and the increase in child mortality in west Sweden (the most populated area of the country) as an evidence of population surplus.

[3]A statistical argument explains that fact: famines were local and with low pairwise correlation, so in the aggregate they smoothed out. Wrigley and Schofield (1991) show that between 1550 and 1750 mortality crisis accounted for less than 6% of total English deaths.

[4]McKeown argued that the improvment in general health of the population caused a positive externality among peerage because their exposure to infection decreased. Fogel (1986) also suggests a high consumption of alcohol by pregnant mathers as a major cause of child death among peerage and a decline of that habits after 1725. Both reasons should be able to explain this paradox.

However, this evidence remains elusive as not enough additional data have been presented.

In contrast, the effect of cities on mortality through contaminated water supply and more exposure to contagious diseases seemed dramatic. In the United States *circa* 1830, cities with more than 50.000 inhabitants had a death rate more than double than rural areas (Fogel *et al.* (1978) and Haines and Anderson (1988). Bairoch (1988) presents similar data for Europe). Moreover, it is not as much the size of the city as its rate of growth what matters as a strong growth rate tend to strain public utilities as sewerage and waste disposal (indeed as cities were upgraded and modern health system built, they turned out to be healthier environments than the countryside). And the observed growth of cities was indeed without precedent in history. In England, the proportion of population living in towns of more than 2500 inhabitants increased from 34 per cent to 54 per cent between 1801 and 1851 (Wrigley (1987)).

Table 1. Death Rates (per 1000).

Period	Western Europe	Eastern Europe
1800-1820	28.0	38.0
1821-1830	26.1	38.0
1831-1840	27.2	38.0
1841-1850	26.2	38.0
1851-1860	26.1	38.0
1861-1865	25.5	36.3
1866-1870	26.8	35.6
1871-1875	26.5	37.0
1876-1880	24.9	35.1
1881-1885	24.3	34.2
1886-1890	23.4	33.3

Source: Chesnais (1992)

After that momentary pause, mortality rates reassumed their downward trend in most European countries countries around 1870 and in America several years later (Haines (1995) shows that the sustained fall in mortality did not begin until around 1880), accelerating after the discovery by Pasteur in 1884 of the microbic origin of infectious diseases. Immediately after the first world war, most non-European countries experienced a sudden decline in mortality (most significantly China and India) that reached Africa in the fifties, as the emergence of antibiotics (Flemming discovered penicillin in 1929 and its synthesis in 1943) and modern vaccines led to an efficient treatment of contagious diseases. Nowadays, mortality is still falling worldwide, slowly in rich countries, and rapidly in poor ones and there is now clear agreement about if this process has some kind of biological barrier.

The history of fertility is both simpler and less informative. It is simpler since the fall in fertility occurred later in time and, except for the *baby boom* after the second world war, rather monotone and rapid. At the same time it is less informative as the experiences of the premodern world were widely different. For instance, it is well known the dissimilar reproductive patterns of the European nations west and east of the imaginary line between Trieste and Saint Petersbourg, since west of that line, from the Renaissance on (and even before in some cases) fertility was controlled via nuptiality (delayed marriage and lifelong celibacy).

However, thanks to the recopilation of data accomplished by the *European Fertility Project* undertaken in 1963 by an international team of demographers, the statistical picture of the process seems clear. First declines in fertility appeared in France around 1760[5] but a general phenomenon of fertility restriction does not appear in most rich countries until 1870, well over a century after the initial improvement in life expectancy. Not only did fertility not decrease on the eve of industrial revolution but there is evidence that in England reproduction rates increased substantially in the last decades of eighteenth century and beginning of the nineteenth (Wrigley and Schofield (1981)).

The modern phenomenon of fertility was rather simultaneous over western populations, moving from Scandinavian countries (Sweden and Norway) to northern European ones (England, Netherlands) to the center and south of Europe as well as European stock populations (United States, Australia, Argentina)[6]. It is important to notice that most of this fall was consequence of a reduction in marital fertility and not in a retardation of marriage age or increments in the share of population practicing celibacy.

The decline of fertility in poor countries does not appear until after the second world war, first in the middle fifties in small, densely populated societies as Cyprus, Mauritius, Taiwan or Singapore and in the sixties in middle income countries as Brazil or Egypt. China and India began the fall around 1970, specially pushed in China by an aggressive public policy. Today, extremely high fertility rates are only found in specific cases as Afghanistan, Haiti or Laos and in Arab middle east (where several countries keep a strong pro-fertility policy) and most subsaharian Africa.

Table 2. Fertility Rates (per 1000).

Period	1850	1875	1900	1925	1950	1975
France	26.7	25.4	21.7	18.8	20.7	15.0
Germany	35.6	39.1	35.5	20.3	16.0	10.6
Netherlands	33.0	36.2	32.0	24.5	23.3	13.8
Norway	31.5	31.0	29.6	20.1	19.3	14.2
Spain	-	38.5	34.8	29.2	21.1	18.1
United Kingdom	36.8	36.5	29.1	18.7	16.4	13.3

Source: Chesnais (1992) and own calculations.

At the same time, most rich countries experienced after the second world war a general increment in the number of births, commonly known as the 'baby boom'. For instance, in the United States, the country where this phenomenon was more important, reproduction rate went form 2.19 in 1940 to 3.58 in 1957. Despite important efforts to explain that substantial rise (the classical study by Easterlin (1968) attributes the change to increases

[5] France case is special in a lot of aspects as it keep a extremely low fertility over the nineteenth century. It seems that the land propiety system, based in a huge number of fragmented owners (and exacerbated by the repartisions of land during the Revolution) forced down fertility as farmers tried to avoid further subdivisions of already too small plots.

[6] If the fertility rate is to be considered to test that statement, some care must be taken about the strong inmigration of young people in that years. Without appropiate corrections, Spain and Ireland, countries of strong immigration and low income levels, would seem to lead the reduction in fertility around 1865. The reproduction rate usually does not suffer from that problem.

in relative income) the causes of the 'baby boom' are still unknown. A second short-run movement in fertility could have happen in the last decades as fertility rates declined below replacement levels in several countries and now seem to be raising again, although data are still inconclusive.

2. The Environment

2.1. Households

Description of probabilities:

1. We can think of s_t^t as the probability of surviving gestation and birth. Even if in demography it is common to include in the birth process the delivery and the first 24 hours of life (when most post-natal deaths occur), in the model the 'prenatal' period spans the first year of life.

2. The age-specific parameters that account for different mortality risk over life can change over time because of scientific discoveries, diffusion of medical knowledge or adaptation of different social institutions as health systems.

3. There is wide evidence of the relation between nutrition and other consumption expenditures and infection (Scrimshaw et al. (1968)).

The reference to dynamic inconsistency is in the sense of Strozt (1957).

Fertility decisions: $n_t \in \left(0, \bar{n}\right)$ since descendants must be nonnegative and there is a maximum biological capacity of reproduction known as *natural fertility*. For a social group, the highest childbearing record documented is hold by the Hutterites, a group of Anabaptist who lives in communes in Canada and the Dakotas. In the thirties hutterite women were averaging more than 12 children per woman (Westoff and Westoff (1971)), than in the framework of the present model would be equivalent to a bit more than 6 *per* households. It is assumed that parents have all the children at the beginning of the period. Note also that we do not require independence of the realizations of survival.

Interpretation of altruism: $b(n_t) \geq 0$ (individuals never dislike having children), $b^{'}(n_t) > 0$ (having more children is better than less), $b^{''}(n_t) \leq 0$ (some form of nonincreasing marginal utility of having children) and $b\left(\bar{n}\right) < 1$ (a bound on total altruism). Other constraints we may impose are $b^{'}(n_t)\, n_t + b(n_t) \leq 0$ (having more children is better than less), $b^{''}(n_t)\, n_t \leq 2b^{'}(n_t)$ (some form of nonincreasing marginal utility of having children) and $b\left(\bar{n}\right) \leq \frac{1}{\bar{n}}$ (a bound on the total degree of utility from having children given the biological constraint).

Bequest: the existing empirical evidence (Hurd (1989)) indicates that most bequests are accidental, as the result of uncertainty about the date of death, and that planned bequest are small on average.

The difference between the services of skilled and unskilled labor allows us basically to consider different types of labor and the effect of changes in its relative prices even with within cohort homogeneous agents.

Household problem is not convex because of the term $e_{t+1}s_{1t}n_t$ on the budget constraint. This feature makes it difficult to find analytic expressions for optimality (although not computation beyond higher time requirements). However, as shown below, comparative statics intuition can be carried over using slope arguments about the value function, following Milgrom and Shannon (1994). Also note that another interesting point is the mix between finite-horizon dynamic programming and infinite-horizon in the household problem. This mix follows from the life cycle considerations involved in the accumulation of assets. However this mix does not change usual methods of solution.[7]

Accounting: let $\sigma(\Omega)$ a sigma-algebra defined over the power set of $\mathcal{V}$ and the Borel sets of $A \times H$. Some other accounting identities are active population $L_t \equiv s_{1t-1}N_{t-1} + s_{2t-2}N_{t-2}$, the labor supply, in units of human capital $H_t \equiv s_{1t-1}l_t^{t-1}h_{t-1}N_{t-1} + s_{2t-2}h_{t-2}N_{t-2}$, the reproduction rate $R_t \equiv \frac{N_t}{s_{1t-1}N_{t-1}}$ and the population growth rate $\lambda_t \equiv \frac{N_t}{N_{t-1}} - 1$.

2.2. Firms

Elasticities restriction can also be written as $\frac{1}{1-\rho} > \frac{1}{1-\omega}$.

Other reference for elasticities: Brown and Christensen (1981).

Taking as given the aggregate production function and endowments $\{K_t, U_t, S_t\}$, the vector of prices $\{w_t^s, w_t^u, r_t\}$:

$$w_t^u = A_t\left[\left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}} + U_t^{\rho}\right]^{\frac{1}{\rho}-1} U_t^{\rho-1} \quad (1)$$

$$w_t^s = A_t\left[\left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}} + U_t^{\rho}\right]^{\frac{1}{\rho}-1} \left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}-1} S_t^{\omega-1} \quad (2)$$

$$r_t = A_t\left[\left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}} + U_t^{\rho}\right]^{\frac{1}{\rho}-1} \left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}-1} B_tK_t^{\omega-1} \quad (3)$$

Then, the skill premium π_t (Katz and Murphy (1992)) is given by the ratio:

$$\pi_t = \frac{w_t^s}{w_t^u} = \frac{U_t^{1-\rho}}{S_t^{1-\omega}}\left((B_tK_t)^{\omega} + S_t^{\omega}\right)^{\frac{\rho}{\omega}-1} \quad (4)$$

or in logs $\log \pi_t = \alpha_1 \log U_t + \alpha_2 \log S_t + \alpha_3 \log\left((B_tK_t)^{\omega} + S_t^{\omega}\right)$ where $\alpha_1 = 1-\rho$, $\alpha_2 = \omega - 1$ and $\alpha_3 = \frac{\rho}{\omega} - 1$. If we normalize by U_t ($ku_t \equiv K_t/U_t$ and $su_t \equiv S_t/U_t$) and take first differences, the skill-premium change follows $\Delta \log \pi_t = \alpha_2 \Delta \log su_t + \alpha_3 \Delta \log\left((A_tku_t)^{\omega} + su_t\right)$.

2.3. Equilibrium

The aggregate states are the levels of the technology, A_t, q_t, B_t and m_t, and the distribution of population $\Psi_t(v, da, dh)$. However, since the evolution of the technology is assumed to be deterministic and each value function is already indexed by date, time is a sufficient statistic for the technologies, avoiding non-stationarity problems. There is no aggregate uncertainty since the idiosyncratic risk of death is integrated out by the application of the law of large numbers. In addition, each household must also keep track of its human capital level, h. After this characterization of states, a definition of equilibrium is the following:

[7] An intuitive way to think about that is comparing this life cycle problem with the problem of a household that lives just one period and consumes three different goods with a nonserable utility function.

Definition 1. *A Recursive Competitive Equilibrium is a value function* $V_t(h_t; \Psi_t(\cdot))$ *and a set of policy functions* $\{c_j^t(h_t; \Psi_t(\cdot)), l_j^t(h_t; \Psi_t(\cdot)), a_j^t(h_t; \Psi_t(\cdot)), n_t(h_t; \Psi_t(\cdot))\}$ *for each generation of households, and functions for allocations* $\{Y_t(\Psi_t(\cdot)), C_t(\Psi_t(\cdot)), X_t(\Psi_t(\cdot))\}$*, aggregate inputs,* $\{K_t(\Psi_t(\cdot)), U_t(\Psi_t(\cdot)), S_t(\Psi_t(\cdot))\}$*, their rental prices* $\{r_t(\Psi_t(\cdot)), w_t^u(\Psi_t(\cdot)), w_t^s(\Psi_t(\cdot))\}$*, the relative price of capital* q_t *and a law of motion* $\Phi_t(\Psi_t(\cdot))$ *for the measure of the population measure* $\{\Psi_t(\cdot)\}$ *for all* t *such that:*

1. *All households solve their recursive problem:*

$$\begin{aligned} V_t(h_t; \Psi_t(\cdot)) &= \max_{c_{t+1}^t, c_{t+2}^t, c_{t+3}^t, e_{t+1}, n_t \leq \bar{n}} u\left(c_{t+1}^t\right) + \beta E u\left(c_{t+2}^t\right) + \\ &\quad + \beta^2 E u\left(c_{t+3}^t\right) + b(n_t) E V_{t+1}(h_{t+1}; \Psi_{t+1}(\cdot)) \end{aligned} \tag{5}$$

$$\begin{aligned} s.t. \left(1+\kappa_1+\kappa_2 s_t^t\right) c_{t+1}^t + a_{t+1}^t &= \left(w_{t+1}^s h_t + w_{t+1}^u\right) l_{t+1}^t + tr_{t+1} \\ c_{t+2}^t + a_{t+2}^t &= w_{t+2}^s h_t + w_{t+2}^u + (1+r_{t+2}) a_{t+1}^t + tr_{t+2} \\ c_{t+3}^t &= (1+r_{t+3}) a_{t+2}^t + tr_{t+3} \\ l_{t+1}^t &= (1 - e_{t+1} s_{1t} n_t),\ l_j^t \in [0,1] \end{aligned}$$

2. *The lump-sum transfers are equal to the aggregate involuntary bequests:*

$$tr_t \int_{j=2,3,4} d\Psi_t(j, da, dh) = \int_{j=2,3,4} \left(1 - s_j^t\right) a_j^t(h_t; \Psi_t(\cdot)) \, d\Psi_t(v, da, dh) \tag{6}$$

3. *Prices for inputs are the marginal productivities:*

$$w_t^u = A_t \left[\left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}} + U_t^\rho\right]^{\frac{1}{\rho}-1} U_t^{\rho-1} \tag{7}$$

$$w_t^s = A_t \left[\left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}} + U_t^\rho\right]^{\frac{1}{\rho}-1} \left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}-1} S_t^{\omega-1} \tag{8}$$

$$r_t = A_t \left[\left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}} + U_t^\rho\right]^{\frac{1}{\rho}-1} \left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}-1} K_t^{\omega-1} \tag{9}$$

4. *Markets Clear:*

$$C_t = \int \left(1+\kappa_1+\kappa_2 s_t^t\right) c_j^t(\cdot) \, I(v) \, d\Psi_t(v, da, dh) \tag{10}$$

$$K_t = \int a_j^t(\cdot) \, d\Psi_t(v, da, dh) \tag{11}$$

$$U_t = \int l_j^t(\cdot) \, d\Psi_t(v, da, dh) \tag{12}$$

$$S_t = \int l_j^t(\cdot) \, h_j^t d\Psi_t(v, da, dh) \tag{13}$$

$$K_{t+1} = (1-\delta) K_t + X_t \tag{14}$$

$$Y_t = C_t + \frac{X_t}{q_t} = A_t \left[\left((B_t K_t)^\omega + S_t^\omega\right)^{\frac{\rho}{\omega}} + U_t^\rho\right]^{\frac{1}{\rho}} \tag{15}$$

5. *And the law of motion $\Phi_t(\Psi_t(\cdot))$ is consistent with individual behavior:*

$$\Psi_{t+1}(\cdot) = \Phi_t(\Psi_t(\cdot)) \tag{16}$$

This function can be written explicitly as:

$$\Psi_{t+1}(1, da', dh') = \int_{a'=a(1,da,dh),\ h'=h(1,da,dh)} s_{t+1t} d\Psi_t(2, da, dh) \tag{17}$$

$$\Psi_{t+1}(2, da', dh') = \int_{a'=a(1,da,dh)} s_{t+1t-1} d\Psi_t(1, da, dh) \tag{18}$$

$$\Psi_{t+1}(3, da', dh') = \int_{a'=a(1,da,dh)} s_{t+1t-2} d\Psi_t(2, da, dh) \tag{19}$$

$$\Psi_{t+1}(4, da', dh') = \int_{a'=a(1,da,dh)} s_{t+1t-3} d\Psi_t(3, da, dh) \tag{20}$$

Two aspects of this definition deserve some comment. First the presence of $I(v)$ in the consumption aggregator. This is an indicator function with value 1 if $v = 2$ and 0 otherwise. It takes account of the described cost of children in terms of the consumption good. It is important to note that the cost of educating children does not appear explicitly in the definition. The reason is simple: education is costly in terms of time, not resources. This time cost appears in the labor supply and then, via aggregation, in a lower final output Y_t. This raises the question of how to account for this education activity. Here a simple (and arguably arbitrary) solution is adopted: as the service of education in not sold in the market, it is not counted as final output. The second issue is the distribution of involuntary bequests in a lump-sum fashion to the rest of households. This quantity is just computed as the ratio between bequest and the total adult population.

In a *Steady State Competitive Equilibrium* the level of the technologies is constant over time. Note that if $\frac{1}{1-\rho}$ is big enough endogenous growth is possible.

3. Parametrizing the Model

This section describes how I choose standard functional forms and accepted parameter values to facilitate comparison with other studies, especially since, as suggested by Browning, Hansen and Heckman (1998), choices based on micro evidence gathered in very different contexts can lead to misleading choices.

3.1. Stationarity of the Data

Unit Root test. The pretesting regression $x_t = \alpha + \delta t + \varepsilon_t$ is estimated for $x_t \in \{CBR, CDR\}$ with the results below[8].

Trend Test

	$\widehat{\delta}$	P-value	P-value (robust s.e.)
CBR	0.00	0.75	0.78
CDR	0.03	0.40	0.48

[8]The test involving δ are asymptotically equivalent to the usual t-test (even if convergence rates change).

Let $x_t \in \{CBR_t, CDR_t\}$ and $\overline{x}$ be its sample mean. The numerator of the statistic is:

$$\eta = \frac{\sum_{i=1}^{T}\left(\sum_{t=1}^{i}(x_t - \overline{x})\right)^2}{T^2} \tag{21}$$

and the denominator is a consistent estimator of $\sigma^2 = \lim_{T\to\infty} \frac{E\left(\sum_{t=1}^{T}(x_t-\overline{x})\right)^2}{T}$. The likely presence of weakly dependent and heterogeneously distributed observations suggests using the Newey-West (1987) estimator:

$$s^2(q) = \frac{\sum_{t=1}^{T}(x_t - \overline{x})^2}{T} + \frac{2}{T}\sum_{v=1}^{q}\left(1 - \frac{v}{q+1}\right)\sum_{t=v+1}^{T}(x_t - \overline{x})(x_{t-v} - \overline{x}) \tag{22}$$

with the Barlett Spectral Window to assure the positivity of $s^2(q)$[9]. The values (with the 10% significance level in parenthesis[10]) are 0.2 (0.347) for the birth rate and 0.16 (0.346) for the death rate. These results strongly support the null hypothesis of stationarity and, with the non-significativity of the time trend, the existence of constant first moments.

Leybourne and McCabe (1994) propose an alternative test, analogous to an Augmented Dickey-Fuller, with higher power. However, their test accounts for autocorrelation in a parametric fashion. I am reluctant to accept this feature given the stated lack of restrictions in the data process.

Robustness is checked changing the burden of the proof with a test of the null of the presence of a unit root. To minimize the well-known size and power small sample problems of unit root test against local alternatives, two different test are performed.

The first test is the Augmented Dickey-Fuller (Dickey and Fuller (1979))[11]. For $x_t \in \{CBR, CDR\}$ the regression:

$$x_t = \alpha + \rho x_{t-1} + \sum_{i=1}^{k-1}\phi_i \Delta x_{t-i} + \varepsilon_t \tag{23}$$

is estimated and the t-statistic $\frac{\widehat{\rho}-1}{\widehat{\sigma_\rho}}$ computed for different lags. The values of the test are (in parenthesis the last significative difference lag)[12]:

	t-statistic	P-value
CBR ($k=1$)	-2.41	0.01
CDR ($k=0$)	-5.73	0.00

[9] For consistency a rate $q = o\left(T^{\frac{1}{2}}\right)$ is appropriate under both the null and the alternative. For a sample of 52 observations $q = 7$. To check robustness $q = 6, 5, 4$ and 3 are tested without any rejection at conventional levels.

[10] The asympototic distribution of the test is $\frac{\eta}{s^2(l)} \overset{w.c.}{\to} \int_0^1 (W(r) - rW(1))^2\, dr$ where $W(r)$ is a Brownian Motion.

[11] Nonparametric treatments of serial dependence (as based on Phillips (1987)), even if more intuitively apelling, suffer important power problems in small samples (see Nabeya and Tanaka (1990)).

[12] Note that under the null, the limiting distribution in both cases is Gaussian.

The point estimations of ρ (0.64 and 0.2), despite small-sample downward bias, seem also too separate from 1 for the unit root hypothesis. Thus the evidence against the presence of a unit root is also quite strong, coinciding fully with the findings in the stationary test.

The second test is the Elliot, Rothemberg and Stock (1996). The estimated regression now is:

$$(x_t - \overline{x}) = \rho \left(x_{t-1} - \overline{x}\right) + \sum_{i=1}^{k-1} \phi_i \Delta \left(x_{t-i} - \overline{x}\right) + \varepsilon_t \tag{24}$$

This test has an asymptotic power function tangent to the power envelope at one point selected given the sample size and never falls below that envelope. That feature substantially improves the power and the small sample behavior of the test. Critical values and asymptotic power are those of the conventional Dickey-Fuller t statistic for $\rho = 0$ when there is no intercept. The results are:

	t-statistic	Critical Value (0.05)
CBR ($k = 1$)	-2.42	-1.95
CDR ($k = 2$)	-0.65	-1.95

The result of this test are mixed. It is able to reject at 5 per cent significativity the null of unit root in fertility (but not at the 1 per cent level). However it is not able to reject the null of a unit root in the mortality rate. The reason probably is related with the higher variance induced in the series by the presence of a few outliers in the data or the fall in the rate toward the end of the sample. However, it is known that with a number high enough of lags this will be the case for most series as the size of the acceptance interval grows larger. In addition it is well known that Unit Root Tests lack enough power against local alternatives in small samples (Blough (1992)).

3.2. A Simulated Method of Moments

The fluctuations of the age-specific components are unobserved by the researcher, not by the households who know their evolution. For an exposition of this interpretation see Sargent (1981).

Simulated Method of Moments references: McFadden (1989), Pakes and Pollard (1989) and in a time series context Lee and Ingram (1991).

The Chi-Squared Test for the overidentifying restrictions is $T\vartheta(\xi,\theta)\widetilde{S}^{-1}\vartheta(\xi,\theta)' \to \chi^2\,(2)$. The test rejects the overidentifying restriction at conventional levels. That is not surprising given the very low discrepancy allowed for only two restrictions and 52 observations.

4. Quantitative Results

4.1. The Steady State

Mortality rates: the model demographic structure is not as rich as desirable because of computational constraints (*i.e.* there are only four generations, fertility timing is trivial, *etc.*) and this simplicity rules out a better fit. In particular, it seems that the main reason for the low mortality is that deaths are concentrated only at the end of each period. That makes the population structure move in jumps instead of a smooth curve. As a consequence the fertility rate does not need to be so high to generate the same birth rate (while english fertility rate is 2.23 for the studied period, the model rate is 1.95) and this reduces the total number of deaths per period.

To compute life-expectancy a midpoint of the generation age is taken. This procedure creates a small upward bias in the computation.

Is the lack of a better fit in population structure an important drawback of the model? As discuss in more detail in the section 6, it does not seem to be the case, at least from a qualitative perspective. The intuition for this result lies in the small response of fertility choices with respect to earlier deaths of children in their first period. On one hand children do not get any utility and consequently they do not enter into the value function of their parents until they reach adulthood. On the other hand, lower expected per child cost (due to shorter average lives) will tend to cancel off with a slightly higher number of children per household.

4.1.1. Changes in education,

Beyond secondary schooling, universities also showed an important increase in enrollment although still at very low total levels, with a percentage of male birth cohort attaining a degree that went form 0.9% before 1886 to 2.2% in 1907-1916 (Matthews, Feinstein and Odling-Smee (1982)). Finally, even if the evidence for technical training is much more sparse than for formal schooling, indirect measures seem to show an increase in the decades pervious to the first world war. This evidence indicate that, for the first time in English history, most of the components of a cohort was getting primary and secondary education just when fertility was falling and that other, minor areas of the educational system were also experiencing an increase in enrollment.

4.2. A change in mortality

O'Hara (1975) was the first to point out that the total effect on fertility of a fall in mortality is ambiguous. Another ambiguous effect is the age structure change. Since the share of children that reach adulthood is higher, Crude Birth Rate can go up as percentage of the total population even if fertility falls in each particular family.

Eckstein, Mira and Wolpin (1999) analyze the relationship between fertility, mortality and real wages in Sweden during the demographic transition. Their paper, however, does not try to account from feedbacks of the endogenous variables to wages or capital accumulation. Also, to simplify computation, their model does not link generations in an altruistic matter, so they need to specify a utility function that induces fertility regardless of descendants welfare. That feature basically kills the trade-off between quality and quantity and the consequent dynamic interactions and induces the effect of the reduction of infant mortality on total fertility.

Learning processes: Mira(1995) estimates a explicit learning about the new mortality conditions and does not find important lags. Blau (1992) shows how immigrants from high-fertility source countries were found to have very similar fertility to american-born women in the 70's and 80's: despite huge cultural and educational differences, fertility is adjusted within one generation.

5. What Matters

5.1. Changes in Functional Forms

The human capital investment function $h_{t+1} = \varphi(e_{t+1})$ only has as argument the parents effort. Two reasons justify this choice. First, it reduces notably the number of parameters while keeping a reasonable structure in the budget constraint. Second there is no strong evidence in favor of constant or increasing returns to scale in education. A more complicated function, with the human capital of the parents as an input has been tried without important changes.

Meltzer's case (and under very special assumptions) requires $\frac{\varepsilon'(n)}{\varepsilon(n)} < \frac{(1-\gamma n^{\varepsilon(n)})'}{1-\gamma n^{\varepsilon(n)}}$ but in addition $\frac{\varepsilon'(n)}{\varepsilon(n)} > -\frac{1}{n \log n}$ must hold for all $n < \overline{n}$ to avoid a bliss point (this second condition assures that the derivative of $n^{\varepsilon(n)}$ is positive) in the number of children. In my model the conditions do not even have an analytical expression. However this possibility needs a tight parametrization to work that does not seem very intuitive.

5.2. Changes in Experiment Design

Changes in the value of exogenous parameters were introduced in the experiments in only one period. The reason is basically computational: repeated, small changes length the transition and the time needed to find the equilibrium prices. Experiments have been performed when the change on the parameter takes several periods: the same steady states are reached with longer transitions. The response of the model to changes in the size of the change of the exogenous parameter have been also studied. The answer to the experiments goes in the same qualitative direction with only variations in the size of the response.

5.3. Which Moments to Match

To illustrate this point we can modified the model with new age-specific factors[13]. The new age distribution (39.8, 34.3, 18.8 and 7.1) is extremely close to the observed one (now the KLIC is 0.79 versus the benchmark model of 4.37) while the other results of the model stay basically constant. The intuition is simple: the reduction in the probability of getting to the last periods only changes substantially saving decisions but not fertility because of the relatively high discount factors. These results indicates that matching only two moments is a convenient choice.

[13] Survival probabilities are reduced between the second and the third period of life by a third and between the third and the last by two thirds.

6. Conclusions

In contrast other alternatives proposed in the literature do not seem to generate the desired results or, if they do, they need to rely on nonstandard assumptions. In comparison the model presented here does not depart form standard theory except in the form of the production function. The departure is backed by important empirical evidence and casual observation that strongly suggests the presence of different elasticities of substitution between input factors. The main conclusion of the paper is then simple: Neoclassical Theory can account for the demographic transition.

Goldin and Katz (1999) presents evidence on the skill premium during the twentieth century.

7. Appendix

7.1. Population Data

The raw data present some important problems, specially regarding their completeness (*i.e.* nonconformist groups tried to avoid the established church control of births and deaths) and some treatment must be applied.

The main techniques used in these works have been inverse projection and family reconstitution, both of which give a similar picture of facts. Indeed, the far more detailed estimations from the latter can be used to refined measures from the former. Inverse projection is similar in spirit to population forecast but in the opposite direction. If the size and basic characteristics of a population are known (age structure, births and deaths) and migration is properly accounted for, then the underlying levels of fertility and mortality can be calculated and information about changes in the size and structure of the population recovered. Wrigley and Schofield (1981) gathered data from 404 parishes registers and applied the technique of inverse projection to them. Family Reconstitution is also a simple method. The idea, developed by Gautier and Henry (1958), behind family reconstitution consists in the systematic assemblage and articulation of information about the life histories of families using the data on parishes registers. Wrigley *et al.* (1997) used data on 26 parishes representatives of England to elaborate the quinquennial demographic data used for the period 1541-1837.

7.2. Granger Causality Test

Reference : Granger (1969).

First, an optimal length of the Bivariate VAR is chosen using a log-likelihood test with a small sample correction is introduced following Sims (1980). If $\mathcal{L}_0^*$ is the loglikelihood evaluated at the null and $\mathcal{L}_1^*$ at the alternative, T the period length and p_1 the lag of the alternative, the value of the test is $2 * \frac{(T-2*p_1-1)}{T} * (\mathcal{L}_1^* - \mathcal{L}_0^*)$and distributed as a $\chi(4)$:

Optimal VAR Lag

Lags	Test value	P-value
$H_0 = 1, H_1 = 2$	5.40	0.25
$H_0 = 2, H_1 = 3$	5.60	0.23

These results of the log-likelihood test clearly support a small lag structure (1 or 2). Indeed, after this point, if the lags are increased until the maximum allowed given the length of the sample, clear signs of lack of degrees of freedom appear. Alternatively, the robustness of the test can be assessed using the Schwarz criterion as a first order asymptotic approximation of the Bayes factor. This test also prefers 1 lag over 2 (0.88 over 0.94). For the case with 3 lags, the lack of degrees of freedom turns out a non reliable result.

Then, a Wald test on the null hypothesis of lack of causality of infant mortality on fertility is performed on VARs with 1 and 2 lags. Let RS_0 be the squared sum of residuals under the null and RS_1 under the alternative. For T observations, the test is given by $T * \frac{RS_0 - RS_1}{RS_1}$ and distributed as a $\chi(2)$. The values of the test for lag length of 1 and 2 are reported below.

Wald Test of Granger Causality

Lags	Test value	P-value
1	0.25	0.87
2	0.66	0.72

Using the same test in opposite direction:

Wald Test of Granger Causality

Lags	Test value	P-value
1	0.47	0.49
2	9.73	0.01

7.3. Life Tables

Survival table and interpolation table are computed (see Figure A1):

P(*live at* 1)	0.8252
P(*live at* 21|1)	0.7999
P(*live at* 41|21)	0.7624
P(*live at* 61|41)	0.5763

Given this probabilities, the survival functions are equal to:

$$1 - e^{-m_0 c_{t+1}^t} = 0.8252 \quad (25)$$
$$1 - e^{-m_1 c_{t+1}^t} = 0.7999 \quad (26)$$
$$1 - e^{-m_2 c_{t+1}^t} = 0.7624 \quad (27)$$
$$1 - e^{-m_3 c_{t+1}^{t-1}} = 0.5763 \quad (28)$$

Now, using the first three equations, m_0 =1.0839m_1 and m_2 =0.8931m_1. Another argument for $c_{t+1}^t = c_{t+1}^{t-1}$ is that we can think that the really relevant factor is not as much consumption but consumption on food and health and even if total consumption fluctuates, this particular item fluctuates much less. In any case, the model is robust to changes in this parameter: it does not influence fertility decisions directly but only through indirect changes in capital accumulation (for retirement) and even here the influence is of second order. Applying this assumption, the computed value for m_3 is $0.5337 m_1$.

These tables are also used to compute the age-distribution of population when available data is not divided in the desired bins.

7.4. Computation

This length is selected with a Global to Particular Procedure (GPP): a very high number is selected and it is recursively reduced until a point with further reductions alter the behavior of the transition path.

Another additional checked was performed in the size of the grid, recomputing only the steady states for a finer grid. Results were basically unchanged.

Source Code was written in C++, compiled by GNU CC and run on Sun Workstation Ultra 2 with SunOS 5.6.

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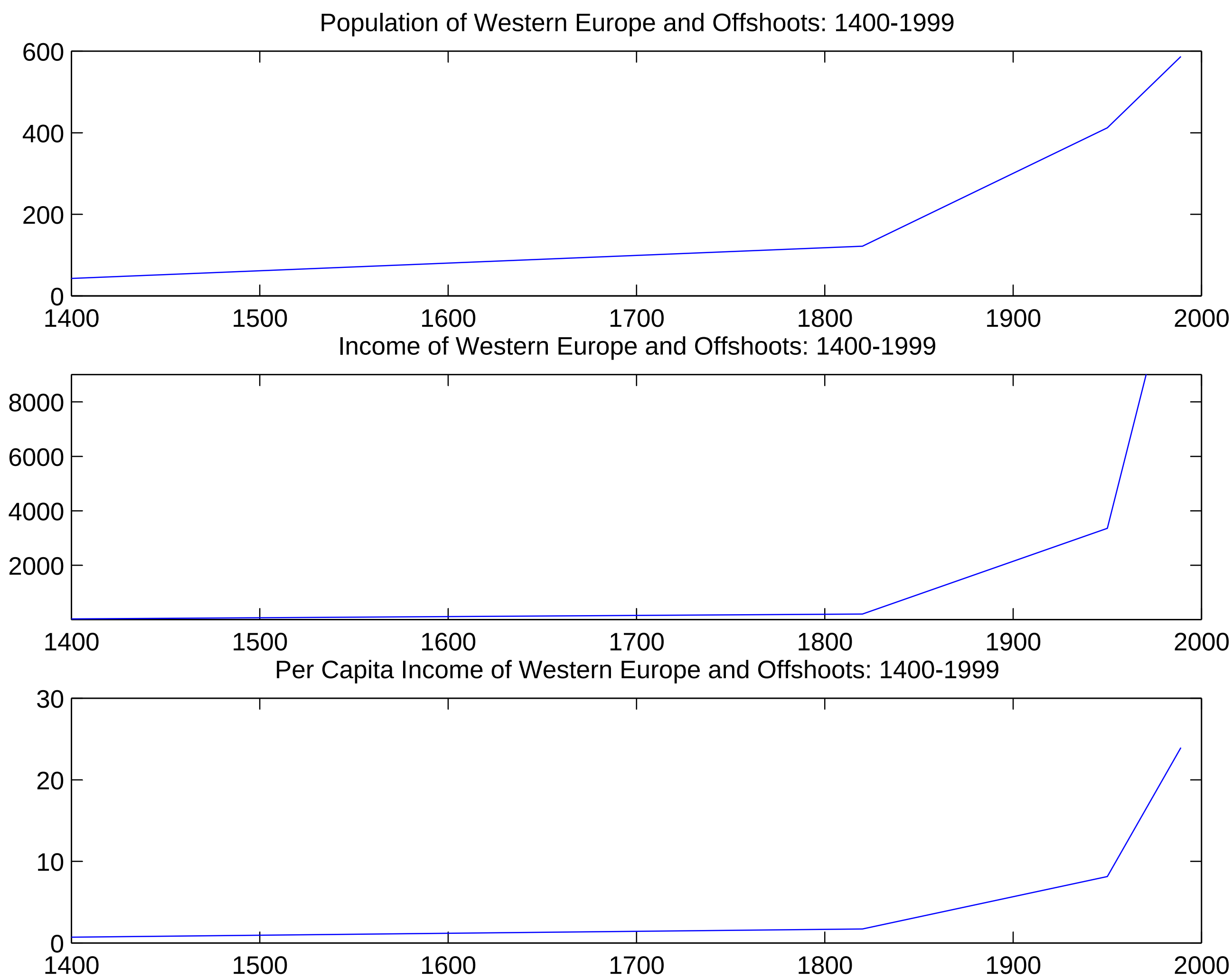
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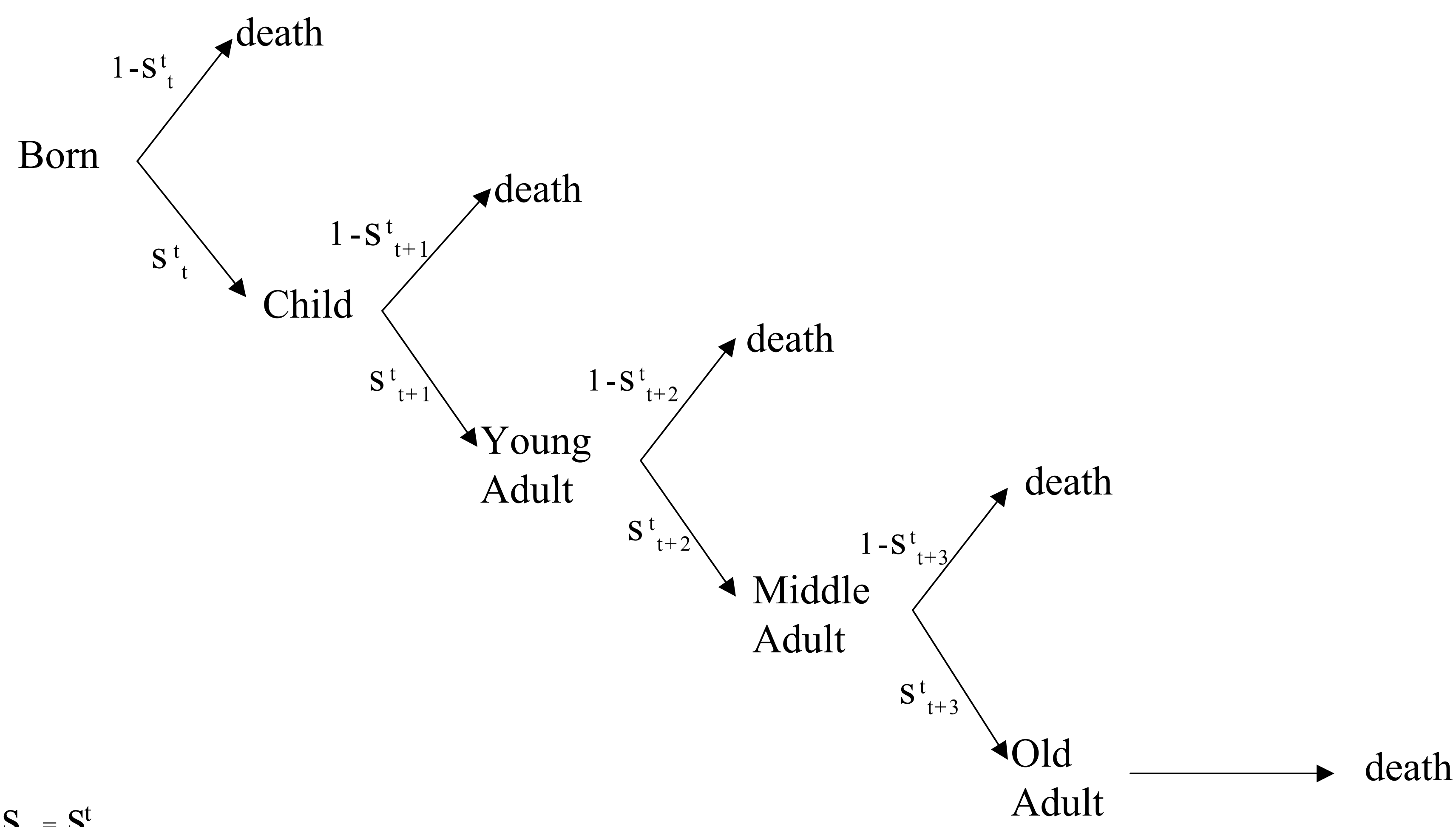
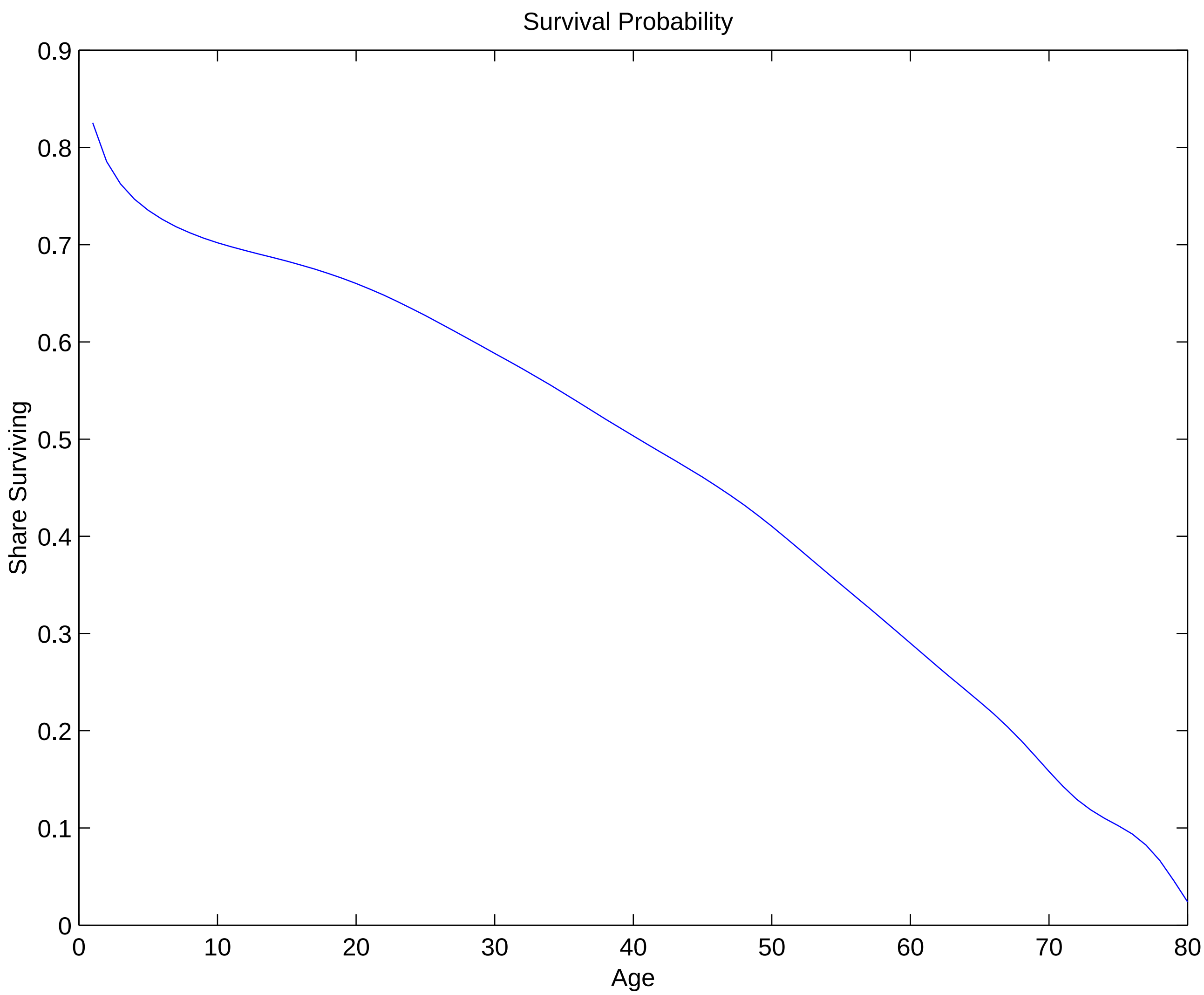


$S_{1t} = S^t_t$

$S_{2t} = S^t_t \; S^t_{t+1}$

$S_{3t} = S^t_t \; S^t_{t+1} \; S^t_{t+2}$

$S_{4t} = S^t_t \; S^t_{t+1} \; S^t_{t+2} \; S^t_{t+3}$



Survival Probability
Share Surviving
Age
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
10
20
30
40
50
60
70
80