

http://economix.fr

The falling sperm counts story": A limit to growth?

Document de Travail Working Paper 2016-36 Johanna Etner Natacha Raffin Thomas Seegmuller



Université de Paris Ouest Nanterre La Défense (bâtiment G) 200, Avenue de la République 92001 NANTERRE CEDEX

Tél et Fax: 33.(0)1.40.97.59.07 Email: nasam.zaroualete@u-paris10.fr



"The falling sperm counts story": A limit to growth?*

Johanna Etner[†], Natacha Raffin[‡] and Thomas Seegmuller[§]

July 4, 2016

Abstract

We develop an overlapping generations model of growth, in which agents differ through their ability to procreate. Based on epidemiological evidence, we assume that pollution is a cause of this health heterogeneity, affecting sperm quality. Nevertheless, agents with impaired fertility may incur health treatments in order to increase their chances of parenthood. In this set-up, we analyse the dynamic behaviour of the economy and characterise the situation reached in the long run. Then, we determine the optimal solution that prevails when a social planner maximises a Millian utilitarian criterion and propose a set of available economic instruments to decentralise the optimal solution. We underscore that to correct for both the externalities of pollution and the induced-health inefficiency, it is necessary to tax physical capital while it requires to overall subsidy mostly harmed agents within the economy. Hence, we argue that fighting against the

^{*}We would like to thank participants to the Conferences Dyniper 2016, SURED 2016 and EAERE 2016 for their helpful comments.

[†]University Paris Ouest Nanterre la Défense, EconomiX and Climate Economics Chair. Building G, office 603b, 200 av. de la République, 92001 Nanterre cedex, France. Tel: +33 1 40 97 40 85. E-mail: jetner@u-paris10.fr

[‡]Corresponding author. University Paris Ouest Nanterre la Défense, EconomiX and Climate Economics Chair. Building G, office 604b, 200 av. de la République, 92001 Nanterre cedex, France. Tel: +33 1 40 97 77 81. E-mail: nraffin@u-paris10.fr

[§]Aix-Marseille University (Aix-Marseille School of Economics), CNRS-GREQAM and EHESS, Centre de la Vieille Charité, 2 rue de la Charité, 13236 Marseille CEDEX 02, France. E-mail: thomas.seegmuller@univ-amu.fr.

sources of an altered reproductive health is more relevant than directly inciting agents to incur health treatments.

JEL classification: O44; Q56; I18.

Keywords: Pollution; Growth; Fertility; Health.

1 Introduction

As a striking starting point of the paper, it has been observed that impaired fertility affects around 9% of procreating-aged couples worldwide (Boivin et al., 2007; Mascarenhas et al., 2012). Far from being negligible, this estimation comes along with an other interesting observation according to which it is indeed male infertility that is a major contributor to global childlessness - between a third to a half of all cases - (Inhorn and Patrizio, 2015). The downward trend in semen quality has simultaneously raised concerns that semen quality could be falling to thresholds levels which could impact fecundity at population level (Carlsen et al., 1992; Swan et al., 1997; Swan et al., 2000). Face to these evidence, identifying their origins and assessing their potential outcome is clearly warranted.

In addition, it appears that the reduced ability of fathering is a dynamic process that is concomitant with a period of rapid economic development. It seems that post-industrial societies have created the potential for increasing the exposure to specific lifestyle factors and behaviours that might negatively affect the reproductive health. And interestingly, increased environmental chemical pollution has often been implicated in poor semen quality, among others more health-related diseases or disorders (WHO, 2013). A bench of toxicological data and studies can be referred to on that subject and a large majority has concluded, so far, to an emerging evidence for adverse reproductive outcomes associated with the exposure, even at low levels of concentration, to more and more chemical pollutants that directly interfere with hormonal systems (Persistent Organic Pollutants (POPs), pesticides, metals, textiles, air particulates and the like). We therefore argue that male infertility and environmental quality are tightly related and might together have an impact on the long-run behaviour of one economy. Even if delaying the age of the first pregnancy has been established as a major driver of fertility rates decline in western economies (Myrskylä et al., 2009; ESHRE,

2010), we do not contradict these observations: We rather argue that the postponement of the first pregnancy and the induced difficulties to conceive could also be explained by impaired reproductive health functions so that the time to pregnancy mechanically becomes larger in a more polluted world. This phenomenon might be true all the more that substantial gains have been realised in longevity so that, coupled with sub-replacement fertility rate, many economies must face an inexorable ageing population.

Nevertheless, the growth process has also been accompanied by the development of new medical technologies and a better access to Assisted Reproductive Technologies (ARTs), going from basic hormonal treatments to most sophisticated methods of procreation broadly called In-Vitro Fecundation (IVF). From the first successful IVF performed in 1978 in the UK, the number of babies born thanks to ARTs amounts up to 3.5 millions of the overall births worldwide in 2008 (Connolly et al., 2010). All these various stylised facts are more deeply discussed in Section 2.

In this paper and based upon these epidemiological evidence, we aim at understanding the long-run consequences of such phenomenon, that is the reduced fertility of households due to pollution, in terms of growth and pattern of development, taking into account that households can invest in medical treatments. We further determine the social optimum and design the optimal policy.

We present an overlapping generations model where we assume that pollution induces a health heterogeneity to the extent that two types of household co-exist within one generation: Fertile and Infertile ones. Nevertheless, couples with impaired fertility may incur health treatments in order to increase their chances of parenthood, depending on the level of wealth that prevails in the economy. In this set-up, we analyse the dynamic behaviour of the economy and characterise the situation reached in the long run.

One key feature of the model is that pollution, by reducing the size of next generations, entails a perverse effect, that is an increase in future physical capital stocks *per capita*. This vicious effect is large all the more that production is highly polluting. Hence, a dirty economy initially poorly endowed with physical capital can experience an enhanced growth process and a faster accumulation of physical capital. As the economy gets richer, in order to improve their reproductive health, households might trigger investment in fertility healthcare, detrimental to savings. Obviously, the impact of pollution on the demography is reduced and the accumulation of capital

might slacken. Then, the economy reaches a stationary solution.

Because such an economy is characterised by several externalities, we determine the optimal solution of a social planner that maximises a Millian utilitarian criterion. Indeed, pollution creates negative externalities on the population growth and the number of infertile couples. In addition, the individual choice of health investment affects population growth, giving birth to an adding externality. We show that at the optimum, households should always invest in fertility treatments - and thus misbehave at the competitive equilibrium if they don't. Finally, we propose a set of available economic instruments to decentralise the optimal solution. To rule out the three sources of inefficiency and even though households should invest in fertility treatments, both capital accumulation and healthcare expenditure are taxed. However, in order to deal with heterogeneity between households, the government should provide transfers to the young infertile couples and levy lump-sum taxes on revenues of all the old. Eventually, infertile households are overall subsidised. Hence, we recommend to implement a curative health policy that does not directly create incentives to invest in fertility treatments. Nonetheless, the tax on physical capital should be understood as a preventive policy tool that aims at controlling the sources of an altered reproductive health.

Our paper contributes to our knowledge to two main different strands of the economic literature. Concerns about the negative effects of pollution on health have already been developed in the literature. However most papers have used mortality or morbidity health indicators, like for instance life expectancy, productivity at work or at school (see for instance Williams III, 2000, 2003; Pautrel, 2008; Mariani et al., 2010; Varvarigos, 2010; Raffin, 2012; Raffin and Seegmuller, 2014, 2016). We focus in this paper on an alternative dimension of health which is fertility and thus, we tackle different issues directly linked to demographic ones. In line with fertility-related research, which is concerned with the determinants of fertility behaviour and emphasizes the role played by parents preferences, we contribute to the economic literature by considering an alternative variable as a determinant of fertility behaviour which is the environmental quality. Hence, we depart from the standard endogenous fertility growth models (Galor and Weil, 2000; de la Croix and Doepke, 2003; Galor, 2005) since we consider that demography evolves endogenously but is driven by environmental conditions changes rather than the usual quality-quantity trade-off. We also differ from more

recent contributions that focus on childlessness (Gobbi, 2013; Baudin et al., 2015). Indeed, this literature is interested in the endogenous choices of having children, whereas in our paper, couples suffer from the inability to conceive children. Therein, our paper is closer to Momota (2016) who also introduces heterogeneity among households due to the ability of having children. However, his concern is drastically different to ours: He focuses on the effect of exogenous population growth on the level of capital accumulation in a model without environment, whereas in our framework, population growth is endogenous and pollution determines the share of infertile couples.

The paper is organised as follows: Following the Introduction, Section 2 provides with more precise stylised facts; Section 3 presents the set-up of the model and Section 4 the *laissez-faire* equilibrium. In Section 5, we study the dynamic behaviour of the economy and the convergence towards a stationary solution. Section 6 investigates the optimal stationary solution and presents the set of optimal tools in order to decentralise the optimal solution. Finally Section 7 concludes. Technical proofs are relegated to Appendices.

2 Stylised facts

As previously mentioned, we provide some stylised facts with regards to the changes in the human reproductive health and the induced effects of pollution.

Impairment of male fertility

Impairment of male fertility has been widely studied in the epidemiological literature from a functional perspective using relatively easily collectible biomarkers of semen quality (like concentration, volume number motility and morphology) for descriptive purposes and to study trends over time.

In particular, a wide part of the literature on fertility trends has specifically related to sperm concentration: This issue was raised in the late twentieth century in the seminal paper by Carlsen et al. (1992) published in the British Medical Journal, which establishes "the falling sperm counts story" (Joffe, 2010). In their article, the authors review 61 studies published between 1938 and 1991 which had analysed sperm concentration. They conclude that mean sperm concentration had fallen from 113 to 66 million/ml over the period. This meta-analysis constitutes the starting point of a whole strand of epidemiological literature meanwhile it has met with scepticism on

grounds of laboratory methods, statistical issues, selection etc. Nonetheless, this study stimulates the analysis of time trends on sperm quality and according to more recent papers, we might still conclude to a global declining quality of spermatogenesis over the century at least in some places (see for instance, Auger et al., 1995, on French data; Van Waeleghem et al., 1996, on Belgian ones; Irvine et al., 1996, on Scottish ones; Swan et al., 2000, on Europe and North America). As an illustration, we have reported on Figure 1 more recent empirical evidence provided by various studies that cover different time periods, geographical areas.

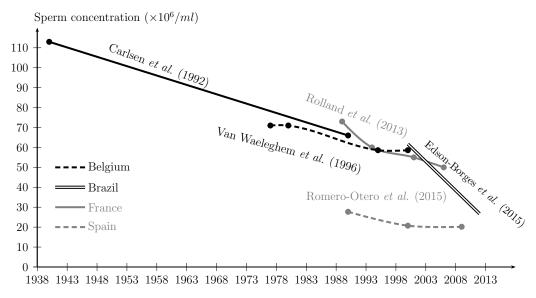


Figure 1: Evolution of sperm concentration.

Pollution and reproductive health

Once human reproductive problems are established, it remains to wonder why such issues arise and why did they increase in the last decades. One prevalent hypothesis to justify the deterioration of the male reproductive health is that it is due to ubiquitous chemical pollutants that affect the hormonal system and interfere with developmental processes in humans: the Endocrine Disruptor Chemical (EDC) hypothesis. Those EDCs are found among many classes of chemicals, including POPs, currently used pesticides,

phytoestrogens, metals, additive or contaminants in food, personal care products, cosmetics, textiles and construction materials. Human exposure-be it occupational or non-occupational to EDCs occurs via ingestion of food, dust and water, inhalation of gases and particles in the air or through dermal uptake (WHO, 2013). To illustrate our argument we may cite for instance the papers by De Rosa et al. (2003), Martenies and Perry (2013) or Zhou et al. (2014) which provides us with estimations of the harmful effects of air pollution and Particulate Matter on the male reproductive health in Italy and China, respectively. We can also cite the study by Meeker et al. (2008) in which non-occupational exposure to metals, like Molybdenum, has been implicated to explain poor semen quality. In addition, we may refer to the articles that emphasize the noxious impact of pesticides like Polychlorinated Biphenyls (PCBs), even at low levels, on human spermatogenesis. Some of them deal with occupational exposure (Tuc et al., 2007; Recio-Vega et al., 2008), non-occupational one (Swan et al., 2003; Bouvier et al., 2006; Aneck-Hahn et al., 2007; Perry et al., 2011) while the others focus on dietary factors (fruits, vegetables, meat, fish) (Mehrpour et al. (2014) or Chiu et al. (2015) among others).

Medical treatments and ARTs

Since 2000, the ART services annually grows by 5\%-10\% in developed countries (American Society of Reproductive Medicine, European Society of Human Reproduction and Embryology). In 2011, over 17 European countries that fully reported their ART activities (Kupka et al., 2016), ART babies represented 2,4% of all infants born, that is around 800 000 births, going from 1.7% in Italy to 5.8% in Denmark. Despite a substantial increase in the number of ART cycles performed worldwide, there are considerable international discrepancies in the availability of ART treatments and per capita utilisation rates (Collins, 2002). Chambers et al. (2009) compare economic aspects of ART in selected developed countries (U.S., Australia, Canada, U.K., Scandinavia and Japan) and show that they represent substantial out-of-pocket expenses. They found that the cost of a standard IVF cycle ranged from 28% of Gross National Income (GNI) per capita in the United States to 10% of GNI per capita in Japan. Moreover, before any public policy, the gross cost of a standard IVF cycle ranged from 50% of an individual's annual disposable income in the U.S., approximately 20% in the UK, Scandinavian countries and Australia, to 12% in Japan. After accounting for government subsidies, the resultant cost to the patient of an ART cycle was unchanged in the USA

and Japan (due to negligible public funding for ART treatment) but fell to approximately 12% of annual disposable income in the UK and Scandinavian countries.

3 The Model

Let us consider a two-period overlapping generations model. During the first period, the adulthood, households work, consume, save and procreate. At each date t, two types of household co-exist depending on their ability to conceive children: We distinguish Fertile households (denoted by superscript F) and Infertile ones (denoted by superscript I). In case of infertility, households may engage in medical treatments in order to improve their ability to father offspring. These fertility treatments include hormonal remedies, assisted reproduction treatments (ART) like in vitro fertilisation (IVF). They aim at augmenting the chances of parenthood so that with a probability q, initially infertile couples may have children. During the second period of life, households retire and consume their saving.

Demography. The population size of a generation born at period t is N_t . The proportion of fertile households within the population is denoted by π_t and the proportion of infertile ones obviously equals $(1 - \pi_t)$. This probability to be fertile (π_t) is randomly distributed among the population and is endogenous as it will be discussed later. On the other hand, the potential number of children per household, n, is exogenous. The total number of children at date t is given by the number of children of fertile households $(N_t n \pi_t)$ plus the number of children of successfully treated infertile households $(N_t n \pi_t)$ plus the number of children evolves overtime according to

$$N_{t+1} = N_t \times n \times [\pi_t + (1 - \pi_t)q_t]$$
 (1)

where N_t is the size of the adult generation born at date t, that is the labour force.

Households. Households derive utility from current and future consumptions as well as parenthood. Agents do not choose the number of children they have, but rather they might suffer from being not able to procreate. We do not here aim at investigating fertility behaviours $per\ se$ but consider that there is an average exogenous targeted level of fertility within the economy (or a a desired number of children) given by n. Agents are identical

ex-ante but become heterogeneous ex-post once the risk of infertility has realised. Households' preferences are represented by a utility function, which is additively separable between consumption levels and parenthood, so that

$$\begin{cases} u(c_t^F) + \delta u(d_{t+1}^F) + v \\ u(c_t^I) + \delta u(d_{t+1}^I) + q(x_t)v \end{cases}$$
 (2)

where c_t^i and d_{t+1}^i , i = F, I, denote consumptions in the first and the second period, x_t denotes the level of curative health treatment which influences the ability to procreate through the probability q_t .

Notice that the utility of parenthood, v, is constant since the number of children is exogenous. For the sake of simplicity, let us consider the following specifications. On the one hand, the utility function can be given by $u(z_t) = \ln z_t$ and, on the other hand, the probability of a successful treatment can be written as $q(x_t) = \frac{ax_t}{1+x_t}$, with $a \leq 1$. The parameter a merely accounts for the efficiency of the available medical technology or, equivalently, the level of scientific medical knowledge. In addition, and according to physicians and clinical psychologists, we consider for the remaining of the paper that the desire of parenthood is strong or said otherwise, the loss suffered from not being able to procreate is large enough. The medical or experiences studies that have been conducted in order to evaluate the distress associated with the diagnosis of infertility or treatments of infertility causes unequivocally show that the socio-psychological costs of infertility are not negligible but rather substantial, without referring to economic costs of treatments. Indeed, infertility is often felt as a terrible experience and the multi-dimensional consequences of it may encompass a severe degradation of self-esteem, syndromes of depression, loss of gender identity, self-assessed social pressure from families, friendships, or even social stigma in developing countries (Greil, 1997; Moura-Ramos et al., 2012). Using contingent valuation to directly quantify the monetary value of a baby conceived through ART, Neumann and Johannesson (1994) estimate that the willingness-to-pay for a baby was \$177 730 for potential child bearers in the event that they were infertile, and \$1.8 million for society to pay for insurance to allow couples access to ART. Hence, we formally assume that v is sufficiently high and that there exists a threshold value v such that v > v.

Let us present the budget constraints faced by households. For ease of presentation, we right now introduce a set of policy instruments that we will be useful to decentralise the optimal allocation later on (see Section 6).

Hence, during adulthood, each household is endowed with one unit of labour inelastically offered to firms for which they receive the prevailing competitive wage, w_t .¹ In addition, there are some differentiated lump-sum transfers, T_t^i . The net total income can be shared among current consumption, savings, s_t^i , and possibly for infertile households healthcare expenditure. During retirement, each couple consumes the net income which equals the revenue from her savings minus a transfer, θ_t . For both types of household, first period of life budget constraints can be expressed as follows:

$$c_t^F + s_t^F = w_t + T_t^F \tag{3}$$

$$c_t^I + s_t^I + (1 + \sigma_t)x_t = w_t + T_t^I, \tag{4}$$

where σ_t is a proportional tax on health care. The second period of life budget constraints write:

$$d_{t+1}^{i} = R_{t+1}s_{t}^{i} - \theta_{t}, \text{ for } i = F, I$$
(5)

where we assume a complete depreciation of capital and we define $R_{t+1} \equiv (1 - \rho_{t+1})r_{t+1}$, with ρ_t a proportional tax on capital income.

Government. At each date t, the government provides transfers to the young generation financed through taxes on capital, health expenditure and a lump-sum tax on the old. The balanced budget constraint of the government is given by:

$$N_t \sigma_t (1 - \pi_t) x_t + N_{t-1} [\theta_t + \rho_t r_t (\pi_{t-1} s_{t-1}^F + (1 - \pi_{t-1}) s_{t-1}^I)] = N_t (\pi_t T_t^F + (1 - \pi_t) T_t^I)$$
(6)

Firms. In this economy, one good is produced using both physical capital, K_t , and labour, L_t . Let us define *per capita* variables $y_t = Y_t/L_t$, $k_t = K_t/L_t$. In order to obtain tractable results, we assume a fairly standard Cobb-Douglas production function:

$$y_t = f(k_t) = k_t^{\alpha} \tag{7}$$

with $0 < \alpha < 1/2$. Being given the price of capital (r_t) and the competitive wage (w_t) , the optimisation program of firms yields:

¹Notice that here the health status does not affect productivity at work. Also, to keep the analysis as simple as possible, we do not introduce rearing cost of children. Since the number of offspring is assumed to be constant, enriching the analysis with such a cost will not drastically alter our analysis.

$$r_t = \alpha k_t^{\alpha - 1} \tag{8}$$

$$w_t = (1 - \alpha)k_t^{\alpha} \tag{9}$$

Pollution. At each date t, we consider some pollution flows, P_t , which arise as a side-effect of the production process. As standard in the literature, the stock of pollution evolves according to:

$$P_{t+1} = (1-b)P_t + \beta Y_t \tag{10}$$

where $\beta > 0$ captures the dirtiness degree of production and 0 < b < 1 the natural absorption rate of the environment.

As documented in Introduction, this harmful pollution constitutes a negative externality that affects the probability of being fertile. Formally, we state that $\pi_t = \pi(p_t)$, where $p_t = P_t/N_t$ accounts for the pollution "consumed" by each young man. Such an hypothesis can be justified with regards to the epidemiological literature that has identified many potential causes of impaired male reproductive health among which pesticides, metals, textiles ... (Aneck-Han et al., 2007; Mehrpour et al., 2014). In particular, fertility issues are at least partially explained by the pollution that is mostly ingested, inhaled by individuals or the one which is carried to functional organs through dermal exposure. For example, Chiu et al. (2015) found a relation between dietary pesticide exposure via fruit and vegetable intake and semen quality. An other example can be found in the paper by Meeker et al. (2008), where the authors estimate the impact of voluntarily or not metal concentration exposure on semen quality through intake of contaminated food or water.

Before analysing the equilibrium and, further on, the existence of steady states and dynamics, let us clarify our formal assumptions about this endogenous probability of being fertile, according to the above referred epidemiological literature:

Assumption 1 We assume that π is sufficiently close to 1 and $\epsilon_{\pi} \equiv \frac{\pi'(p)p}{\pi(p)}$ is close to 0. In addition $\pi'(p) \leqslant 0 \leqslant \pi''(p)$, $\pi(0) = \pi_0 > 0$, $\pi(+\infty) > (1-b)/n$, $\epsilon_{\pi} > \frac{-1}{\frac{n\pi(+\infty)}{n\pi(+\infty)+b-1} + \frac{\alpha}{1-\alpha}}$.

This assumption means that the chances of parenthood are weakly decreasing with the stock of pollution and sufficiently close to 1, which seems to be a reasonable assumption according to the empirical literature (Slama et al., 2004).

4 The Equilibrium

This section defines the inter-temporal equilibrium in the *laissez-faire* economy, the levels of policy instruments, T_t^i , σ_t , θ_t and ρ_t , being set to zero.

Households' choices. Households maximise their utility (2) under the budget constraints (3)-(5) and a positivity constraint, $x_t \ge 0$. As the framework involves, fertile couples do not expand in health and we can easily deduce their optimal level of savings, which is increasing with labour income:

$$s_t^F = \frac{\delta}{1+\delta} w_t \tag{11}$$

As for the infertile, let us note that if $x_t = 0$, then

$$s_t^F \leqslant \frac{\delta}{av} \tag{12}$$

Importantly, this inequality implies that the loss of utility from a lower level of consumption dominates the potential welfare gain associated with an improved reproductive health. Yet, as the utility function defined over consumption is concave, this inequality is verified all the more that consumption levels are initially low. Then, we can state that for low incomes, it is more likely that households do not invest in curative fertility treatments. In that configuration, $s_t^I = s_t^F$.

If equation (12) is not satisfied, then $x_t > 0$ and we get the optimal savings and effort of curative treatments that can also be expressed as functions of s_t^F :

$$s_t^I + \frac{\delta}{1+\delta} x_t = \frac{\delta}{1+\delta} w_t = s_t^F \tag{13}$$

$$(1+x_t)^2 = \frac{av}{\delta} s_t^I \tag{14}$$

Solving the system (13)-(14), we deduce the expression of x_t :

$$x_t = \sqrt{\frac{av}{\delta} s_t^F - 1 + A^2} - A$$
, with $A \equiv 1 + \frac{av}{2(1+\delta)}$ (15)

Labour market. On the labour market at date t, the supply of labour N_t being inelastic and the demand L_t being the solution to equation (9), we get that:

$$L_t = N_t = N_{t-1} \times n \times [\pi(p_{t-1}) + (1 - \pi(p_{t-1}))q(x_{t-1})]$$
(16)

Capital market. The clearing condition on the capital market entails that the supply of savings by young individuals equals the investment of firms:

$$K_{t+1} = N_t \left[\pi(p_t) s_t^F + (1 - \pi(p_t)) s_t^I \right]$$
 (17)

Using equation (16), we derive the dynamics of capital-labor ratio:

$$k_{t+1} = \frac{\pi(p_t)s_t^F + (1 - \pi(p_t))s_t^I}{n\Gamma(x_t, p_t)},$$
(18)

where $\Gamma(x_t, p_t) = [\pi(p_t) + (1 - \pi(p_t))q(x_t)]$ and $n\Gamma(x_t, p_t)$ is the growth factor of the young population. Notice that the latter decreases with pollution but this effect might be dampened by positive health investments. We can also rewrite equation (10), using market clearing conditions, to obtain:

$$p_{t+1} = \frac{(1-b)p_t + \beta f(k_t)}{n\Gamma(x_t, p_t)}$$
(19)

As for equation (18), it depicts the way the stock of pollution evolves in the long term.

Given $k_0 = K_0/L_0 \ge 0$, $p_0 = P_0/L_0 \ge 0$, the inter-temporal equilibrium is a sequence (k_t, p_t) that satisfies conditions (18) and (19) for all $t \ge 0$. Note that both k_t and p_t are predetermined variables.

From now on, let us highlight two main mechanisms at stake. First, the endogenous population growth induces a dilution effect which may strongly impact the accumulation of capital and pollution.² Second, health investment also plays a direct role on physical capital by reducing infertile individual savings.

5 Steady states and dynamics

Once we have defined the dynamic system that drives the sequence $(k_t, p_t)_{t \ge 0}$ overtime, we can characterise the equilibrium, given initial conditions (k_0, p_0) .

²Recall that the dilution effect corresponds to a decrease of *per capita* variables following an increase in the labour force or equivalently in the population growth.

Using equation (11), we derive a positive relationship between k_t and s_t^F such that $k_t = \left[\frac{1+\delta}{\delta(1-\alpha)}s_t^F\right]^{1/\alpha}$. Then, choosing for convenience (p_t, s_t^F) as variables of interest, the dynamical system (18)-(19) can be investigated according to the two prevailing configurations exposed previously: In a first step, when households do not invest in curative fertility treatments $(x_t = 0)$; and in a second step, when they do it $(x_t > 0)$.

5.1 No-health expenditure regime, $x_t = 0$

In this regime, the share of fertile households determines the way population evolves over time and the growth factor of population boils down to $n\Gamma(0, p_t) = n\pi(p_t)$. Consequently, as pollution grows, population diminishes. Using equations (18) and (19) and as inequality (12) is strictly satisfied, the dynamic system that describes the behaviour of the economy is given by:

$$\left[\frac{1+\delta}{\delta(1-\alpha)}s_{t+1}^F\right]^{1/\alpha} = \frac{s_t^F}{n\pi(p_t)} \tag{20}$$

$$p_{t+1} = \frac{(1-b)p_t + \beta \frac{1+\delta}{\delta(1-\alpha)} s_t^F}{n\pi(p_t)}$$
 (21)

A steady state, if it exists, is thus a solution (p, s^F) to the following system:

$$s^{F} = \left[\frac{\delta(1-\alpha)}{1+\delta}\right]^{\frac{1}{1-\alpha}} [n\pi(p)]^{-\frac{\alpha}{1-\alpha}} \equiv \varphi(p)$$
 (22)

$$s^{F} = \frac{\delta(1-\alpha)}{\beta(1+\delta)}p[n\pi(p) + b - 1] \equiv \psi(p), \tag{23}$$

Consistently with the fact that the psychological distress induced by the inability to procreate proves to be substantial, we make the following assumption:

Assumption 2
$$v > \delta^{-\frac{\alpha}{1-\alpha}} \left(\frac{1+\delta}{1-\alpha}\right)^{\frac{1}{1-\alpha}} \frac{\left[n\pi(+\infty)\right]^{\frac{\alpha}{1-\alpha}}}{a} \equiv \underline{v}$$

When this assumption is not satisfied, it turns out that the motive to incur fertility treatments vanishes. Thus, in the long run, the only conceivable stationary situation would be characterised by a null investment in health care. Nevertheless, we aim at studying two distinct configurations, which might better fit with real observed situations, so that couples may incur or not fertility treatments. Hence, the long-run situation reached by one economy is not trivial.

To ensure that our stationary solution exists but also fulfils inequality (12), let us state the following lemma:

Lemma 1 Under Assumptions 1 and 2, there exists a finite value \tilde{p} solving $\varphi(\tilde{p}) = \frac{\delta}{av}$ which is given by:

$$\tilde{p} = \pi^{-1} \left(\frac{[av]^{\frac{1-\alpha}{\alpha}} \delta}{n} \left(\frac{1-\alpha}{1+\delta} \right)^{\frac{1}{\alpha}} \right)$$
 (24)

so that any solution (p, s^F) solving the system (22)-(23) belongs to $(0, \tilde{p}) \times (0, \frac{\delta}{av})$.

Proof. See Appendix A.

Let us now characterise the stationary solution so that savings and the stock of pollution are low enough. The polluting intensity of production embedded by the parameter β is crucial to our analysis. Indeed, it captures the impact of production on pollution and thus on population dynamics. Through the dilution effect, the value of β determines whether there exists a steady state without health investment.

Proposition 1 Under Assumptions 1 and 2, there exists a threshold value on dirtiness of production $\overline{\beta}$ such that:

- (i) For $\beta < \overline{\beta}$, there exists a unique steady state $(p, s^F) \in (0, \tilde{p}) \times (0, \frac{\delta}{av})$ with x = 0;
- (ii) For $\beta \geq \overline{\beta}$, there exists no steady state $(p, s^F) \in (0, \tilde{p}) \times (0, \frac{\delta}{av})$ with x = 0.

Proof. See Appendix B.

To have a qualitative picture of the dynamics when infertile househols do not expend in health expenditure, let's say in the corner solution, one has to analyse the dynamic properties of the system (20)-(21), that are the areas where s_t^F and/or p_t grow and go down, respectively. Using equation (20), we can observe that $s_{t+1}^F \geqslant s_t^F$ is equivalent to $s_t^F \leqslant \varphi(p_t)$. Using equation (21), we have that $p_{t+1} \geqslant p_t$ is equivalent to $s_t^F \geqslant \psi(p_t)$. We deduce that, when

it exists, the single steady state is stable. Otherwise, the economy is driven towards an alternative regime studied in the next section.

One key mechanism to figure out this proposition goes through the dilution effect that comes along with the endogenous population growth. Any increase in pollution directly reduces the number of fertile households and this induces a slower growth of population. The overall effect of this slackening demographic growth consists in a negative dilution effect as the stock of capital per worker grows. When β is large, a small rise in production gives birth to large pollution flows and exhibits a heavy effect on the population growth. The negative dilution effect is important and allows for a continuous increase in physical capital as well as pollution. When β is lower, pollution responds less to a rise in production and the share of fertile and infertile households barely changes. Therefore, the dilution effect is more negligible. Due to the concavity of the wage with respect to capital, the increase in income is not sufficient enough to permit a further rise in investment. Eventually, the economy can reach a stationary solution.

5.2 Positive health expenditure regime, $x_t > 0$

Let us now examine the behaviour of the economy when inequality (12) is not satisfied, that is $s_t^F > \frac{\delta}{av}$. In this configuration, the evolution of the population depends also on the level of health care expenditure that might compensate the negative impact of pollution. Finally, three types of households co-exist: As before, the economy is composed of fertile couples, but the infertile ones are themselves divided into two categories, the cured and uncured infertile households. Using equations (18) and (19), the dynamic system that describes the behaviour of the economy can be reduced to:

$$F(s_{t+1}^F) = H(p_t, s_t^F) (25)$$

$$p_{t+1} = J(p_t, s_t^F) (26)$$

where:

$$F(s_{t+1}^F) = \left[\frac{1+\delta}{\delta(1-\alpha)}s_{t+1}^F\right]^{1/\alpha} \tag{27}$$

$$H(p_t, s_t^F) = \frac{\left[s_t^F - (1 - \pi(p_t))\frac{\delta}{1 + \delta}x(s_t^F)\right](1 + x(s_t^F))}{n[\pi(p_t) + x(s_t^F)(\pi(p_t) + a(1 - \pi(p_t)))]}$$
(28)

$$J(p_t, s_t^F) = \frac{[(1-b)p_t + \beta \frac{1+\delta}{\delta(1-\alpha)} s_t^F](1+x(s_t^F))}{n[\pi(p_t) + x(s_t^F)(\pi(p_t) + a(1-\pi(p_t)))]}$$
(29)

and $x(s_t^F)$ is given by (15).

A steady state with $\overline{x} > 0$, if it exists, is a solution $(\overline{p}, \overline{s}^F)$ that solves the above dynamic system (25)-(26) evaluated at the steady state. The existence and uniqueness of such a steady state is shown in the following proposition³

Proposition 2 Under Assumptions 1 and 2, there exists $\overline{a} > 0$ such that, for $a < \overline{a}$ and $s^F > \frac{\delta}{av}$:

- 1. For $\beta < \overline{\beta}$, there is no steady state $(\overline{p}, \overline{s}^F)$ with $\overline{x} > 0$;
- 2. For $\beta \geq \overline{\beta}$, there exists a unique steady state with $\overline{x} > 0$.

Proof. See Appendix C. ■

To have a qualitative picture of the dynamics when infertile households do incur fertility treatments in health expenditures, we can determine the areas where s_t^F and/or p_t grow and go down, respectively. Using equation (25), $s_{t+1}^F \geqslant s_t^F$ is equivalent to $H(s_t^F, p_t) \geqslant F(s_t^F)$, which implies that $s_t^F \leqslant \widetilde{\varphi}(p_t)$. Using equation (26), $p_{t+1} \geqslant p_t$ is equivalent to $J(s_t^F, p_t) \geqslant p_t$, which implies that $s_t^F \geqslant \widetilde{\psi}(p_t)$. Finally, we deduce that, when it exists, the steady state is stable.

When the technology is highly polluting, the dilution effect is strong, but new effects arise. First, investing in fertility treatments to augment chances of parenthood is costly and induces an eviction effect on savings. Second, by increasing the share of procreating households, the demographic growth is boosted. Since the effort in health care is a growing function of savings, these two adding effects promote the convergence to a steady state with x > 0.

5.3 Dynamics with regime-switching

Using the previous results, let us now present a global picture of the dynamics of the economy. We then consider both regimes together, i.e. $x_t = 0$ and $x_t > 0$, and describe the convergence of one economy towards the steady state. The following corollary sums up the main results and is illustrated in Figure 2 below:

 $^{^{3}}$ Let us notice that the parameter a, which captures the medical technology, is bounded above. This assumption can be supported by empirical evidence since the success of ART treatments is still limited.

⁴Note that $\widetilde{\varphi}(p_t)$ and $\widetilde{\psi}(p_t)$ are defined in Appendix C.

Corollary 1 Under Assumptions 1-2 and $a < \overline{a}$,

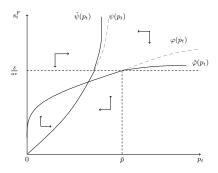
- (i) when $\beta < \overline{\beta}$, the economy converges towards the unique and stable steady state (p, s^F) with x = 0;
- (ii) when $\beta \geq \overline{\beta}$, the economy converges towards the unique and stable steady state $(\overline{p}, \overline{s}^F)$ with $\overline{x} > 0$.

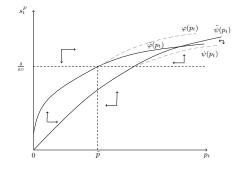
If the technology is clean enough $(\beta < \overline{\beta})$, a rise in production weakly affects pollution. Since the sensitivity of $\pi(p_t)$ to pollution is arbitrarily low, population growth is left almost unaffected and the so-called dilution effect is very small. In this configuration, the economy converges to a unique steady state, which belongs to the no-health expenditure regime. This is due to the concavity of the wage with respect to capital so that, even though the level of capital in the economy is initially large enough, it eventually decreases through time, because the labour income cannot sustain further increases in investment.

On the contrary, when the technology is dirty $(\beta \geq \overline{\beta})$, the dilution effect is stronger. Both capital and pollution can grow continuously from the no-health regime to the positive health expenditure one. Because fertility treatment entails an eviction effect on savings and pushes up the demographic growth, the economy eventually reaches a steady state with x > 0.

We would like to draw the reader's attention to the fact that the threshold value $\overline{\beta}$ is a decreasing function of v, the utility gain of being fertile. Intuitively, when infertility does not bring so much inconvenience, then the long-term solution is more likely to be featured by a null investment in health care expenditure. As we consider that the utility loss induced by the inability of being parent is large enough, one might thought that it would trigger the investment in fertility treatments. This is not necessarily true when production is clean enough.

Interestingly, note that in configuration (ii), the demographic growth pattern might be related to some fertility trends observed from the last fourty years. Let us consider that the initial stock of pollution and capital are low enough, so that p_t and s_t^F continuously grow. At the beginning, $x_t = 0$, but after a finite number of periods, the economy enters the regime with positive health expenditure and converges to the steady state with x > 0. The rise of s_t^F and p_t imply first a continuous decrease in the population growth factor





(a) a unique steady state with x = 0 (b) a unique steady state with $\overline{x} > 0$ Figure 2: Global dynamics

through time. When one switches to the regime with $x_t > 0$, a positive effect arises since infertile households invest in health and some of them succeed in having children. Then two opposite forces drive the evolution of the demographic rate of growth: A negative effect through the growing pollution stock; A positive effect through fertility treatments. If the positive effect would dominate, then the rate of growth of population would rise in this regime. As for an illustration, Habbema et al. (2009) have highlighted that early IVF treatments could substantially increase total fertility rates in western economies up to 0,11-0,25.

The analysis of the competitive equilibrium allows to highlight the role played by the pollution externality in the long run and to emphasize the outcome of pollution-induced heterogeneity. In the following, we aim at exploring what would be a first-best optimal solution.

6 Optimality and policy

Let us now consider a social planner who seeks to maximise a social welfare objective that takes into account the sum of individuals' preferences. In this context of endogenous population, this corresponds to Millian utilitarian criterion, as soon as the social planner does not grant any particular weight to the overall size of the population. To comply with this goal, she chooses the optimal levels of consumption (c^F, c^I, d^F, d^I) , curative health treatment (x), physical capital (k) and pollution (p), under the two constraints of resources and pollution. In order to derive clear cut results, we focus on the stationary solution. Consequently, the program of the central planner evaluated at the steady state can be written as follows⁵:

$$\begin{cases} \max_{c^F, c^I, d^F, d^I, x, k, p} & \pi(p) \left[\ln c^F + \delta \ln d^F + v \right] \\ & + (1 - \pi(p)) \left[\ln c^I + \delta \ln d^I + \frac{ax}{1 + x} v \right] \\ s. \ t. & k^{\alpha} \ge \pi(p) c^F + (1 - \pi(p)) (c^I + x) + \frac{\pi(p) d^F + (1 - \pi(p)) d^I}{n\Gamma(x, p)} + nk\Gamma(x, p) \\ & p = \frac{\beta k^{\alpha} + (1 - b)p}{n\Gamma(x, p)} \\ & x \ge 0 \end{cases}$$

We denote by λ , μ and ξ the Lagrange multipliers (or shadow prices) associated to the resource constraint, the law of motion of the pollution stock and the positivity constraint on x, respectively.

First of all, at the optimum, we can establish that consumption levels should be equalised among heterogeneous agents, $c^F = c^I = c^*, d^F = d^I = d^*$. Second, we obtain the trade-off between consumptions over life cycle:

$$\delta c^* = \frac{d^*}{n\Gamma(x, p)};\tag{30}$$

The trade-off between consumption and health:

$$-\frac{1}{c^*} + \frac{av}{(1+x^*)^2} = -\frac{c^*\Gamma_x(x^*, p^*)}{(1-\pi(p^*))} \times \left[\frac{d^*}{n\Gamma(x^*, p^*)^2} - nk^* \right]$$

$$-\frac{\mu^*}{(1-\pi(p^*))} \frac{p^*\Gamma_x(x^*, p^*)}{n\Gamma(x^*, p^*)} + \xi;$$
(31)

The trade-off between generations:

$$\alpha(k^*)^{\alpha-1} = f'(k^*) = n\Gamma(x^*, p^*) + \mu^* c^* \frac{\beta \alpha(k^*)^{\alpha-1}}{n\Gamma(x^*, p^*)}.$$
 (32)

Let us show that μ^* is strictly positive:

⁵See Appendix D for more details.

⁶Superscripts * indicate the utilitarian optimal solution.

Lemma 2 Under Assumptions 1 and 2, we have $\mu^* > 0$ for all $x^* \ge 0$ because v is sufficiently large, such that

$$v > \frac{\alpha - \delta(1 - 2\alpha)}{(1 - \alpha)\pi(+\infty)} \tag{33}$$

Proof. See Appendix E. ■

By inspection of equation (31), curative health expenditure entails a marginal utility gain and a marginal cost in terms of consumption but also induces externalities that go through their impact on the endogenous growth rate of population. When households incur more fertility treatment, the growth factor of population rises so that the weight granted to old households' consumption diminishes but, simultaneously, the overall production must afford increased productive investment. In addition, it also reduces the pollution stock per worker.

As for equation (32), it represents a green golden rule. Keeping in mind that investment, production and pollution are closely related, more capital means more pollution and thus less population, lowering the cost of productive investment. Nevertheless, more pollution induces more infertile households and thus lower welfare. This last negative effect reduces the optimal level of capital accumulation.

As we will see in Section 6.2, the analysis of policy instruments will help us to clear up the comparison between the *laissez-faire* equilibrium and the social optimal one. Let us now study the existence and the properties of an optimal allocation.

6.1 Social optimal allocation

Although we do not know whether the optimal level of health expenditure is higher than the *laissez-faire* one, we can show that it should be strictly positive.

Proposition 3 Consider that Assumptions 1-2 and inequality (33) hold. There is no optimal allocation with $x^* = 0$ because v is sufficiently large.

Proof. See Appendix F.

Following proposition 3, infertile households misbehave at the competitive equilibrium if they choose not to expand in fertility treatments. Thus, an optimal allocation satisfies $x^* > 0$. Let us now prove the existence of such an allocation.

Proposition 4 Consider that Assumptions 1-2 and inequality (33) hold, there exists an optimal allocation (x^*, k^*, p^*) with $x^* > 0$.

Proof. See Appendix G. ■

Then, we can establish that this optimal solution is indeed a maximum as stated in Proposition 5 below:

Proposition 5 Consider that Assumptions 1-2 and inequality (33) hold. There exists a threshold value \tilde{a} , such that for $a < \tilde{a}$, any optimal allocation with $x^* > 0$ satisfies the second order conditions of the central planner programme and implies that such a solution is unique.

Proof. See Appendix H.

Through Propositions 4 and 5, the unique solution is such that the level of health investment is strictly positive. This result can be understood as a direct consequence of the utilitarian criterion, but does not reflect here any potential natalist bias of the social planner. In particular, this does not reveal the preference of the social planner with regards to the size of the global population at the steady state. In addition, it is worth to mention that since x^* is strictly positive, fertility treatments are a source of externality through its stimulating effect on population growth. This creates an adding inefficiency, besides the effect of pollution which is double. On the one hand, pollution affects the sharing between fertile and infertile households. On the other hand, more pollution means less population.

6.2 Optimal policy

Once we have identified the optimal solution, we naturally wonder how to reach it in a private equilibrium. Let us now examine how the social optimum can be decentralised. Comparing the *laissez-faire* solution with the first-best optimum, one sees that the social optimum can be decentralised

with appropriate choices of taxes for the environmental damage. We have highlighted three sources of inefficiency. Moreover, taking into account that optimal consumptions are the same for all agents, the positive costs induced by health investment should be offset by a government intervention. Consequently, we come out with five instruments, one being used to balance the government budget. The following proposition summarises our results.

Proposition 6 Consider that Assumptions 1-2, $a < \min\{\tilde{a}, \overline{a}\}$ and inequality (33) hold. The social optimum can be decentralised by means of the following instruments:

- (i) Intra-generational lump-sum transfers allowing the equalisation of consumption levels across the fertile and the infertile, T^F and T^I satisfying $T^F = 0$ and $T^I = (1 + \sigma)x^* > 0$;
- (ii) A proportional tax on capital income allowing the capital stock to reach its optimal level, k^* , $\rho = \frac{\mu^* c^* \beta}{n\Gamma^*} = 1 \frac{n\Gamma^*}{\alpha(k^*)^{\alpha-1}} \in (0,1);$
- (iii) A proportional tax on fertility treatment, σ , allowing the level of health expenditure to reach its optimal level, x^* , such that $\frac{\sigma}{1+\sigma} = \frac{1}{v} \left(\frac{nk^*}{c^*} \frac{\delta + \mu^* p^*}{\Gamma^*} \right) > 0$;
- (iv) A positive lump-sum tax to old, $\theta > 0$, to balance the government budget.

Proof. See Appendix I.

Firstly, notice that we need not to impose transfer for young fertile couples $(T^F=0)$. Thus, to implement the optimal level of curative health investment and to guarantee that young and old consumptions are identical among the two types of couples, we use two instruments: A lump-sum transfer to the infertile and a tax on fertility treatments. Secondly, the decentralisation of the social optimum requires a positive tax on capital income to control the level of pollution and to ensure the achievement of the green golden rule (see equation (32)). Notice that the introduction of capital taxation is more relevant than a policy that would aim at reducing the dirtiness of production (β) . Indeed, the role of capital taxation is twofold: First, it corrects for the level of polluting emissions and second, it avoids a potential physical capital over-accumulation. Finally, θ permits to implement the social level of consumption when old.

The positiveness of θ involves a taxation of old households' consumption. This incites households to save more and it displays two outcomes. On the one hand, it triggers capital accumulation and therefore pollution. The capital tax restores the optimal level of capital. On the other hand, it allows for positive health expenditure through the household's trade-off between consumption and health. However, in order to reach the positive and optimal level of fertility treatments, a positive tax on healthcare is required. Finally, the lump-sum transfer to infertile young is used to rule out heterogeneity between young consumptions.

This result might be at first sight surprising and counter-intuitive since it drives the government to tax fertility treatments. Nevertheless, let us reexamine the infertile household's budget constraint: $c^I + (1 + \sigma - T^I/x)x + s^I = w$. Taking into account the redistribution effect through T^I , the health policy design can be summarised by $t(x) \equiv \sigma - T^I/x$. At the optimum, we easily see that $t(x^*) < 0.7$ Overall, the curative treatments are subsidised by the government. Indeed, despite the positive tax on fertility treatment, the redistribution from the old $(\theta > 0)$ to the infertile couples provides an incentive health policy. It can be interpreted as a sanitary curative policy, whereas the tax on physical capital should be understood as a preventive policy tool. Indeed, capital taxation serves as an environmental policy instrument that controls the level of pollution and shapes the sharing between the fertile and the infertile. Hence, we argue that fighting against the sources of altered reproductive health is more relevant than directly inducing households to incur health treatments.

7 Concluding remarks

Based on epidemiological evidence, we assume that pollution reduces fertility. In this paper, we analyse the implications of such a feature considering an OLG economy where households with impaired fertility may incur health treatments in order to increase their chances of parenthood and characterise the situation reached in the long run. We also examine the optimal allocation and determine the policy that allows its decentralisation.

Our main results are the following: As the economy gets dirtier, there is a stronger negative dilution effect that enhances the growth process and the

The such that t(x) < 0 and $T^I > 0$, t(x) is strictly increasing and there exists $\hat{x} = T^I/\sigma$ such that t(x) < 0 for $x < \hat{x}$ and t(x) > 0 for $x > \hat{x}$.

accumulation of physical capital. Then, the economy might reach in the long run a stationary equilibrium with higher levels of capital and pollution per worker, despite the health cost incurred by infertile couples and the fact that population growth declines. On the normative side, our analysis highlights that the long-run social optimum can be decentralised. We underscore that to correct for both the externalities of pollution and the induced-health inefficiency, it is necessary to tax physical capital while it requires to globally subsidy mostly harmed agents within the economy.

References

- [1] Aneck-Hahn, N., Schulenburg, G. W., Bornman, M. S., Farias, P. and De Jager, C. (2007), "Impaired semen quality associated with environmental DDT exposure in young men living in a malaria area in the Limpopo province, South Africa", *Journal of Andrology*, 28, 423-434.
- [2] Auger, J., Kunstmann, J. M., Czyglik, F. and Jouannet, P. (1995), "Decline in semen quality among fertile men in Paris during the past 20 years", *The New England Journal of Medicine*, 332(5), 281-285.
- [3] Baudin, T., de le Croix, D. and Gobbi, P. E. (2015), "Fertility and childlessness in the United States", *American Economic Review* 105, 1852-1882.
- [4] Boivin, J., Bunting, L., Collins, J. A. and Nygren, K. G. (2007), "International estimates of infertility prevalence and treatment seeking: Potential need and demand for infertility medical care", *Human Reproduction*, 22, 1506-1512.
- [5] Bouvier, G., Blanchard, O., Momas, I. and Seta, N. (2006), "Pesticide exposure to non-occupationally exposed subjects compared to some occupational exposure: A french pilot study", *Science of the Total Environment*, 366, 74-91.
- [6] Carlsen, E., Giwercman, A., Keiding, N. and Skakkebk, N. (1992), "Evidence for decreasing quality of semen quality during past 50 years", British Medical Journal, 305, 609-613.

- [7] Chambers G.M., Sullivan E.A., Ishihara O., Chapman M.G., FRANZCOG and Adamson G.D. (2009), "The economic impact of assisted reproductive technology: A review of selected developed countries", Fertility and Sterility, 91(6), 2281-2294.
- [8] Chiu Y. H., Afeiche M. C., Gaskins A. J., Williams P. L., Tanrikut C., Hauser R. and Chavarro J. E. (2015), "Fruit and vegetable intake and their pesticide residues in relation to semen quality among men from a fertility clinic", *Human Reproduction*, 30, 1352-1341.
- [9] Collins J. (2002), "An international survey of the health economics of IVF and ICSI", *Human Reproduction Update*, 8, 265-277.
- [10] Connolly, M., Hoorens, S. and Chambers, G. (2010), "The costs and consequences of assisted reproductive technology: An economic perspective" *Human Reproduction Update*, 16, 603-613.
- [11] de la Croix, D. and Doepke, M. (2003), "Inequality and growth: Why differential fertility matters", *The American Economic Review*, 93, 1091-1113.
- [12] De Rosa, M., Zarrilli, S., Paesano, L. Carbone, U., Boggia, B., Petretta, M., Maisto, A., Cimmino, F., Puca, G., Colao, A. and Lombardi, G. (2003), "Traffic pollutants affect fertility in men", *Human Reproduction*, 18, 1055-1061.
- [13] ESHRE Capri Working Group (2010), "Europe the continent with the lowest fertility", *Human Reproduction Update*, 16, 590-602.
- [14] Galor, O. (2005), "From stagnation to growth: Unified growth theory", Volume Handbook of Economic Growth, Chapter 1, 171-293, Aghion, P and Durlauf, S.N eds.
- [15] Galor, O. and Weil, D. (2000), "Population, technology, and growth: From Malthusian stagnation to the demographic transition and beyond", *The American Economic Review*, 90, 806-828.
- [16] Greil, A. (1997), "Infertility and psychological distress: A critical review of the literature", *Social Science and Medicine*, 45, 1697-1704.

- [17] Gobbi, P. E. (2013), "A model of voluntary childlessness", *Journal of Population Economics*, 26, 963-982.
- [18] Inhorn, M. and Patrizio P. (2015), "Infertility around the globe: New thinking on gender, reproductive technologies and global movements in the 21st century", *Human Reproduction Update*, 21, 411-426.
- [19] Irvine, S., Cawood, E., Richardson, D., MacDonald, E. and Aitken, J. (1996), "Evidence of deteriorating semen quality in the United Kingdom: Birth cohort study in 577 men in Scotland over 11 years", *British Medical Journal*, 312, 467-471.
- [20] Joffe M. (2010) "What has happened to human fertility?", Human Reproduction, 25(2), 295-307.
- [21] Kupka, M., D'Hooghe, T., Ferraretti, A., de Mouzon, J., Erb, K., Castilla, J., Calhaz-Jorge, C., De Geyter, Ch. and Goossens, V. (2016), "Assisted reproductive technology in Europe, 2011: Results generated from European registers by ESHRE", Human Reproduction, 31, 233-248.
- [22] Mariani, F., Perez-Barahona, A. and Raffin, N. (2010), "Life expectancy and the environment", *Journal of Economic Dynamics and Control* 34, 798-815.
- [23] Martenies, S. E. and Perry, M. J. (2013), "Environmental and occupational pesticide exposure and human sperm parameters: A systematic review", *Toxicology*, 307, 66-73.
- [24] Mascarenhas, M., Flaxma, S., Boerma, S., Vanderpeol, T. and Stevens, G. (2012), "National, regional and global trends in infertility prevalence since 1990: A systematic analysis of 277 health surveys", PLOS Medicine, 9, 1-12.
- [25] Meeker, J., Rossano, M., Protas, B., Diamond, M., Pushel, E., Daly, D., Paneth, N. and Wirth, J. (2008), "Cadnium, lead and other metals in relation to semen quality: Human evidence for molybdenum as a male reproductive toxicant", *Environmental Health Perspectives*, 116, 1473-1479.

- [26] Mehrpour, O., Karrari, P., Zamani, N., Tsatsakis, A. and Abdollahi, M. (2014), "Occupational exposure to pesticides and consequences on male semen and fertility: A review", *Toxicology Letters*, 230, 146-156.
- [27] Momota, A. (2016), "Intensive and extensive margins of fertility, capital accumulation, and economic welfare", *Journal of Public Economics* 133, 90-110.
- [28] Myrskylä, M., Kohler, H. and Billari, F. (2009), "Advances in development reverse fertility declines", Nature, 460, 740-743.
- [29] Moura-Ramos, M., Gameiro, S., Canavarro, M.C. and Soares, I. (2012), "Assessing infertility stress: Re-examining the factor structure of the Fertility Problem Inventory", *Human Reproduction*, 27, 496-505.
- [30] Neumann P.J., Johannesson M. (1994), "The willingness to pay for in vitro fertilization: A pilot study using contingent valuation", *Medical Care*, 32, 686-699.
- [31] Pautrel, X. (2008) "Reconsidering the impact of the environment on long-run growth when pollution influences health and agents are finite-lifetime", *Environmental and Resource Economics* 40, 37-52.
- [32] Perry, M. J., Venners, S. A., Chen, X., Liu, X., Tang, G., Xing, H., Barr, D. B. and Xu, X. (2011) "Organophosphorous pesticide exposures and sperm quality", *Reprod. Toxicol.* 31, 75-79.
- [33] Raffin, N. (2012) "Children's environmental health, education and economic development", Canadian Journal of Economics, 45, 996-1022.
- [34] Raffin, N. and Seegmuller, T. (2014) "Longevity, pollution and growth", *Mathematical Social Sciences*, 69, 22-33.
- [35] Raffin, N. and Seegmuller, T. (2016) "The cost of pollution on longevity, welfare and economic stability", *Environmental and Resource Economics*, forthcoming.
- [36] Recio-Vega, R., Ocampo-Gomez, G., Borja-Aburto, V. H., Moran-Martinez, J. and Cebrian-Garcia, M. E. (2008), "Organophosphorus pesticide exposure decreases sperm quality: Association between sperm parameters and urinary pesticide levels", Journal of Applied Toxicology, 28, 674-680.

- [37] Slama, R., Jensen, T. K., Scheike, T., Ducot, B., Spira, A., and Keiding, N. (2004) "How would a decline in sperm concentration over time influence the probability of pregnancy?", *Epidemiology*, 15(4), 458-465.
- [38] Swan, S., Elkin, E. and Fenster, L. (1997),"Have sperm densities declined? A reanalysis of global trend data", *Environmental Health Perspectives*, 105, 1228-1232.
- [39] Swan, S., Elkin, E. and Fenster, L. (2000),"The question of declining sperm density revisited: An analysis of 101 studies published 1934-1996", Environmental Health Perspectives, 108, 961-966.
- [40] Swan, S. H., Kruse, R. L., Liu, F., Barr, D. B., Drobnis, E. Z., Redmon, J. B., Wang, C., Brazil, C., Overstreet, J. W. and Study for Future Families Research Group (2003), "Semen quality in relation to biomarkers of pesticide exposure", Environmental Health Perspectives, 111, 1478-1484.
- [41] Tuc, V. P., Wangsuphachart, V., Tasanapradit, P., Fungladda, W., Van Trong, P. and Nhung, N. T. (2007), "Impacts of pesticide use on semen characteristics among rice farmers in Kienxuong District, Thaibinh Province, Vietnam", Studies, 38, 569-575.
- [42] Van Waeleghem, K., De Clercq, N., Vermeulen, L., Schoonjans, F. and Comhaire, F. (1996), "Deterioration of sperm quality in young healthy Belgian men", *Human Reproduction* 11, 325-329.
- [43] Varvarigos, D (2010), "Environmental degradation, longevity and the dynamics of economic development", Environmental and Resource Economics, 46, 59-73. 25
- [44] World Health organization (2013), State of the science of Endocrine Disrupting Chemicals 2012, ISBN: 9789241505031.
- [45] Williams, R. (2002) "Environmental tax interactions when pollution affects health or productivity", *Journal of Environmental Economics and Management* 44, 261-70.
- [46] Williams, R. (2003), "Health effects and optimal environmental taxes", Journal of Public Economics 87, 323-35.

[47] Zhou, N., Cui, Z., Yang, S., Han, X., Chen, G., Zhou, Z., Zhai, C., Ma, M., Li, L., Cai, M., Li, Y., Ao, L., Shu, W., Liua, J. and Cao, J. (2014), "Air pollution and decreased semen quality: A comparative study of Chongqing urban and rural areas", Environmental Pollution, 187, 145-152.

Appendices

A Proof of Lemma 1

Let us examine the properties of equation (22) in order to determine the conditions under which inequality (12) strictly holds. Indeed, thanks to Assumption 1, we can easily see that $\varphi(0) > 0$ and is bounded above. In addition, the derivative of the function with respect to p allows us to state that $\varphi(p)$ is an increasing function since $\epsilon_{\varphi}(p) \equiv \frac{\varphi'(p)p}{\varphi(p)} = -\frac{\alpha}{1-\alpha}\epsilon_{\pi} > 0$.

that $\varphi(p)$ is an increasing function since $\epsilon_{\varphi}(p) \equiv \frac{\varphi'(p)p}{\varphi(p)} = -\frac{\alpha}{1-\alpha}\epsilon_{\pi} > 0$. In addition, we can show that that $\varphi(+\infty) > \frac{\delta}{av}$ if v is sufficiently large so that $v > \delta^{-\frac{\alpha}{1-\alpha}} \left(\frac{1+\delta}{1-\alpha}\right)^{\frac{1}{1-\alpha}} \frac{[n\pi(+\infty)]^{\frac{\alpha}{1-\alpha}}}{a}$. Then, there exists a unique threshold value \tilde{p} solution of $\varphi(\tilde{p}) = \frac{\delta}{av}$, such that for $p \in (0, \tilde{p})$ inequality (12) holds, otherwise it doesn't.

B Proof of Proposition 1

To prove Proposition 1, let us first study the properties of the function $\psi(p)$. Thanks to Assumption 1, we can easily see that $\psi(0)=0$ and $\epsilon_{\psi}(p)\equiv \frac{\psi'(p)p}{\psi(p)}=1+\frac{n\pi(p)}{n\pi(p)+b-1}\epsilon_{\pi}>0$. Moreover, we can observe that $\psi(p)$ is bounded below by $\frac{\delta(1-\alpha)}{\beta(1+\delta)}p[n\pi(+\infty)+b-1]$. Since $\varphi(0)>\psi(0)$, there exists a steady state with x=0 if $\psi(\tilde{p})>\varphi(\tilde{p})$, where \tilde{p} is given by (24). Since $\psi(p)$ is the only equation that depends on the parameter β , we use this parameter as a scaling parameter. Then, we can rephrase the previous conditions so that there exists a steady state if $\beta<\overline{\beta}$ where

$$\overline{\beta} \equiv \frac{av(1-\alpha)}{1+\delta} \tilde{p} \left[[av]^{\frac{1-\alpha}{\alpha}} \delta \left(\frac{1-\alpha}{1+\delta} \right)^{\frac{1}{\alpha}} + b - 1 \right]$$
(B.1)

and $\overline{\beta}$ is strictly positive under Assumption 1. Moreover, Assumption 1 allows us to state that the steady state is unique since $\epsilon_{\psi}(p) > \epsilon_{\varphi}(p)$ for all

 $p \in (0, \tilde{p})$ as soon as

$$\epsilon_{\pi} \left[\frac{n\pi(p)}{n\pi(p) + b - 1} + \frac{\alpha}{1 - \alpha} \right] > -1 \tag{B.2}$$

which is ensured as ϵ_{π} is sufficiently close to 0 for all p > 0.

Finally, if $\beta > \overline{\beta}$ and $\varphi(0) > \psi(0)$, then it means that $\psi(\tilde{p}) < \varphi(\tilde{p})$. In that configuration, there is no steady state with x = 0. Indeed, according to Assumption 1, $\epsilon_{\psi}(p) > \epsilon_{\varphi}(p)$ for all $p \in (0, \widetilde{p})$. Then, the two functions can never cross.

C Proof of Proposition 2

As a first step, we study the implicit relationships between s^F and p involved by equations (25) and (26) evaluated at the steady state. It yields:

$$F(s^F) = H(s^F, p) \tag{C.3}$$

$$p = J(s^F, p) \tag{C.4}$$

Lemma C.1 Consider that Assumptions 1 and 2 hold, then there exists $\overline{a} > 0$ such that equation (C.3) implicitly defines $s^F = \widetilde{\varphi}(p)$, with $\epsilon_{\widetilde{\varphi}} \equiv \widetilde{\varphi}'(p)p/\widetilde{\varphi}(p) > 0$ if $a < \overline{a}$.

Proof. Differentiating equation (C.3), we get:

$$\epsilon_{\widetilde{\varphi}} = \frac{\epsilon_{H/p}}{\frac{F'(s^F)s^F}{F'(s^F)} - \epsilon_{H/s^F}} \tag{C.5}$$

where $\epsilon_{H/p} \equiv \frac{\partial H(s^F,p)}{\partial p} \frac{p}{H(s^F,p)}$ and $\epsilon_{H/s^F} \equiv \frac{\partial H(s^F,p)}{\partial s^F} \frac{s^F}{H(s^F,p)}$. We first focus on the sign of ϵ_{H/s^F} . As a preliminary result, using equation (15), we compute the following elasticity:

$$\frac{x'(s^F)s^F}{x(s^F)} = \frac{\frac{av}{2\delta}s^F}{x(s^F)[x(s^F) + A]}$$
 (C.6)

Differentiating (28) evaluated at the steady state and using (15) and (C.6), we obtain:

$$\epsilon_{H/sF} = \frac{s^F}{x+A} \left[\frac{x+1+\pi \frac{av}{2(1+\delta)}}{s^F - (1-\pi)\frac{\delta}{1+\delta}x} - \frac{a^2(1-\pi)\frac{v}{2\delta}}{(1+x)[\pi + x(\pi + a(1-\pi))]} \right]$$

Using (13) and (14), we deduce that $s^F = \frac{\delta}{av}(1+x)^2 + \frac{\delta}{1+\delta}x$. Using this expression and equation (15), we can show that:

$$\epsilon_{H/sF} < \frac{\frac{\delta}{av}(1+x)^2 + \frac{\delta}{1+\delta}x}{\frac{\delta}{av}(1+x)^2 + \pi\frac{\delta}{1+\delta}x} \frac{x+1+\pi\frac{av}{2(1+\delta)}}{x+1+\frac{av}{2(1+\delta)}}$$

The right-hand side of this inequality is lower than $1/\alpha$ if and only if:

$$(\alpha - 1)\frac{\delta}{av}(1+x)^3 + (\alpha - \pi)\frac{\delta}{1+\delta}x(1+x) + (\alpha \pi - 1)\frac{\delta}{2(1+\delta)}(1+x)^2 + (\alpha - 1)\pi\frac{av\delta}{2(1+\delta)^2}x < 0$$

This inequality holds because $\alpha < \pi$. Since $\frac{F'(s^F)s^F}{F(s^F)} = \frac{1}{\alpha}$, it means that $\frac{F'(s^F)s^F}{F(s^F)} - \epsilon_{H/s^F} > 0$.

Differentiating now (28) at the steady state with respect to p and using again the expression of s^F , we get:

$$\epsilon_{H/p} = -\epsilon_{\pi} \pi \left[\frac{1 + x(1 - a)}{\pi + x[\pi + a(1 - \pi)]} - \frac{x}{(1 + x)^2 \frac{1 + \delta}{av} + \pi x} \right]$$
(C.7)

This elasticity is lower than one and its sign is given by the following polynomial:

$$x^{3} \frac{(1-a)(1+\delta)}{av} + x^{2} \left[\frac{(1+\delta)(3-2a)}{av} - a \right] + x \frac{(1+\delta)(3-a)}{av} + \frac{1+\delta}{av}$$

It is strictly positive if the coefficient of x^2 is positive. There exists $\overline{a} > 0$ such that this last condition is satisfied if $a < \overline{a}$. In this case, $\epsilon_{H/p} > 0$, which concludes the proof of the lemma.

Lemma C.2 Consider that Assumptions 1 and 2 hold, then equation (C.4) implicitly defines $s^F = \widetilde{\psi}(p)$, with $\epsilon_{\widetilde{\psi}} \equiv \widetilde{\psi}'(p)p/\widetilde{\psi}(p) > 0$.

Proof. Using equation (C.4), we get:

$$\epsilon_{\widetilde{\psi}} = \frac{1 - \epsilon_{J/p}}{\epsilon_{J/s^F}} \tag{C.8}$$

where $\epsilon_{J/p} \equiv \frac{\partial J(s^F,p)}{\partial p} \frac{p}{J(s^F,p)}$ and $\epsilon_{J/s^F} \equiv \frac{\partial J(s^F,p)}{\partial s^F} \frac{s^F}{J(s^F,p)}$. Differentiating (29) at the steady state with respect to p and using (26),

Differentiating (29) at the steady state with respect to p and using (26), we get:

$$\epsilon_{J/p} = \frac{(1-b)(1+x) - n\pi(1+x(1-a))\epsilon_{\pi}}{n[\pi + x(\pi + a(1-\pi))]}$$
(C.9)

Using (C.9), we deduce that:

$$1 - \epsilon_{J/p} = \frac{nxa(1 - \pi - \pi\epsilon_{\pi}) + (1 + x)[n\pi(1 + \epsilon_{\pi}) - (1 - b)]}{n[\pi + x(\pi + a(1 - \pi))]} > 0$$

under Assumption 1. Differentiating now (29) at the steady state with respect to s^F , and using (26) and (C.6), we obtain:

$$\epsilon_{J/s^F} = \frac{s^F}{(1-b)p + \frac{\beta(1+\delta)}{\delta(1-\alpha)}s^F}B$$

with

$$B \equiv \frac{\beta(1+\delta)}{\delta(1-\alpha)} - \frac{av}{2\delta} \frac{a(1-\pi)np}{(x+A)(1+x)^2}$$

Using (29) and (C.4), we have that $n\pi p \leqslant (1-b)p + \beta \frac{1+\delta}{\delta(1-\alpha)}s^F$, which means that $p \leqslant \frac{\beta \frac{1+\delta}{\delta(1-\alpha)}s^F}{n\pi+b-1}$. Therefore,

$$B \geqslant \frac{\beta(1+\delta)}{\delta(1-\alpha)} \left[1 - \frac{\frac{va^{2}(1-\pi)}{2\delta}}{(x+A)(1+x)^{2}} \frac{ns^{F}}{n\pi + b - 1} \right]$$

Since $s^F < (1+x)^2(\frac{\delta}{av} + \frac{\delta}{1+\delta})$, we get:

$$B > \frac{\beta(1+\delta)}{\delta(1-\alpha)} \left[1 - \frac{vna^2(1-\pi)}{2\delta(x+A)} \frac{\frac{\delta}{av} + \frac{\delta}{1+\delta}}{n\pi + b - 1} \right]$$

The right-hand side of this inequality is strictly positive if:

$$(n\pi + b - 1)\left(1 + \frac{av}{2(1+\delta)}\right) > \frac{a(1-\pi)n}{2}\left(1 + \frac{av}{1+\delta}\right)$$

which is satisfied for π sufficiently close to 1 as stated in Assumption 1. In this case, $\epsilon_{J/s^F} > 0$, which proves the lemma.

Lemma C.3 Consider that Assumption 1 and 2 hold, for $s^F > \frac{\delta}{av}$, we have:

- $s^F = \widetilde{\psi}(p) > \psi(p);$
- $s^F = \widetilde{\varphi}(p) < \varphi(p)$.

where $\varphi(p)$ and $\psi(p)$ are respectively given by (18) and (19).

Proof. As a preliminary result, we can show that $\frac{1+x}{\pi+x(\pi+a(1-\pi))}$ is decreasing with respect to x.

Using (26) and (29), we deduce that:

$$p = J(s^F, p) < \frac{(1-b)p + \frac{\beta(1+\delta)}{\delta(1-\alpha)}s^F}{n\pi(p)}$$

which is equivalent to:

$$s^{F} > \frac{\delta(1-\alpha)}{\beta(1+\delta)}p[n\pi(p) + b - 1] = \psi(p)$$

This proves the first part of the lemma. To prove the second part, we note, using the preliminary result and (28), that $H(s^F, p) < \frac{s^F}{n\pi(p)}$. Using (25) and (27), it implies that:

$$\left[\frac{1+\delta}{\delta(1-\alpha)}s^F\right]^{1/\alpha} < \frac{s^F}{n\pi(p)}$$

This inequality is equivalent to:

$$s^F < \left[\frac{\delta(1-\alpha)}{1+\delta}\right]^{\frac{1}{1-\alpha}} [n\pi(p)]^{-\frac{\alpha}{1-\alpha}} = \varphi(p)$$

A direct implication of this lemma is that, using the beginning of the proof of Proposition 1, we deduce that $\lim_{p\to+\infty}\widetilde{\psi}(p)=+\infty$, while $\lim_{p\to+\infty}\widetilde{\varphi}(p)$ has a finite value. In addition, Assumption 1 implies that $\epsilon_{\widetilde{\varphi}}$ is close to 0, which ensures uniqueness. We easily deduce Proposition 2.

D The optimal programme

To solve the optimal programme, let us write the Lagrangian as:

$$\mathcal{L} = \pi(p) \left[\ln c^{F} + \delta \ln d^{F} + v \right] + (1 - \pi(p)) \left[\ln c^{I} + \delta \ln d^{I} + \frac{ax}{1 + x} v \right]$$

$$+ \lambda \left[k^{\alpha} - \pi(p)c^{F} - (1 - \pi(p))(c^{I} + x) - \frac{\pi(p)d^{F} + (1 - \pi(p))d^{I}}{n\Gamma(x, p)} - nk\Gamma(x, p) \right]$$

$$+ \mu \left[p - \frac{\beta k^{\alpha} + (1 - b)p}{n\Gamma(x, p)} \right] + \xi x$$

we obtain the following First Order Conditions:

$$c^i$$
: $\frac{1}{c^i} = \lambda$, $i = F, I$ (D.10)

$$d^{i}$$
: $\frac{\delta n\Gamma(x,p)}{d^{i}} = \lambda$, $i = F, I$ (D.11)

$$x : \frac{(1-\pi(p))av}{(1+x)^2} + \lambda \Gamma_x(x,p) \left\{ \frac{\left[\pi(p)d^F + (1-\pi(p))d^I\right]}{n\Gamma(x,p)^2} - nk \right\}$$
$$-\lambda(1-\pi(p)) + \mu \Gamma_x(x,p) \frac{\left[\beta k^{\alpha} + (1-b)p\right]}{n\Gamma(x,p)^2} + \xi = 0$$
(D.12)

$$k : \lambda \left[\alpha k^{\alpha - 1} - n\Gamma(x, p) \right] - \mu \frac{\beta \alpha k^{\alpha - 1}}{n\Gamma(x, p)} = 0$$
 (D.13)

$$p : \pi'(p) \left[\ln c^{F} + \delta \ln d^{F} + v - \ln c^{I} - \delta \ln d^{I} - \frac{ax}{1+x} v \right]$$

$$-\lambda \left\{ \frac{\pi'(p) \left(d^{F} - d^{I} \right) \Gamma(x, p) - \Gamma_{p}(x, p) \left[\pi(p) d^{F} + (1 - \pi(p)) d^{I} \right]}{n \Gamma(x, p)^{2}} \right\}$$

$$-\lambda \pi'(p) \left(c^{F} - c^{I} - x \right) - \lambda n k \Gamma_{p}(x, p)$$

$$+\mu \left\{ 1 - \left[\frac{(1 - b) \Gamma(x, p) - \Gamma_{p}(x, p) \left(\beta k^{\alpha} + (1 - b) p \right)}{n \Gamma(x, p)^{2}} \right] \right\} = 0 \quad (D.14)$$

$$k^{\alpha} = \pi(p)c^{F} + (1 - \pi(p))(c^{I} + x) + \frac{\pi(p)d^{F} + (1 - \pi(p))d^{I}}{n\Gamma(x, p)} + nk\Gamma(x, p) \quad (D.15)$$

$$p = \frac{\beta k^{\alpha} + (1 - b)p}{n\Gamma(x, p)}$$
 (D.16)

$$\xi x = 0 \tag{D.17}$$

with complementary slackness and

$$\Gamma_x(x,p) = (1-\pi(p))\frac{a}{(1+x)^2} > 0$$
 (D.18)

$$\Gamma_p(x,p) = \pi'(p) \frac{1+x(1-a)}{1+x} < 0$$
 (D.19)

E Proof of Lemma 2

If $x^* = 0$, we have $\Gamma(x, p) = \pi(p)$, $\Gamma_x(x, p) = a(1 - \pi(p))$ and $\Gamma_p(x, p) = \pi'(p)$. Using (D.14), we get:

$$\mu^* = \frac{-\epsilon_\pi \left[v\pi(p) + \delta - nk\pi(p)/c^* \right]}{p \left[1 - \frac{1-b}{n\pi(p)} + \epsilon_\pi \right]}$$
 (E.20)

Substituting this expression into (D.13), and using (D.14) and (D.16), an allocation with $x^*=0$ is defined by:

$$\alpha k^{\alpha-1} - n\pi(p) = \frac{\beta \alpha k^{\alpha-1}}{n\pi(p)} \frac{(-\epsilon_{\pi}) \left[(v\pi(p) + \delta)c^* - nk\pi(p) \right]}{p \left[1 - \frac{1-b}{n\pi(p)} + \epsilon_{\pi} \right]} \quad (E.21)$$

$$n\pi(p)p = \beta k^{\alpha} + (1-b)p \tag{E.22}$$

$$k^{\alpha} = (1+\delta)c^* + nk\pi(p) \tag{E.23}$$

From (E.22), we define c^* as a function of k and $\pi(p)$ and then using (E.21) and (E.23), we obtain after some computations:

$$\alpha k^{\alpha-1} \{ (1+\delta)(n\pi(p) - (1-b)) + \epsilon_{\pi} [(v\pi(p) + \delta)(n\pi(p) - (1-b)) + (1+\delta)n\pi(p)] \} = n\pi(p) \{ (1+\delta)(n\pi(p) - (1-b)) + \epsilon_{\pi} [\alpha(v\pi(p) + \delta) + (n\pi(p) - (1-b)) + \alpha(1+\delta)n\pi(p) + (1+\delta)(n\pi(p) - \alpha(1-b))] \} (E.24)$$

Taking into account that $\pi(p)$ is close to 1 and bounded above and below by $\pi(0)$ and $\pi(+\infty)$ respectively, and p is implicitly defined by (E.21) as a function of k, there exists a solution k^* to this equation. Moreover, we have $\alpha k^{\alpha-1} > n\pi(p)$ if the term into brackets on the left-hand side is lower than the one on the right-hand side. We deduce that $\alpha k^{\alpha-1} > n\pi(p)$ if and only if

$$\frac{v\pi(p) + \delta}{1 + \delta} > \frac{\alpha}{1 - \alpha}$$

Then, in this case where $x^* = 0$ and using (D.13), we deduce that $\mu^* > 0$. Let us now consider that $x^* > 0$. Using (D.12), (D.14), (D.18) and (D.19), we get:

$$(1+x)(1+x(1-a))\pi'(p)\xi = \pi'(p)\frac{1-\pi(p)}{c^*}[1+2x+(1-a)x^2] + a(1-\pi(p))\mu\left(1-\frac{1-b}{n\Gamma(x,p)}\right)$$
(E.25)

If $x^* > 0$, we have $\xi^* = 0$. We immediately deduce that $\mu^* > 0$.

F Proof of Proposition 3

To demonstrate Proposition 3, we prove that the inequality $\xi > 0$ cannot be satisfied if v is sufficiently large. Let us consider equation (E.25) with x = 0. We have $\xi > 0$ if and only if:

$$\epsilon_{\pi} < -a \frac{\alpha k^{\alpha - 1} - n\pi(p)k}{\alpha \pi(p)}$$
 (F.26)

Using (E.24), we get:

$$\alpha k^{\alpha - 1} - n\pi(p) = \frac{n\pi(p)[n\pi(p) - (1 - b)]\epsilon_{\pi}}{\mathcal{A}(p)} [\alpha(1 + \delta) - (1 - \alpha)(v\pi(p) + \delta)]$$
(F.27)

with

$$\mathcal{A}(p) \equiv (1+\delta)(n\pi(p)-(1-b)) + \epsilon_{\pi}[(v\pi(p)+\delta)(n\pi(p)-(1-b)) + (1+\delta)n\pi(p)]$$

Substituting (F.27) into (F.26), we obtain:

$$1 > \frac{ak}{\alpha\pi(p)}n\pi(p)[n\pi(p) - (1-b)]\frac{(1-\alpha)(v\pi(p)+\delta) - \alpha(1+\delta)}{\mathcal{A}(p)}$$
$$> \frac{ak}{\alpha\pi(p)}n\pi(p)\frac{(1-\alpha)(v\pi(p)+\delta) - \alpha(1+\delta)}{1+\delta}$$
(F.28)

Note that because ϵ_{π} is close to 0, k is lower but close to $[n\pi(p)/\alpha]^{\frac{1}{\alpha-1}}$. Recall also that $\pi(p)$ belongs to $(\pi(+\infty), \pi(0)]$. Therefore, if v is sufficiently large, inequality (F.28) is violated, which concludes the proof.

G Proof of Proposition 4

Using (30), the system of equations (D.16), (31), (32) with $x^* > 0$ and $\xi^* = 0$, satisfied by such an allocation, can be written:

$$\alpha k^{\alpha - 1} - n\Gamma(x, p) = \mu c^* \frac{\beta \alpha k^{\alpha - 1}}{n\Gamma(x, p)}$$
 (G.29)

$$p[n\Gamma(x,p) - (1-b)] = \beta k^{\alpha}$$
 (G.30)

$$\frac{\delta + \mu p}{\Gamma(x, p)} - \frac{nk}{c^*} + v = \frac{(1+x)^2}{ac^*}$$
 (G.31)

$$a\mu c^* \left(1 - \frac{1-b}{n\Gamma(x,p)}\right) = -\pi'(p)[1 + 2x + (1-a)x^2]$$
 (G.32)

$$(1+\delta)c^* + (1-\pi(p))x + n\Gamma(x,p)k = k^{\alpha}$$
 (G.33)

Equations (G.29) and (G.33) are equivalent to:

$$c^* = \frac{1}{1+\delta} [k^{\alpha} - (1-\pi(p))x - n\Gamma(x,p)k]$$
 (G.34)

$$\mu = \frac{n\Gamma(x,p)}{c^*\beta\alpha k^{\alpha-1}} [\alpha k^{\alpha-1} - n\Gamma(x,p)]$$
 (G.35)

Using these two equations and (G.30), equation (G.31) becomes:

$$\frac{nk}{\alpha[n\Gamma(x,p) - (1-b)]} [\alpha k^{\alpha-1} - n\Gamma(x,p)] - nk
+ \frac{\delta/\Gamma(x,p) + v}{1+\delta} [k^{\alpha} - (1-\pi(p))x - n\Gamma(x,p)k] = \frac{(1+x)^2}{a}$$
(G.36)

Now, substituting (G.35) in (G.32), we get:

$$\frac{a}{\beta\alpha}[\alpha - n\Gamma(x, p)k^{1-\alpha}][n\Gamma(x, p) - (1-b)] = -\pi'(p)[1 + 2x + x^2(1-a)]$$
 (G.37)

An optimal allocation is a solution (x^*, k^*, p^*) to the system (G.30), (G.36) and (G.37).

As a preliminary result, we note that Assumption 1 implies that $n\Gamma(x, p)$ – $(1-b)+np\Gamma_p(x,p)>0$. Differentiating the left-hand side of (G.30) with

respect to p, we deduce that it is strictly increasing in p. This means that (G.30) implicitly defines p as a function of k and x, i.e. $p \equiv p(k, x)$, with⁸:

$$p_x \equiv \frac{dp}{dx} = -\frac{pn\Gamma_x}{n\Gamma - (1-b) + np\Gamma_p} < 0$$
 (G.38)

Substituting p = p(k, x) into (G.36)-(G.37), an optimal allocation is a solution (x, k) solving this last system of two equations. Let us consider (G.37). It can be written:

$$G(x,k) \equiv \frac{a}{\beta\alpha} [\alpha - n\Gamma k^{1-\alpha}] [n\Gamma - (1-b)] + \pi'(p) [1 + 2x + x^2(1-a)] = 0$$
 (G.39)

with p = p(k, x). It implicitly defines x as a function of k, i.e. x = x(k), if $G_x \equiv \partial G/\partial x \neq 0$. Differentiating (G.39) with respect to x, we obtain:

$$G_x = \frac{an}{\beta\alpha} (\Gamma_x + \Gamma_p p_x) [-(n\Gamma - (1-b))k^{1-\alpha} + \alpha - n\Gamma k^{1-\alpha}]$$

+\pi'(p)p_x[1 + 2x + x^2(1-a)] + \pi'(p)[2 + 2x(1-a)] (G.40)

Using (G.38), we have:

$$\Gamma_x + \Gamma_p p_x = \frac{\Gamma_x (n\Gamma - (1-b))}{n\Gamma - (1-b) + np\Gamma_p}$$
 (G.41)

Using (G.39) to substitute $\alpha - n\Gamma k^{1-\alpha}$ and using (G.41), we deduce that $G_x < \pi'(p)\mathcal{B}$, with:

$$\mathcal{B} \equiv 2 + 2x(1-a) - (1-\pi) \frac{an[1 + 2x + x^2(1-a)]}{(1+x)^2[n\Gamma - (1-b) + np\Gamma_p]}$$
 (G.42)

Since $\pi(p) \in (\pi(+\infty), \pi(0)]$ is close to 1, the expression \mathcal{B} is strictly positive. Therefore, $G_x < 0$, meaning that (G.39) implicitly defines x = x(k).

Hence, an optimal allocation is a solution k to equation (G.36), with x=x(k) and $p=p(k,x(k))\equiv p(k)$. Of course, k>0. Since $\mu>0$, we also have $\alpha k^{\alpha-1}>n\Gamma(x(k),p(k))$, where $\Gamma(x(k),p(k))\geqslant \pi(p(k))>\pi(+\infty)$. Therefore, there exists $\overline{k}>0$ defined by $\alpha \overline{k}^{\alpha-1}=n\Gamma(x(\overline{k}),p(\overline{k}))$ such that $\alpha k^{\alpha-1}>n\Gamma(x,p)$ for all $k<\overline{k}$. Hence, k belongs to $(0,\overline{k})$.

⁸To simplify the notations, we omit the arguments of the functions in this proof.

Let us note LHS(k) the left-hand side and RHS(k) the right-hand side of (G.36), respectively. When k tends to 0, we deduce, using (G.30) and (G.33), that x(k) and p(k) tend to 0 too. We get RHS(0) = 1/a > 0 = LHS(0). Moreover,

$$LHS(\overline{k}) = -n\overline{k} + \frac{\delta/\Gamma(x(\overline{k}), p(\overline{k})) + v}{1 + \delta} [(1 - \alpha)\overline{k}^{\alpha} - (1 - \pi(p(\overline{k})))x(\overline{k})]$$

$$RHS(\overline{k}) = \frac{(1 + x(\overline{k}))^{2}}{a}$$

with
$$(1 - \alpha)\overline{k}^{\alpha} > (1 - \pi(p(\overline{k})))x(\overline{k})$$
.

Since \overline{k} has a bounded value and (G.33) is satisfied, $x(\overline{k})$ is bounded above. This implies that $LHS(\overline{k}) > RHS(\overline{k})$ if v is sufficiently large. Then, there exists a solution $k \in (0, \overline{k})$ to equation (G.36).

H Proof of Proposition 5

Let us consider that $\pi(p) = \pi$ is constant, i.e. $\epsilon_{\pi} = 0$. In this case, the constraint on pollution is no more relevant (the multiplier $\mu = 0$). Since x > 0, the social planner solves:

$$\begin{cases} \max_{c^F, c^I, d^F, d^I, x, k} & \pi(\ln c^F + \delta \ln d^F + v) + (1 - \pi)(\ln c^I + \delta \ln d^I + \frac{ax}{1 + x}v) \\ s. \ to & k^{\alpha} = \pi c^F + (1 - \pi)(c^I + x) + \frac{\pi d^F + (1 - \pi)d^I}{n\Gamma(x)} + nk\Gamma(x) \end{cases}$$

with $\Gamma(x) \equiv \pi + (1-\pi)\frac{ax}{1+x}$. Maximising this objective function is equivalent to maximise:

$$\ln(c^F)^{\pi}(c^I)^{1-\pi} + \delta \ln(d^F)^{\pi}(d^I)^{1-\pi} + (1-\pi)\frac{ax}{1+x}v$$
 (H.43)

This program can be solved in two steps. In a second step, we maximise $\ln C = \ln(c^F)^\pi (c^I)^{1-\pi}$ under the constraint $\pi c^F + (1-\pi)c^I = P^c C$ with respect to c^F and c^I , taking the level of consumption expenditures $P^c C$ as given. We perform the same exercise for $\ln D = \ln(d^F)^\pi (d^I)^{1-\pi}$ under the constraint $\pi d^F + (1-\pi)d^I = P^d D$ with respect to d^F and d^I , taking the level of consumption expenditures $P^d D$ as given. Using the first order conditions, we deduce that $P^c = 1$ and $P^d = 1$.

Therefore, in a first step, we have to solve:

$$\begin{cases} \max_{C,D,x,k} & \ln C + \delta \ln D + (1-\pi) \frac{ax}{1+x} v \\ s. \ to & k^{\alpha} = C + \frac{D}{n\Gamma(x)} + (1-\pi)x + nk\Gamma(x) \end{cases}$$

Note that this program above, using the constraint, can be rewritten $\max_{D,x,k} V$, with:

$$V \equiv \ln\left[k^{\alpha} - \frac{D}{n\Gamma(x)} - (1 - \pi)x - nk\Gamma(x)\right] + \delta \ln D + (1 - \pi)\frac{ax}{1 + x}v \quad (\text{H}.44)$$

where $C = k^{\alpha} - \frac{D}{n\Gamma(x)} - (1-\pi)x - nk\Gamma(x)$. We can then derive the following first order conditions:⁹

$$V_D = -\frac{1}{n\Gamma(x)C} + \frac{\delta}{D} = 0 \tag{H.45}$$

$$V_x = \frac{D\Gamma'(x)/[n\Gamma(x)^2] - (1-\pi) - nk\Gamma'(x)}{C} + \frac{(1-\pi)av}{(1+x)^2} = 0 \quad (H.46)$$

$$V_k = \frac{\alpha k^{\alpha - 1} - n\Gamma(x)}{C} = 0 \tag{H.47}$$

We easily deduce that:

$$D = \delta n \Gamma(x) C \tag{H.48}$$

$$D\Gamma'(x)/[n\Gamma(x)^{2}] - (1-\pi) - nk\Gamma'(x) = -C\frac{(1-\pi)av}{(1+x)^{2}}$$
 (H.49)

$$\alpha k^{\alpha-1} = n\Gamma(x)$$
 (H.50)

Establishing the second order conditions for this last program gives us the second order conditions for the program (H.43). Hence, we differentiate (H.45)-(H.47) and use (H.48)-(H.50), $\Gamma'(x) = (1-\pi)\frac{a}{(1+x)^2}$ and $\Gamma''(x) = -2(1-\pi)\frac{a}{(1+x)^3}$ to compute the following Hessian matrix:

$$H \equiv \left[\begin{array}{ccc} V_{DD} & V_{Dx} & V_{Dk} \\ V_{xD} & V_{xx} & V_{xk} \\ V_{kD} & V_{kx} & V_{kk} \end{array} \right]$$

⁹In the following, we note $V_u \equiv \partial V/\partial u$ and $V_{uv} \equiv \partial^2 V/\partial v \partial u$, with $\{u,v\} = \{D,x,k\}$.

with

$$V_{DD} = -\frac{1+\delta}{n^2\Gamma(x)^2C^2\delta} \tag{H.51}$$

$$V_{Dx} = \frac{\Gamma'(x)}{nC\Gamma(x)^2} [1 - \Gamma(x)v] = V_{xD}$$
 (H.52)

$$V_{Dk} = 0 = V_{kD}$$
 (H.53)

$$V_{xx} = \frac{2}{C(1+x)} [C\Gamma'(x)v - (1-\pi)] - \frac{2\delta\Gamma'(x)^2}{\Gamma(x)^2}$$

$$-\frac{\Gamma'(x)v}{(1+x)^2}[2+\Gamma'(x)v]$$
 (H.54)

$$V_{xk} = -\frac{n\Gamma'(x)}{C} = V_{kx} \tag{H.55}$$

$$V_{kk} = \frac{(\alpha - 1)n\Gamma(x)}{Ck} \tag{H.56}$$

To prove that an optimal allocation is a maximum, we have to show that $\mathcal{H}_1 \equiv V_{DD} < 0$, $\mathcal{H}_2 \equiv V_{DD}V_{xx} - V_{Dx}V_{xD} > 0$ and $\mathcal{H}_3 \equiv detH < 0$.

 $\mathcal{H}_1 < 0$ is obvious. Let us now determine the sign of \mathcal{H}_2 . Using (H.51), (H.52) and (H.54), we get:

$$\mathcal{H}_{2}n^{2}C^{2}\Gamma(x)^{4} = \frac{1+\delta}{\delta}\Gamma(x)^{2} \left[\frac{2(1-\pi)}{C(1+x)} - \frac{2\delta\Gamma'(x)^{2}}{\Gamma(x)^{2}} \right] - \Gamma'(x)^{2}$$

$$+2\Gamma(x)\Gamma'(x) \left[\Gamma'(x) - \frac{x}{(1+x)^{2}} \frac{1+\delta}{\delta}\Gamma(x) \right] v$$

$$+\Gamma(x)^{2}\Gamma'(x)^{2} \left[\frac{1+\delta}{\delta} \frac{1}{(1+x)^{2}} - 1 \right] v^{2}$$
(H.57)

We observe that $\mathcal{H}_2 > 0$ for v sufficiently large if and only if:

$$(1+x)^2 < \frac{(1+\delta)}{\delta} \tag{H.58}$$

Using (G.31), (G.34) and (G.35),

$$(1+x)^2 < ak^{\alpha} \left[\frac{n}{n\Gamma(x) - (1-b)} + \frac{\delta/\Gamma(x) + v}{1+\delta} \right]$$
 (H.59)

Using (H.50) and $\Gamma(x) \ge \pi$, we have $k \le [\alpha/(n\pi)]^{\frac{1}{1-\alpha}}$, meaning that

$$(1+x)^2 < a[\alpha/(n\pi)]^{\frac{\alpha}{1-\alpha}} \left[\frac{n}{n\pi - (1-b)} + \frac{\delta/\pi + v}{1+\delta} \right].$$

We deduce that inequality (H.58) is satisfied if:

$$a[\alpha/(n\pi)]^{\frac{\alpha}{1-\alpha}} \left[\frac{n}{n\pi - (1-b)} + \frac{\delta/\pi + v}{1+\delta} \right] < \frac{(1+\delta)}{\delta}$$
 (H.60)

which is fulfilled if $a < \hat{a}_0$ with $\hat{a}_0 \equiv \frac{(1+\delta)}{\delta} \times \left[\frac{n}{n\pi - (1-b)} + \frac{\delta/\pi + v}{1+\delta}\right]^{-1} \times [n\pi/\alpha]^{\frac{\alpha}{1-\alpha}}$.

Finally, let us investigate the properties of \mathcal{H}_3 . Using (H.53), we have $\mathcal{H}_3 = V_{DD}V_{xx}V_{kk} - V_{xD}^2V_{kk} - V_{xk}^2V_{DD}$. Then, using (H.51), (H.52) and (H.54)-(H.56), we obtain after some computations:

$$\mathcal{H}_{3}nC^{3}k\Gamma(x)^{3} = -(1-\alpha)\Gamma(x)^{2}\frac{1+\delta}{\delta}\left[\frac{2(1-\pi)}{C(1+x)} + \frac{2\delta\Gamma'(x)^{2}}{\Gamma(x)^{2}}\right]$$

$$+(1-\alpha)\Gamma'(x)^{2} + n\frac{1+\delta}{\delta}\Gamma'(x)^{2}\Gamma(x)k$$

$$+2(1-\alpha)\Gamma'(x)\Gamma(x)\left[\Gamma(x)\frac{1+\delta}{\delta}\frac{x}{(1+x)^{2}} - \Gamma'(x)\right]v$$

$$+(1-\alpha)\Gamma'(x)^{2}\Gamma(x)^{2}\left[1 - \frac{1+\delta}{\delta}\frac{1}{(1+x)^{2}}\right]v^{2}$$

We deduce that $\mathcal{H}_3 < 0$ if v is sufficiently large and inequality (H.58) is satisfied. This happens when inequality (H.60) holds, i.e. for $a < \hat{a}_0$.

By a continuity argument, our result still holds if π weakly depends on p, i.e. ϵ_{π} is close to 0. Therefore, there exits $\hat{(}a) > 0$ such that any optimal allocation is a maximum if $a < \hat{a}$, v sufficiently large and ϵ_{π} close to 0. Note also that since this last result holds for any optimal allocation, such an allocation is unique.

Proof of Proposition 6 Ι

Each household maximises the utility (2) under the budget constraints (3)-(4). Solving the households' program, we obtain:

$$\frac{1}{c^F} = \frac{R\delta}{d^F} \tag{I.61}$$

$$\frac{1}{c^I} = \frac{R\delta}{d^I} \tag{I.62}$$

$$\frac{1}{c^{I}} = \frac{R\delta}{d^{I}}$$

$$\frac{av}{(1+x)^{2}} \leqslant \frac{1+\sigma}{c^{I}}$$
(I.62)

Recall that the optimal allocation is characterised by a positive investment in health care, $x^* > 0$. Hence, the instruments will be chosen such that (I.63) holds as an equality. These first order conditions above and the budget constraints allow us to derive the stationary levels of consumption and saving for both types of household:

$$c^{F} = \frac{1}{1+\delta} \left(w + T^{F} - \frac{\theta}{R} \right) \tag{I.64}$$

$$d^{F} = \frac{R\delta}{1+\delta} \left(w + T^{F} - \frac{\theta}{R} \right) \tag{I.65}$$

$$s^{F} = \frac{\delta}{1+\delta} \left(w + T^{F} \right) + \frac{\theta}{R(1+\delta)}$$
 (I.66)

and

$$c^{I} = \frac{1}{1+\delta} \left[w + T^{I} - \frac{\theta}{R} - (1+\sigma)x \right]$$
 (I.67)

$$d^{I} = \frac{R\delta}{1+\delta} \left[w + T^{I} - \frac{\theta}{R} - (1+\sigma)x \right]$$
 (I.68)

$$s^{I} = \frac{\delta}{1+\delta} \left[w + T^{I} - (1+\sigma)x \right] + \frac{\theta}{R(1+\delta)}$$
 (I.69)

We can also express the chosen level of health expenditure:

$$(1+x)^2 = \frac{av}{(1+\sigma)\delta} \left(s^I - \frac{\theta}{R} \right) \tag{I.70}$$

Finally, the government that perceives the different taxes balances its budget at each period of time. Taking into account the population size, this means that 10 :

$$\frac{\theta}{n\Gamma} + \sigma(1-\pi)x + \frac{\rho\alpha k^{\alpha}}{n\Gamma} = \pi T^{F} + (1-\pi)T^{I}$$
 (I.71)

Using the previous section, we recall that an optimal allocation is characterised by equations (30), (G.29), (G.30), (G.31), (G.32) and (G.33).

We are now able to derive the appropriate policy design that allows for decentralising the stationary optimal allocation. Using (I.64), (I.65), (I.67) and (I.68), the condition (30) is, partly, satisfied for:

$$T^{F} = T^{I} - (1 + \sigma)x^{*} \tag{I.72}$$

Obviously, we can set T^F to zero and thus, $T^I = (1 + \sigma)x^*$. Then, the heterogeneity in consumption among the two types of household is eliminated.

Substituting (30) into (G.29) and comparing with (I.61) and (I.62), we should have that $R = (1 - \rho)\alpha k^{\alpha - 1} = \alpha k^{\alpha - 1}(1 - \mu c^* \frac{\beta}{n\Gamma})$, i.e.

$$\rho = \frac{\mu^* c^* \beta}{n \Gamma^*} = 1 - \frac{n \Gamma^*}{\alpha (k^*)^{\alpha - 1}} \in (0, 1)$$
 (I.73)

Using now (I.63) and (G.31), we obtain:

$$\frac{\sigma}{1+\sigma} = \frac{1}{v} \left(\frac{nk^*}{c^*} - \frac{\delta + \mu^* p^*}{\Gamma^*} \right) \tag{I.74}$$

where μ^* is given by (G.32).

It is straightforward that $\sigma > -1$. To go further and determine the sign of σ , let us consider equation (G.31). Using the proof of Proposition 4, we have that $1 + (x^*)^2 < (1+\delta)/\delta$, which means that $x^* < \sqrt{(1+\delta)/\delta} - 1 \equiv \overline{x}$. Using (G.29), equation (G.32) implies $c^* > [(1-\alpha)(k^*)^{\alpha} - (1-\pi^*)x^*]/(1+\delta)$. Since we know that k^* has a finite and strictly positive value and $x^* < \overline{x}$, this means that c^* is bounded below by a strictly positive value \underline{c} , i.e. $c^* > \underline{c}$. We deduce that the right-hand side of (G.31) is bounded above by $(1+\overline{x})^2/(a\underline{c})$. Therefore, v large enough implies:

$$\frac{nk^*}{c^*} - \frac{\delta + \mu^* p^*}{\Gamma^*} > 0 \Leftrightarrow \sigma > 0 \tag{I.75}$$

¹⁰When this is not a source of confusion, we skip the arguments of the functions.

Note that this last inequality requires that $nk^*\Gamma^* > \delta c^*$. Using (I.61), (I.63) and the optimality condition (30), we deduce that $s^F = s^I = s^*$. Hence, the equilibrium on the capital market writes $nk^*\Gamma^* = s^*$. We deduce that $s^* > \delta c^*$. Using (I.64) and (I.66), we obtain that $\theta > 0$.