INCOME VARIATION, ENDOGENOUS POPULATION GROWTH AND HEALTH SUBSIDY

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Abstract

This paper presents a fertility choice model in which the mortality rate is also endogenously determined and health expenditure provides utility to individuals as well as affects the mortality rate. The analysis shows that the model predictions agree with the empirical evidence on the relationship between demography trends and economic development. Public expenditure represents a large amount of total expenditure on health care in many countries. Thus, we also study the effects that introducing a subsidy to health expenditure has on economic and demographic variables. These effects are found to depend on the way the subsidy is financed.

Resumen

Este trabajo presenta un modelo de elección de la fertilidad en el que la tasa de mortalidad se determina endógenamente y el gasto sanitario proporciona utilidad al individuo además de influir sobre la tasa de mortalidad. El análisis muestra que las predicciones del modelo concuerdan con la evidencia empírica sobre la relación entre tendencias demográficas y desarrollo económico. El gasto público representa una gran proporción del gasto sanitario total en muchos países. Por ello, también se estudian los efectos de un subsidio al gasto sanitario sobre las variables económicas y demográficas, que dependerán de la fuente de financiación del subsidio.

JEL Classification*: J13, I12, O47, H51.*

Keywords*: Fertility; Mortality; Growth; Health Expenditure.*

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1. Introduction

The hypothesis of exogenous population growth implicit in the neoclassical growth model, which neglects interactions between the economic growth process and demographic trends, is clearly unsatisfactory. There is a large body of empirical evidence on the relationship between demography and development. Barro and Sala-i-Martin (1995, p. 432) find that life expectancy is an important growth factor since it is calculated that a 13 year increase in life expectancy is estimated to provoke an increase in the annual growth rate of the economy of 1.4 percentage points. Jablonski *et al*. (1988) reach similar empirical conclusions in terms of life expectancy on carrying out a direct study into the relationship between life expectancy, productivity and health. According to the World Bank (1993), falls in mortality and morbidity rates are important in promoting economic productivity and fomenting economic development. Thus, a genuine understanding of the economic growth process should take into account the existence of important linkages between development and demographic trends, so demographic and economic variables should be jointly determined by the model of the economy.

Following the seminal work of Becker (1960), several authors have studied the feedback between population growth and development. Important works of reference are that of Becker and Barro (1988) and Barro and Becker (1989) where fertility is endogenously determined. But the population growth is determined by the difference between contemporaneous rates of fertility and mortality, so it would interesting to introduce the mortality rate in the model as an endogenous variable so that the demographic behavior could be characterized completely.

Using data from the OECD Health Database 1998, the ratio of health expenditure to GDP in the USA increased from 5.2 percent in 1960 to 9.1 percent in 1980, and added up to 14 percent of GDP in 1997. Expenditure on health care accounted for a considerable fraction of GDP, and has been increasing over time, in many other developed and developing countries. The relationship between health expenditure and demographic outcomes has been well established, so a model that tries to explain the population growth should take expenditure on health care into account.

In this paper, we develop a growth model of fertility choice, in which mortality is also endogenously determined. The positive and strong relationship between life expectancy and health expenditure will be captured by the assumption that the mortality rate depends negatively on health expenditure. Newhouse (1977) points out that the medical care could affect not only to the mortality rate but also to the so-called subjective components of health. Grossman (1972) also suggests that the health levels could affect the levels of well-being. Accordingly, we assume that individuals derive utility from health expenditure, used as a proxy of health status.

The findings of the model accord with the empirical evidence on the relationship between population growth and economic growth (see, for example, Rostow (1990), Kirk (1996), Barro and Sala-i-Martín (1995) and Ehrlich and Lui (1997)). As the economy develops and per capita income increases toward its stationary value, the rates of fertility, mortality and population growth decrease. In addition, we find that per capita health expenditure increases along with per capita income as the economy evolves. The comparative steady state analysis shows that the higher individuals value their health status, the higher per capita health expenditure, the lower the mortality rate and the greater per capita capital and output in the steady state.

Public expenditure on health care represents a large fraction of total expenditure. In the USA, public expenditure on health care accounted for 24.8 percent of total health expenditure in 1960, 42.4 percent in 1980, and 46.7 percent in 1997. In many other countries, it represents even a higher fraction of total expenditure. For example, in 1997 public expenditure accounted for 84.5 percent of total health expenditure in the United Kingdom, 83.3 percent in Sweden and 77.4 percent in Japan. Given the above, it would be of interest to analyze the effects that introducing a subsidy to health expenditure has on economic and demographic variables. As one might expect, we find that these effects depend on the way the subsidy is financed. If a consumption tax is used, output increases, while the opposite occurs when a capital income tax is used. In welfare terms, whether financing the subsidy by a consumption tax or a capital income tax depends on the extent of the subsidy.

This paper is organized into five sections. Section 2 presents the model. Section 3 studies the transitional dynamics of the model and make a comparative steady state analysis after parameter variations. Section 4 analyses the effect of introducing a subsidy to expenditure on health care. Our concluding remarks are given in Section 5.

2. Setup of the Model

This paper considers an extension to the fertility choice model of Becker and Barro (1988) and its continuous time version set out in Barro and Sala-i-Martin (1995) and Blackburn and Cipriani (1998). Here, mortality depends on health expenditure and fertility is endogenously determined in an infinite horizon model. This assumption implies that each generation of a family is linked altruistically. In this context, adults within each household take into account the welfare and resources of their actual or future descendants.

The Grossman model (1972) is an important reference point within the field of health economics. He assumes that individuals attempt to augment their health levels examining the consequent effects on levels of well-being and income. Better levels of health yield higher levels of welfare. Accordingly, we assume that households derive utility from the sequences of effective consumption $z = c + jg$, that is, a linear combination of per capita consumption c and per capita health expenditure g, where $0 < \varphi < 1$ is a parameter that indicates the extent to which each individual values his or her welfare in terms of health, as compared to consumption. Time is continuous, all markets are perfectly competitive, and the economy has a large number of identical households that seek to maximize utility. Their preferences are described by the intertemporal utility function:

$$U = \int_0^\infty e^{-\rho t} \left\{ \psi \ln N + \ln(c + \varphi g) + \phi \ln(n - d(\hat{g})) \right\} dt, \tag{1a}$$

where ρ is the rate of time preference and represents parental altruism. Here, N is the size of the typical dynasty, n is the family's fertility rate, and $d(\hat{g})$ is the family's mortality rate, where x is the (exogenous) growth rate of technological progress, and $\hat{g} = ge^{-xt}$ is per capita health expenditure in terms of effective labor.

The mortality rate at time t is assumed to be only affected by instantaneous health expenditure per efficiency unit of labor. Although "health" has characteristics like capital that could be accumulated (e.g., investing in health has a positive effect on mortality throughout the lifetime of a person), the modeling of this effect would be probably intractable. Blackburn and Cipriani (1998) argue that greater spending in health is a means of mitigating the potential adverse effects on child welfare of greater economic activity (such as noise, congestion, pollution, …). The hypothesis that the mortality rate depends on health expenditure per efficiency unit of labour seeks to capture this idea: per capita health expenditure should grow more than economic activity as proxied by the technological progress, in order to yield a lower mortality rate. As it will be shown later in the paper, the growth rate of per capita output in the steady state is precisely the growth rate of the technological progress.

We assume that $d(\hat{g})$ verifies that $d(\hat{g}) \geq 0$, $d'(\hat{g}) < 0$ and $d''(\hat{g}) > 0$, $\forall\, \hat{g} \geq 0$. In other words the mortality rate decreases as $\hat{g}$ increases, but the higher $\hat{g}$ is the lower the decrease of the mortality rate as a consequence of increasing $\hat{g}$. This last assumption is justified by certain studies (as Newhouse, 1977) that argue that in countries with high expenditure, the marginal utility of medical care is more likely to produce improvements in so-called subjective components of health rather than improvements in morbidity and mortality rates. Parkin *et al*. (1987) argue instead that it is equally plausible to assume that the "marginal utility of medical care" does produce an improvement in objective health status, but that the cost of this marginal utility is greater for higher-income/higher-expenditure countries. Some other desirable features of the mortality rate function might be that $d(0) > 0$, so that in the absence of health expenditure the mortality rate would be the "natural" mortality rate. We also assume that health gains are effectively bounded, so that $\lim_{\hat{g} \to \infty} d(\hat{g}) = \bar{d}$, with $0 < \bar{d} < d(0)$. The size of the family changes continuously according to

$$\dot{N} = (n - d(\hat{g}))N \tag{1b}$$

Households own the stock of physical capital in the economy and each adult supplies inelastically one unit of labor per unit of time. We also introduce the cost of child rearing Φ, as different authors have already suggested, which would tend to increase as parental income increases or with other measures of the opportunity costs of parental time. We use a simplified linear function

$$\Phi = bk$$

where bk represents the opportunity costs that increase as parental capital increases. Thus, the family's budget constraint in per capita terms can be expressed as

(1c) $$\dot{k} = w + ((1-\tau_k)r - n + d(\hat{g}))k - bnk - (1+\tau_c)c - (1-s_g)g - R,$$

where w is the wage rate, r is the interest rate, τ_c is the consumption tax rate, τ_k is the tax on capital income rate, s_g is the subsidy to health expenditure rate and R is a lump sum tax (or transfer). Assuming that the government cannot issue debt to finance its deficit, its budget constraint can be expressed as

(1d) $$\tau_k rk + \tau_c c + R = s_g g.$$

We do not introduce a tax on labor income since it acts much like a lump-sum tax because of the assumptions implicit within the model, which does not include human capital or the labor-leisure choice, for example, to keep it tractable.

The household optimization problem lies in maximizing (1a), subject to (1b) and (1c). Solving the problem (see Appendix), we eventually arrive at the following expressions:

(2a) $$\frac{\dot{z}}{z} = (1-\tau_k)r - \rho - (1+b)n + d(\hat{g}),$$

(2b) $$n = d(\hat{g}) + \frac{\phi\rho(1+\tau_c)\hat{z}}{\rho(1+b)\hat{k} - \psi(1+\tau_c)\hat{z}} = d(\hat{g}) + \frac{\phi\rho(1+\tau_c)(\hat{c}+\varphi\hat{g})}{\rho(1+b)\hat{k} - \psi(1+\tau_c)(\hat{c}+\varphi\hat{g})},$$

(2c) $$\hat{k} = \frac{\varphi(1+\tau_c) - (1-s_g)}{bd'(\hat{g})}$$

where $\hat{k} = ke^{-xt}$ represents per capita capital in terms of effective labor. Equation (2a) links the growth rate of per capita effective consumption with the rate of return on capital. Equation (2b) indicates that, *ceteris paribus*, a higher fertility rate is associated with a higher mortality rate (and, therefore, a lower health expenditure), a higher ϕ (which raises the marginal utility of the children), a higher ψ (which raises the marginal utility of the family size), a lower rate of time preference, ρ, a lower b and a higher consumption tax rate, τ_c. Furthermore, there exists a positive relationship between n and $(c+\varphi g)/k$. Equation (2c) shows that, *ceteris paribus*, a higher per capita capital in terms of effective labor is associated with a lower φ (which decreases the weight of the health expenditure in the effective consumption, and therefore decreases the marginal utility of health expenditure), a lower b, a lower τ_c, and a lower subsidy to health expenditure, s_g. In addition, there exists a positive one-on-one relationship between per capita capital and per capita health expenditure in terms of effective labor.

Firms take technological progress that grows at the rate x, as given. Hence, the Cobb-Douglas production function can be expressed as $\hat{y} = A\hat{k}^{\alpha}$, $0<\alpha<1$, where $\hat{y} = ye^{-xt}$ represent per capita income in terms of effective labor. Capital depreciates at the constant rate δ. Profit maximization implies that firms pay the marginal product of factors

$$r = aA\hat{k}^{a-1} - d, \tag{3a}$$

$$w = (1 - a)A\hat{k}^{a} e^{xt}. \tag{3b}$$

Substituting (3) into (1c), using the budget constraint of the government (1d), and given that $\dot{\hat{k}} = \dot{k}e^{-xt} - x\hat{k}$, equation (1c) leads to

$$\dot{\hat{k}} = A\hat{k}^{\alpha} - (\delta + (1+b)n - d(\hat{g}) + x)\hat{k} - \hat{c} - \hat{g}. \tag{4}$$

Substituting (2b) into (2a), and given that $\dot{\hat{z}} = \dot{z}e^{-xt} - x\hat{z}$, the motion of effective consumption is given by the following expression:

$$\frac{\dot{\hat{z}}}{\hat{z}} = (1 - t_k)(aA\hat{k}^{a-1} - d) - r - (1+b)n + d(\hat{g}) - x. \tag{5}$$

Differentiating the definition of effective consumption, $\hat{z} = \hat{c} + j\hat{g}$, with respect to time we obtain that

$$\dot{\hat{z}} = \dot{\hat{c}} + j\dot{\hat{g}},$$

so that (5) can also be expressed as

$$\dot{\hat{c}} + \varphi\dot{\hat{g}} = \left((1-\tau_k)(\alpha A\hat{k}^{\alpha-1} - \delta) - \rho - (1+b)n + d(\hat{g}) - x\right)(\hat{c} + \varphi\hat{g}). \tag{6}$$

Differentiating (2c) with respect to time gives that

$$\dot{\hat{g}} = -\frac{bd'(\hat{g})^2}{(\varphi(1+\tau_c) - (1-s_g))d''(\hat{g})}\dot{\hat{k}}. \tag{7a}$$

Substituting (7a) into (6), we obtain the following expression

$$\dot{\hat{c}} = \left((1-\tau_k)(\alpha A\hat{k}^{\alpha-1} - \delta) - \rho - (1+b)n + d(\hat{g}) - x\right)(\hat{c} + \varphi\hat{g}) + \frac{\varphi bd'(\hat{g})^2}{(\varphi(1+\tau_c) - (1-s_g))d''(\hat{g})}\dot{\hat{k}}. \tag{7b}$$

Substituting (4) into (7), and using (2b) and (2c) to express n and $\hat{k}$, respectively, as functions of $\hat{g}$ and $\hat{c}$, the dynamical system (7) that drives the economy can be expressed in terms of $\hat{g}$ and $\hat{c}$. The stationary values of $\hat{g}$ and $\hat{c}$ can be obtained by solving the system $\dot{\hat{c}} = 0$ and $\dot{\hat{g}} = 0$. Then, the steady state values of n and $\hat{k}$ could be calculated by means of (2b) and (2c), respectively.

3. Comparative Steady State Analysis

Now let us make some assumptions about the function $d(\hat{g})$, and the parameters of the model. Firstly, we assume that the mortality rate is related to health expenditure through a negative exponential function

$$d(\hat{g}) = L + M \ \exp\left\{T\hat{g}^{S}\right\}. \tag{8}$$

In order for *d* to be a decreasing function, then $T < 0$. This function verifies all the desirable features for the mortality rate implicit within the assumptions of the model. The *natural mortality rate* (in the absence of health expenditure) is *L*+*M*, and the threshold value of the mortality rate is *L*. The mortality rate cannot fall below this threshold value. The parameter *S* reflects the rate at which the mortality rate decreases. We consider the baseline displayed in Table 1, taking into account the common parameters used in Barro and Sala-i-Martin (1995), and setting all tax and subsidy rates at zero. With these parameter values, the fertility and mortality rates in the steady state are 1.5 percent and 0.8 percent respectively, similar to the observed USA rates.

TABLE 1
PARAMETER BASELINE VALUES

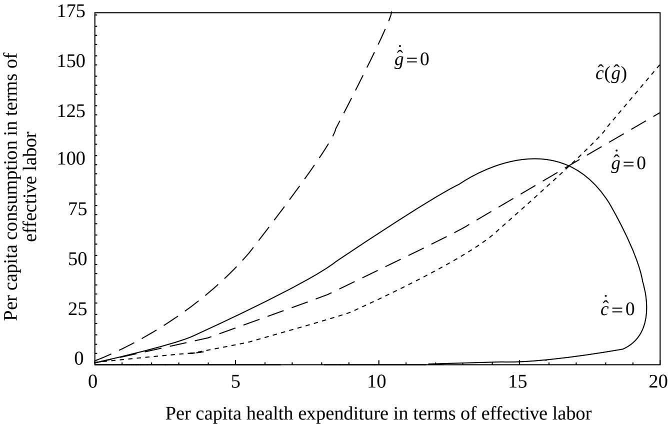
L	M	T	S	α	A	b	ρ	ψ	ϕ	φ	δ	x
0.005	0.195	–1	0.5	0.75	1	1	0.02	0.2	0.2	0.2	0.05	0.02

The phase diagram is represented in Figure 1. Equating (7a) to zero and solving for the variable $\hat{c}$ as a function of $\hat{g}$ we find the two loci of points for which $\dot{\hat{g}} = 0$, depicted in Figure 1 with dashed lines. Equating (7b) to zero and solving for the variable $\hat{c}$ as a function of $\hat{g}$ we find the locus of points for which $\dot{\hat{c}} = 0$, represented in Figure 1 with a solid line. The steady state values of $\hat{c}$ and $\hat{g}$ lie at the point where both loci intersect, $\dot{\hat{c}} = \dot{\hat{g}} = 0$. The system (7) displays saddlepath stability for every parametrization considered in this paper, since only one eigenvalue of the jacobian matrix of (7) is negative at the steady state. The policy function $\hat{c}(\hat{g})$, depicted in Figure 1 with a dotted line, is obtained by using the time elimination method of Mulligan and Sala-i-Martin (1993).

As we have already noted, Equation (2c) implies that there exists a direct and positive relationship between per capita capital and per capita health expenditure in terms of effective labor. Thus, both variables evolve in the same direction, and so does output per capita in terms of effective labor, $\hat{y}$, since $\hat{y} = A\hat{k}^{\alpha}$.

By equation (2c), when the economy starts from an initial per capita capital in terms of effective labor below its steady state value, per capita health ex-

FIGURE 1
PHASE DIAGRAM IN ($\hat{g}$, $\hat{c}$) SPACE IN THE BASELINE



penditure in terms of effective labor is also below its steady state value. As the transition path in Figure 1 is positively sloped, per capita health expenditure rises along the transition path towards the steady state. Hence, by (2c), per capita capital, $\hat{k}$, and thus per capita output, $\hat{y}$, in terms of effective labor, also rise as the economy develops. This fact agrees with the observable empirical evidence. For example, using data from the OECD Health Database 1998, the ratio of health expenditure to GDP in USA increased from 5.2 percent in 1960 to 9.1 percent in 1980 and to 14.0 percent in 1997. This tendency can be observed in practically all OECD countries.

Figure 2 displays the transitional behavior of the mortality rate and the fertility rate, this last being obtained by substituting the policy function $\hat{c}(\hat{g})$ into Equation (2b). The transitional behavior of the population growth rate and the stock of capital per capita in terms of effective labor are also illustrated. The fertility rate behaves monotonically. It decreases towards its stationary value as the economy evolves. The way the fertility and the mortality rates behave is in accordance with the results obtained by Rostow (1990) and Kirk (1996). The fertility and mortality rates are correlated both significantly and negatively with per capita income. As the economy develops both the fertility and mortality rates fall, and these falls are accompanied by an increase in per capita income. A low mortality rate and high per capita capital imply a low fertility rate. Reduced mortality levels and a healthier population are major contributors to a rise in living standards, which is often regarded as a major factor in fertility decline.

Considering the effect of parameter variations we obtain Table 2, which shows that changes in ψ and ϕ lead to changes in n and d in the same direction. When ψ rises from its baseline value to 0.3, the mortality rate increases from 0.0082 to 0.0085 and the fertility rate thus changes from 0.015 to 0.0168. The variation in the fertility rate is also greater than the variation in the mortality rate, which reflects the effect of the increase in c/k from 0.0489 to 0.0494.

Thus, the net rate of population growth rises from 0.67% to 0.83%. We observe a similar pattern when ϕ reaches 0.3, although this effect is stronger than that provoked by ψ on n and d. The fertility rate n rises to 1.96% and d rises to 0.909% which gives a net population growth rate of 1.059%.

FIGURE 2

TRANSITIONAL BEHAVIOR OF FERTILITY, MORTALITY AND POPULATION GROWTH RATES, AND PER CAPITA CAPITAL IN TERMS OF EFFECTIVE LABOR IN THE BASELINE

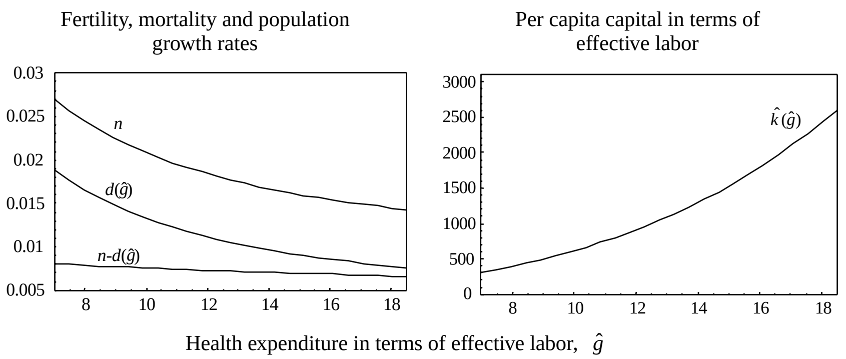


TABLE 2

EFFECTS OF PARAMETER VARIATIONS

	$\hat{c}^*$	$\hat{g}^*$	$\hat{k}^*$	d^* (%)	n^* (%)	$n^* - d^*$ (%)	r^* (%)
Baseline	99.1965	16.7957	2025.52	0.8237	1.5017	0.6780	6.180
$\psi = 0.3$	88.8189	16.0099	1794.72	0.8567	1.6898	0.8331	6.523
$\psi = 0.1$	106.969	17.3459	2200.16	0.8029	1.3769	0.5740	5.951
$\phi = 0.3$	74.4297	14.9349	1512	0.9090	1.9682	1.0593	7.027
$\phi = 0.1$	130.591	18.7435	2696.08	0.7569	1.0826	0.3257	5.408
$\varphi = 0.5$	103.567	20.1927	2061.22	0.7180	1.4245	0.7065	6.131
$\varphi = 0.1$	97.7969	15.9903	2012.89	0.8576	1.5274	0.6698	6.197
$b = 2$	82.1024	20.6415	1752.07	0.7074	1.1000	0.3925	6.592
$b = 0.5$	105.183	12.7028	2065.07	1.0523	2.1187	1.0664	6.126
$T = -0.5$	66.5154	33.2554	1691.53	1.5909	2.1429	0.5519	6.695
$T = -1.5$	108.276	10.1554	2076.48	0.6637	1.3871	0.7234	6.110
$L = 0.008$	88.388	15.9759	1785.19	1.1582	1.8482	0.6900	6.538
$L = 0.003$	107.294	17.3683	2207.49	0.6021	1.2719	0.6698	5.942
$M = 0.25$	97.8154	18.5042	2032.35	0.8387	1.5044	0.6658	6.170
$M = 0.15$	100.591	15.1009	2019.25	0.8079	1.4981	0.6902	6.188

As expected, a variation in the child rearing cost b yields a change in the fertility rate in the opposite direction. For example, as b rises from 0.5 to 2 the fertility rate declines from 2.11% to 1.1%. The mortality rate decreases from 1.05% to 0.7% due to an increase in health expenditure. This is comprehensible, since the "value" of child health rises as the opportunity costs increase. As b has a stronger effect on the fertility rate than its effect on the mortality rate the net growth of population decreases from 1.06% to 0.39%. Higher child rearing costs also detract resources to capital accumulation, so the stock of capital and therefore output decrease as b rises.

An increase in the parameter φ leads to a parallel increase in health expenditure. The more individuals value their health status, the more willing they are to spend on health and hence the per capita capital in the steady state is greater. For example, when φ rises from 0.1 to 0.5 the steady state value of health expenditure changes from 15.99 to 20.19 and the per capita capital varies from 2012.8 to 2061.22. As a consequence of the increase in health expenditure the mortality rate falls from 0.85% to 0.71% and the net growth of population increases from 0.67% to 0.70%. Consequently, the higher the value that individuals give to their health status, the higher per capita health expenditure, the lower the mortality rate and the greater per capita capital and output in the steady state.

In varying those parameters that enter into the functional form of d, we observe that changes in L lead to quantitatively similar changes in d and n. Changing the parameter T from –0.5 to –1.5 it yields a decrease in the mortality rate from 1.59% to 0.66% and a fall in the fertility rate from 2.14% to 1.38%. The greater the absolute value of T the more noticeable the effect of health expenditure on the mortality rate.

Table 2 also shows that variations in the parameters of the utility function or the child rearing costs (ψ, ϕ, φ, b) that result in a higher expenditure on health care, also lead to lower mortality, fertility and population growth rates. This result accords with the assertion that reduced mortality and a healthier population are major contributors to higher living standards, which are often regarded as a major factor in fertility decline. However, while variations in the parameters ψ, ϕ, φ that lead to a higher health expenditure also lead to a higher income, variations in b that lead to a higher health expenditure are associated instead with a lower income.

4. The Effects of a Subsidy Policy

We have already noted that public expenditure accounts for a large fraction of total expenditure on health care. Thus, in this section we analyze the effects that introducing a subsidy to health expenditure has on economic and demographic variables. As we can expect that the way the subsidy is financed is important, we examine the effects of a health subsidy financed first by a tax on capital income and then by a consumption tax. A tax on labor income is not utilized since it would constitute a lump-sum tax. Hence only taxes on capital income and consumption are used to finance the health subsidy. We use the values displayed in Table 1 for the structural parameters, and we consider alternative values for the fiscal policy parameters τ_k, τ_c and s_g.

In the following table we compare the baseline situation in which there is no government intervention with the situation in which there is a health expenditure subsidy, which is financed by either a consumption tax or a tax on capital income. Once the subsidy rate has been fixed we determine the tax rate that maintains a balanced budget without using a lump sum tax in the steady state; that is, $s_g g = \tau_c c$ or $s_g g = \tau_k rk$ at the new steady state. We also present a measurement for the welfare loss denoted by ε, following Lucas (1990). Let c_t^{old}, g_t^{old} and n_t^{old} which are associated with the steady state of the baseline economy, denote the paths of *c*, *g* and *n*, respectively,. Let c_t^{new}, g_t^{new} and n_t^{new} denote the paths that emerge after a change in the policy parameters of the government. The welfare loss associated with this policy change is a value ε such that $U(c_t^{old}(1-\varepsilon), g_t^{old}, n_t^{old}) = U(c_t^{new}, g_t^{new}, n_t^{new})$. Since c_t^{old} grows at a constant rate, ε is set so that consumers would express an equal preference for two distinct situations (i) there is a variation in the government policy parameters and (ii) there is a situation in which there is no government action but their consumption level is reduced by $100\times\varepsilon$ percent in every period. In our simulation we use the time elimination method (Mulligan and Sala-i-Martin (1993)). Table 3 summarizes the results obtained.

TABLE 3

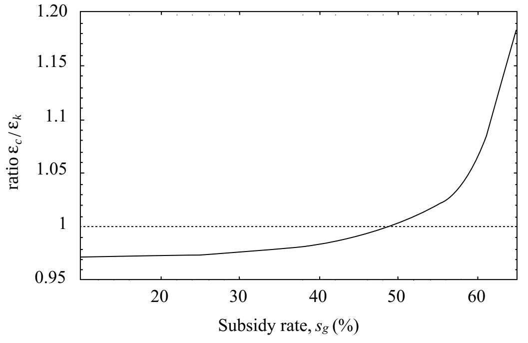
SIMULATION RESULTS

s_g (%)	Taxes (%)	$\hat{c}$*	$\hat{g}$*	$\hat{k}$*	d* (%)	n* (%)	$n^* - d^*$ (%)	ε (%)
0	Baseline (τ_k=τ_c=0)	99.19	16.79	2025.52	0.823	1.501	0.677	0
10	τ_k=1.421	97.23	17.47	1962.57	0.798	1.488	0.690	0.056
	τ_c=1.798	98.83	17.76	2038.28	0.788	1.475	0.687	0.054
20	τ_k=3.033	94.86	18.27	1890.39	0.771	1.476	0.704	0.256
	τ_c=3.854	98.26	18.93	2050.68	0.751	1.448	0.696	0.249
40	τ_k=7.19	88.03	20.42	1702.17	0.712	1.454	0.741	1.422
	τ_c=9.285	96.01	22.28	2073.87	0.673	1.393	0.72	1.399
60	τ_k=14.40	74.40	24.17	1377.49	0.642	1.450	0.807	5.404
	τ_c=19.95	89.46	29.74	2092.69	0.583	1.336	0.752	5.758

In quantitative terms the financing of a health subsidy through a consumption tax increases the output in the steady state with respect to the baseline situation. The opposite happens when we introduce a tax on capital income on the productive sector. The output decreases with respect to the baseline situation. The welfare analysis yields a loss with respect to the baseline situation in both cases. The ratio of loss of welfare on the consumption taxation setting, ε_c, to the loss of welfare in the capital income taxation setting, ε_k, is shown in Figure 3. When the subsidy to health expenditure is below about 49%, the welfare loss is smaller if a consumption tax rather than a tax on capital income is used to finance the subsidy. Henceforth, using a tax on capital income results in a smaller welfare loss.

FIGURE 3

RATIO OF WELFARE LOSSES AFTER A CHANGE IN THE SUBSIDY RATE

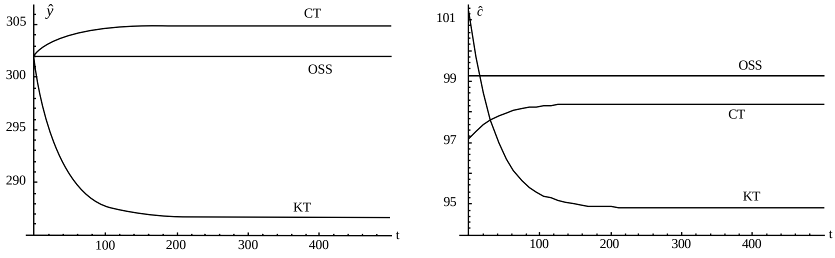


We can observe in Table 3 that the larger the health subsidy, the greater the loss in welfare. Furthermore, both the mortality rate and the fertility rate fall monotonically when the health subsidy increases, independently of whether the subsidy is financed by a consumption tax or by a tax on capital income. The population growth rate increases in both cases. Further, the increase is greater when the government finances the health subsidy by means of a tax on capital income rather than by via a consumption tax.

Figure 4 shows the trends of per capita income, $\hat{y}$, and per capita consumption, $\hat{c}$, in terms of effective labor, using either a consumption tax or a tax on capital income to finance the subsidy. As the behavior of the variables remains consistent for distinct health subsidy values, we consider the following case where s_g=20%. As we can observe, when a tax on capital income is used, per capita income in terms of effective labor falls monotonically towards its new steady state value. Hence, along the transition path, the growth rate of per capita income, y, is below its steady state value of x. The opposite happens when a consumption tax is utilized. Per capita income in terms of effective labor increases monotonically towards its new steady state value, so its growth rate is positive and decreasing towards zero. Hence, the new steady state value of $\hat{y}$ is greater than the one obtained on the baseline.

It is worth noting how little the tax reform affects the growth rate of $\hat{y}$ along the transition path. In the capital income tax setting, it ranges from –0.13 percentage points at the outset to –0.11 after 5 years and –0.09 after 15 years. In the case of a consumption tax, it ranges from 0.023 percentage points at the outset to 0.021 after 5 years and 0.016 after 15 years. This is in accordance with

FIGURE 4
EVOLUTION OF PER CAPITA INCOME AND PER CAPITA CONSUMPTION
IN TERMS OF EFFECTIVE LABOR WHEN s_g=20%
(CT = evolution after imposing a consumption tax; KT = evolution after
imposing a tax on capital income; OSS = old steady state)



the evidence presented in Stokey and Rebelo (1995), who found that the rise in income tax rates produced no noticeable effect on the average growth rate of the economy. This effect though slight can also be observed in the slow convergence towards the new steady state. In the consumption tax setting 50% of the difference between the old and the new steady state income is reduced in 26.5 years, and 75% of the difference in 53 years. Similar results are obtained in the capital income tax setting.

FIGURE 5
EVOLUTION OF POPULATION GROWTH, MORTALITY
AND FERTILITY RATES WHEN s_g = 20%
(CT = evolution after imposing a consumption tax; KT = evolution after
imposing a tax on capital income)

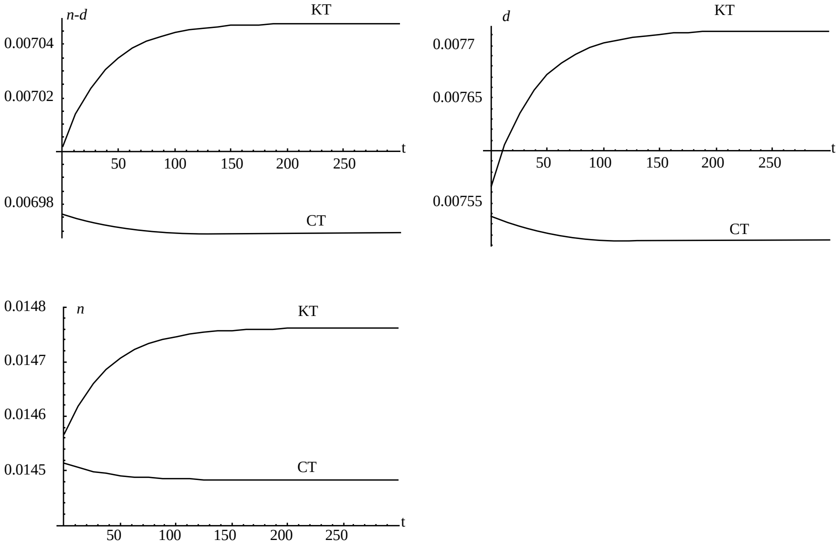


Figure 5 shows that the population growth, mortality and fertility rates exhibit contrary behaviors whether the health subsidy is financed by imposing a tax on capital income or by imposing a consumption tax. In the capital income tax setting, fertility, mortality and population growth rates increase monotonically towards their new steady state values, while they decrease steadily when a consumption tax is utilized. However, all along the transition, both the mortality and the fertility rates lie below their old stationary values shown in the baseline row of Table 3, whereas the population growth rate always lies above its old steady state value, irrespective of which tax is used to finance the subsidy.

5. Conclusions

In this paper, we present a fertility choice model, in which mortality is also endogenously determined. Health expenditure, used as a proxy of health status, is assumed to provide utility to individuals as well as affect the mortality rate. The solution of the model allows to jointly determine economic and demographic variables. The analysis of the transitional dynamics and the steady state of the model provide findings that accord with the empirical evidence on the relationship between development and demography. As the economy develops, per capita income and per capital health expenditure jointly increase toward their stationary values, whereas the rates of fertility, mortality and population growth decrease.

The analysis of a government policy through the introduction of a health subsidy considers the use of a tax on capital income and a consumption tax alternatively, in order to finance the subsidy. As one can expect, the effects of the subsidy on economic and demographic variables depend on the way the subsidy is financed. When a consumption tax is used, the output increases in the steady state. The opposite happens when we impose a tax on capital income. In both cases there is a loss of welfare when compared to the baseline situation. In welfare terms, however, whether financing the subsidy by a consumption tax or a capital income tax depends on the extent of the subsidy.

The representative agent framework used in this paper and the absence of externalities imply that arguments of equity or efficiency cannot be invoked to justify a subsidy to health expenditure. As we have already noted, public expenditure on health care represents a large fraction of total expenditure in many countries. Improving the model so that it could give an explanation for using such a subsidy would be a natural extension of this work.

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APPENDIX

Let υ and μ be multipliers for the constraints (1c) and (1b) in the household optimization problem. The Hamiltonian of the problem is

$$H = e^{-\rho t}(\psi \ln N + \ln(c + \varphi g) + \phi \ln(n - d(ge^{-xt}))) +$$
$$+\upsilon(w + ((1-\tau_k)r - n + d(\hat{g}))k - bnk - (1+\tau_c)c - (1-s_g)g - R) + \mu(n - d(\hat{g}))N.$$

The conditions for a maximum are

(A1) $$\frac{\partial H}{\partial c} = \frac{e^{-\rho t}}{z} - (1+\tau_c)\upsilon = 0,$$

(A2) $$\frac{\partial H}{\partial n} = e^{-\rho t}\frac{\phi}{n - d(\hat{g})} - \upsilon(1+b)k + \mu N = 0,$$

(A3) $$\frac{\partial H}{\partial g} = e^{-\rho t}\left\{\frac{\varphi}{z} + \frac{\phi(-d'(\hat{g}))e^{-xt}}{n - d(\hat{g})}\right\} + \upsilon(d'(\hat{g})e^{-xt}k - (1-s_g)) + \mu(-d'(\hat{g})e^{-xt})N = 0,$$

(A4) $$\dot{\upsilon} = -\frac{\partial H}{\partial k} = -\upsilon((1-\tau_k)r - (1+b)n + d(\hat{g})),$$

and

(A5) $$\dot{\mu} = -\frac{\partial H}{\partial N} = -e^{-\rho t}\frac{\psi}{N} - \mu(n - d(\hat{g})).$$

Equation (A1) can be expressed as

$$\frac{e^{-\rho t}}{z} = (1+\tau_c)\upsilon.$$

Taking logarithms and differentiating with respect to time, then

(A6) $$-\rho - \frac{\dot{z}}{z} = \frac{\dot{\upsilon}}{\upsilon}.$$

Using (A4) and (A6), we obtain (2a). Substituting υ from (A1) into (A2), we arrive at

(A7) $$\mu = -\frac{1}{N}e^{-\rho t}\left[\frac{\phi}{n - d(\hat{g})} - \frac{(1+b)k}{z(1+\tau_c)}\right] = -\frac{1}{N}e^{-\rho t}\Omega,$$

where $\Omega = \dfrac{\phi}{n-d(\hat{g})} - \dfrac{(1+b)k}{z(1+\tau_c)}$. Differentiating (A7) with respect to time gives

$$\text{(A8)} \qquad \dot{\mu} = -\frac{e^{-\rho t}}{N}\left\{-\frac{\dot{N}\Omega}{N} - \rho\Omega + \dot{\Omega}\right\}.$$

Substituting (A7) into (A5), we obtain that

$$\text{(A9)} \qquad \dot{\mu} = -e^{-\rho t}\frac{\psi}{N} + \frac{1}{N}e^{-\rho t}\Omega(n-d(\hat{g})).$$

Substituting (A9) into (A8), after simplification gives

$$\dot{\Omega} = \psi + \rho\Omega.$$

Its general solution

$$\text{(A10)} \qquad \Omega = -\psi/\rho + (\Omega(0) + \psi/\rho)\, e^{\rho t}$$

is unstable, because if $\Omega(0)$ departs from its steady state value $-\psi/\rho$, then Ω moves over time toward $\pm\infty$. Substituting the solution for Ω from (A10) into (A7), the transversality condition corresponding to the state variable N, $\lim_{t\to\infty} \mu N = 0$ gives

$$\lim_{t\to\infty} \mu N = -\lim_{t\to\infty} e^{-\rho t}\Omega = -\lim_{t\to\infty} (-e^{-\rho t}(\psi/\rho) + \Omega(0) + (\psi/\rho)) = 0\,.$$

This equation is verified if and only if $\Omega(0) = -\psi/\rho$. Hence, $\Omega = -\psi/\rho$, and by the definition of Ω, we obtain (2b).

Substituting into (A3) the expressions for υ in (A1) and for μ in (A7), and simplifying, we obtain (2c). Differentiating it with respect to time, we obtain (7a).