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Mortality, Family and Lifestyles*

Gregory Ponthiere†

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Abstract

While there is a large empirical literature on the intergenerational transmission of health and survival outcomes in relation to lifestyles, little theoretical work exists on the long-run prevalence of (un)healthy lifestyles induced by mortality patterns. To examine that issue, this paper develops an overlapping generations model where a healthy lifestyle and an unhealthy lifestyle are transmitted vertically or obliquely across generations. It is shown that there must exist a locally stable heterogeneous equilibrium involving a majority of healthy agents, as a result of the larger parental gains from socialization efforts under a higher life expectancy. We also examine the robustness of our results to the introduction of parental altruistic concerns for children's health and of asymmetric socialization costs.

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1 Introduction

Recent empirical studies support the existence of an intergenerational transmission of health and survival outcomes. In his survey, Ahlburg (1998) underlines the large correlation between parents' health and children's health.[1] As shown by the study of Yashin and Iachine (1997) surveyed in Ahlburg, there is a correlation of about 0.15 to 0.30 between the lifespans of parents and the ones of their children. More recently, Case et al. (2002) find, on the basis of U.S. data, large effects of parental health on children's health: if a child aged 0-3 has a mother in a very good or excellent health, the chances for that child of being also in a very good or excellent health are 27 % larger *ceteris paribus*. Finally, Coneus and Spieb (2008) confirm the existence of an intergenerational transmission of health in early childhood. Other recent studies show also the familial transmission of characteristics directly related to health outcomes.[2]

The high correlation between parental health and children's health (or survival) reflects both the *genetic* component of health and longevity, and, also, the familial *environment* in which children live, which is common to parents and children. Note that it is not trivial to identify the contributions of genetic and environmental determinants of health and survival.[3] Regarding longevity determinants, most studies addressing that issue focused on the correlation of lifespans between identical twins (i.e. monozygotic) and fraternal twins (i.e. dizygotic). Yashin and Iachine (1997) point to larger lifespan correlations for identical twins than for fraternal twins (0.3 against 0.2), which highlights the crucial role of genetic background as a factor explaining longevity.[4] However, even if the genetic component of the intergenerational transmission of health is significant, genes alone cannot suffice to explain longevity outcomes as a whole: as stated by Christensen et al. (2006), the genetic background of individuals accounts for only one quarter to one third of intracohort longevity inequalities. Thus, the social environment, and in particular the familial environment, remain important determinants of the intergenerational correlation of longevity.

How can one explain the contribution of the familial environment to the intergenerational correlation of health and survival? One obvious channel concerns the interfamily transmission of *lifestyles*, which, as this is well-known among demographers, play a significant role in the determination of health and longevity.[5] Lifestyles, defined as general ways of living, consist of various dimensions affecting survival prospects, including, among others, eating behaviour, alcoholism, smoking, and physical activity.[6] To give a concrete idea of the size of the impact of lifestyles on mortality, we can refer to the longitudinal study

[1] Ahlburg regards this intergenerational correlation of health as a major determinant, jointly with the correlation of familial education levels, of intergenerational correlations of income.

[2] See Currie and Moretti (2005) on the intergenerational transmission of birth weight, and Kebede (2003) on the transmission of height.

[3] Actually, as shown by Christensen et al. (2006), the identification of the genetic and non-genetic determinants of longevity is a real challenge, since this presupposes the absence of unobserved heterogeneity. However, many genetic and non-genetic longevity factors are not reported in the data, simply because these are not yet known to be important.

[4] Those results are confirmed by Herskind et al.'s (1996) study of Danish twins.

[5] However, it should be stressed that lifestyles are not the only determinants of longevity: there are also genetic factors (Christensen et al. 2006), and environmental ones (Kinney and Oskanyak 1991), both of these being not related at all with lifestyles.

[6] On those various behavioural determinants of longevity, see Doll and Hill (1950), Kaplan et al. (1987), Peto et al. (1992), Solomon and Manson (1997), and Bender et al. (1998).

carried out by Kaplan et al. (1987) in California. That study highlighted that individuals above age 60 who were smoking in 1965 faced, during the next 17 years, an overall mortality risk that is 1.40 times the mortality risk of those who never smoked. A mortality risk differential of 1.38 was estimated for agents who have little leisure-time physical activity in 1965. Moreover, alcoholic consumption and overweight are shown to an overmortality of about 1.20. That strong influence of lifestyles on health and survival outcomes is also confirmed by recent microeconometric studies taking unobserved heterogeneity into account, including Contoyannis and Jones (2004) and Balia and Jones (2008).

Given the large intergenerational correlations in health and the crucial role played by lifestyles, it does not come as a surprise that empirical evidence confirms the existence of a transmission of unhealthy lifestyles. If we take, for instance, the case of smoking, Jones (1994) showed that parental smoking increases the probability of becoming a smoker, and reduces the age of starting. This supports the idea that the family affects lifestyle choices. Moreover, Goode et al. (2008) show that, among low income households, there is a statistically significant intergenerational transmission of unhealthy eating habits.

Note, however, that many factors affect the adherence to a more or less healthy lifestyle, and the familial environment is not the unique one. Regarding cigarettes consumption, the membership to a high socioeconomic group and the education level tend, according to Jones (1994), to reduce the probability to start smoking and the duration of smoking.[7] Moreover, Hersch (2000) highlighted that the demand for cigarettes is decreasing in the education level (especially for high income persons), and depends on the employment status and on the presence of children.[8] Having stressed that point, it remains that the family plays a significant role in the transmission of (un)healthy lifestyles to children, and, hence, in the transmission of health and survival outcomes.

All in all, the large empirical evidence supporting a strong intergenerational transmission of lifestyles, if taken jointly with the significant influence of lifestyles on longevity, allows us to explain, at least in part, the stylized facts we started from: the observed correlation between parental health and children's health (and survival). However, those empirical studies do not bring us an answer to a particular question, which concerns the *evolution* of lifestyles and mortality in the long-run. True, lifestyles affect mortality, but it is also the case that mortality influences lifestyles, in the sense that ways of life are the outcome of a natural selection process induced by mortality. Hence, while a change in mortality must follow from a change in lifestyles, the opposite is also true: a change in mortality is also affecting the prevailing lifestyles. In the light of that two-directional relationship, a natural question arises: what will be the long-run equilibrium prevalence of unhealthy lifestyles? For instance, does the mere fact that smoking reduces longevity imply that smoking will disappear in the long-run? Under which conditions will unhealthy lifestyles survive?

An answer to that question requires a theoretical study of the transmission of healthy (and unhealthy) lifestyles across generations. The pioneer models of intergenerational transmission of cultural traits and lifestyles are the ones developed by Bisin and Verdier (2000, 2001). In those frameworks, cultural

[7] The impact of the socio-economic status on smoking is confirmed by Chen et al. (2007).

[8] Among other factors, the financial conditions in which agents are seem to be related with their unhealthy lifestyles. As shown by Grafova (2007), there is a correlation between holding a non-collateralized debt (NCD) and smoking.

traits (e.g. preference parameters) can be transmitted vertically (from parents to children) or obliquely (from a role model to children), and parents can, through socialization efforts, raise the probability that their children will acquire their cultural trait. Note, however, that, while a large theoretical literature exists on the transmission of traits across generations, there has been little work so far on the specific transmission of (un)healthy lifestyles. Actually, following the pioneer contribution of Bisin and Verdier, models of cultural transmissions were applied to various issues, such as religions and marriage (Bisin et al. 2004), globalization and trade (Olivier et al. 2008), work organizations (Hiller 2010), unemployment (Michau 2009), and the fertility transition (Baudin 2010).

As far as we know, the unique attempt to modelize the effect of lifestyles transmissions on mortality is the paper by Ponthiere (2010), which consists of a three-period overlapping generations (OLG) model with a socialization process following Bisin and Verdier (2001), and where the length of life depends on the lifestyle adopted by agents when being young adults. However, that paper focused on only one aspect of lifestyle, the labour supply, and was mainly concerned with the question of the optimal taxation of labour income in a society of agents who internalize the consequences of overwork imperfectly. As such, it could only provide a partial analysis of the dynamics of lifestyles and longevity in a heterogeneous society, as well as of the role played by families in that process.

The goal of this paper is to re-examine the intergenerational transmission of healthy lifestyles, by generalizing the previous paper by Ponthiere (2010), in such a way as to better identify the role played by the family in the dynamics of health and longevity outcomes. For that purpose, we develop a three-period OLG model where agents face a probability of survival to the old age, which depends on the lifestyle adopted during childhood. For simplicity, the lifestyle coincides here either with a healthy lifestyle, yielding a higher survival probability to the old age, or an unhealthy lifestyle, leading to a lower life expectancy. In comparison with Ponthiere (2010), which relies on continuous longevity functions, this discrete longevity model is simpler, and allows us to focus more on the role of the family in the socialization process. To do this, two extensions of the model are proposed. First, whereas parents want, in Ponthiere (2010), their children to have the same way of life as themselves, the present model will, in addition, introduce *parental altruism*, to account for the fact that parents may care also about the health prospects of their children, and may adjust their socialization efforts accordingly. Second, we expand also Ponthiere (2010) by introducing *asymmetric socialization costs*, to account for the fact that it may be more painful, for parents, to transmit a healthy lifestyle rather than an unhealthy one. As we shall see, parental altruism and asymmetric socialization costs affect the long-run dynamics of heterogeneity significantly, and, in particular, the issue of the long-run persistence of unhealthy lifestyles.

The paper is organized as follows. Section 2 presents the basic model. The long-run dynamics of lifestyles transmission is studied in Section 3. Parental altruism is introduced in Section 4. Section 5 examines the impact of asymmetric socialization costs across families. Section 6 concludes.

2 The basic model

2.1 Environment

Let us consider a three-period overlapping generations model. Each cohort is a continuum of agents whose size is constant over time, and normalized to unity.

Life is divided in three periods, whose length is normalized to unity. During period 1 (childhood), agents are all identical, and do not belong to any group. Children do not make any decision, but are subject to the socialization process, which will affect their type during the rest of their life.

During period 2 (young adulthood), the population becomes divided in two groups of agents. Those two groups differ in a single aspect: the more or less healthy lifestyle to which they adhere. The population will thus be divided in two groups: on the one hand, the healthy population (i.e. type H); on the other hand, the unhealthy population (i.e. type U). Moreover, the variable q_t will denote the proportion of type H in the cohort who is adult at time t. Each young adult makes one child, and invests in the socialization of his child.

Finally, not all agents will reach the old age. Only a fraction π^i of the population of type $i \in \{H, U\}$ will reach the old age. Following the large empirical evidence on the effects of lifestyles on longevity, we have

$$\pi^H > \pi^U \tag{1}$$

Group-specific life expectancy at birth is $2+\pi^i$, for $i \in \{H, U\}$. Life expectancy for the whole cohort is $q_t\left(2+\pi^H\right)+(1-q_t)(2+\pi^U) = 2+q_t\pi^H+(1-q_t)\pi^U$.

2.2 Socialization

The population follows an adaptation and imitation process of the type modelled by Bisin and Verdier (2001). The transmission of the cultural trait $i \in \{H, U\}$, which consists here of the parameter π^i reflecting the healthy or unhealthy lifestyle, is modelled as a mechanism where socialization *inside* the family and socialization *outside* the family interact. The first type of socialization is called the vertical transmission (from parents to children), whereas the second type is called the oblique transmission (from a "role model" in the society to the child).[9]

Families are composed of one parent and one child. Children are born at time t without any cultural trait $i \in \{H, U\}$. Direct vertical socialization to the parent's trait $i \in \{H, U\}$ occurs with a probability ρ^i_{t+1}. If the direct vertical socialization does not take place, which happens with a probability $1-\rho^i_{t+1}$, the child then picks up the trait of a model chosen randomly in the population of reference, which is the population of young adults. Thus, the child will take the trait H with a probability q_t, and the trait U with a probability $1-q_t$.

[9] At this stage, it should be noted that it is extremely difficult to provide clear empirical evidence supporting one type of socialization process or another: only intergenerational correlations can be observed (see Section 1), and these may hide various, complex mechanisms, which are more or less voluntary. The Bisin-Verdier modelling captures some important aspects of the socialization process, but could hardly account for all of them. That model allows for some form of voluntary socialization, through the choice of socialization efforts by parents, but, at the same time, there can also be an oblique socialization *via* a role model, which is, by nature, not voluntary (since the role model does nothing to be imitated).

Hence, if p_{t+1}^{HH} and p_{t+1}^{HU} (resp. p_{t+1}^{UU} and p_{t+1}^{UH}) denote the probabilities that a child born at t in a family with trait H (resp. U) is socialized to, respectively, trait H and trait U (resp. U and H), the transition probabilities are:

$$\begin{array}{ll} p_{t+1}^{HH} = \rho_{t+1}^{H} + \left(1 - \rho_{t+1}^{H}\right) q_t & p_{t+1}^{HU} = \left(1 - \rho_{t+1}^{H}\right)(1 - q_t) \\ p_{t+1}^{UU} = \rho_{t+1}^{U} + \left(1 - \rho_{t+1}^{U}\right)(1 - q_t) & p_{t+1}^{UH} = \left(1 - \rho_{t+1}^{U}\right)(q_t) \end{array} \tag{2}$$

By the Law of Large Numbers, p_{t+1}^{ij} is also equal to the proportion of children whose parents are of type i who have the cultural trait j. Hence, the proportion q_{t+1} of agents born at time t who become of type H follows the dynamic law:

$$q_{t+1} = \left[\rho_{t+1}^{H} + \left(1 - \rho_{t+1}^{H}\right) q_t\right] q_t + \left[\left(1 - \rho_{t+1}^{U}\right) q_t\right](1 - q_t) \tag{3}$$

The first term is the probability to be socialized to trait H when having a family of type H, multiplied by the probability to belong to a family of type H. The second term is the probability to acquire trait H when being born in a family of type U, multiplied by the probability to belong to a family of type U.

Following Bisin and Verdier (2001), we assume that parents of type $i \in \{H, U\}$ can socialize their children born at time t vertically, by educating them through a (purely physical) socialization effort e_t^i ($0 \leq e_t^i \leq 1$). The socialization effort e_t^i is an input in the cultural production of their children as adults: $\rho_{t+1}^i = \rho(e_t^i)$. A welfare loss $C(e_t^i)$ is generated by a socialization effort e_t^i. For simplicity, the disutility from socialization efforts takes a quadratic form:

$$C(e_t^i) = \delta \frac{\left(e_t^i\right)^2}{2} \tag{4}$$

where δ accounts for the disutility of socialization efforts ($\delta > 0$).

Parents have a welfare gain from coexisting with children with the *same* type as themselves. The welfare derived by a parent of type i born at $t-1$ when he coexists with a child of type i, denoted by φ_{t+1}^{ii}, exceeds the welfare derived by a parent of type i when he coexists with a child of type $j \neq i$, denoted φ_{t+1}^{ij}. Welfare gains from having children of one's own type are independent from one's type and from time: $\varphi_{t+1}^{HH} = \varphi_{t+1}^{UU} = \hat{\varphi}$ and $\varphi_{t+1}^{HU} = \varphi_{t+1}^{UH} = \tilde{\varphi}$, with $\hat{\varphi} > \tilde{\varphi}$.

Parents, when choosing e_t^i, weight the cost of socialization - $C(e_t^i)$ - against its expected gains, which depend on the influence of their effort e_t^i on probabilities p_{t+1}^{ii} and p_{t+1}^{ij}, determined by the relation $\rho_{t+1}^i = \rho(e_t^i)$. Note that, even if parents may look here quite selfish, as they socialize their child in such a way as to make these *like them*, whatever the effects it has on the future health of their children, parents believe that their lifestyle is the best, and just want their children to benefit from the same, "best", lifestyle. Thus parents, even though they do not necessarily choose what is the best for their children, choose the best for their children from their *own* perspective.[10]

While there exist various ways to model the relation $\rho_{t+1}^i = \rho(e_t^i)$, we will assume that the probability of direct vertical socialization to trait i ρ_{t+1}^i equals parent's socialization effort e_t^i, in conformity with what Bisin and Verdier (2001) call the "*It's the family*" transmission technology:

$$\rho_{t+1}^i = e_t^i \tag{5}$$

[10] Thus, one should be cautious before rejecting the idea that unhealthy parents invest in the promotion of the unhealthy lifestyle. For type-U parents, the unhealthy lifestyle is, above all, *their* lifestyle, i.e. the one they adopted themselves.

That technology has the virtue of analytical tractability. However, simplicity has also its drawbacks: a major limitation of that transmission technology is that it makes the probability of direct vertical socialization to the parent's trait independent from the level of q_t. That property is a strong simplification, as it may be the case that even the efficiency of parental socialization efforts depend on the current composition of the population (see Bisin and Verdier 2001). However, and for the sake of simplicity, we shall keep that simple technology here, and leave the exploration of alternative technologies for future research.[11]

2.3 Agents's decision

For simplicity, agents have preferences that can be represented by a function that has the expected utility form, and is additive over time. The expected lifetime utility of an agent of type i is:[12]

$$U^i = -\delta \frac{\left(e^i\right)^2}{2} + \beta \pi^i \left[p^{ii}_{t+1} \hat{\varphi} + p^{ij}_{t+1} \tilde{\varphi} \right] \tag{6}$$

where β is a time preference factor. Socialization efforts are made at young adulthood, whereas the gains from coexisting with a child of one's type occur at old adulthood. This is why welfare gains from coexisting with one's child are weighted by the factor β and by the type-specific survival probability π^i.

Assuming an interior solution, the optimal socialization effort is given by:

$$e^i_t = \frac{\beta \pi^i (1 - q^i) \left(\hat{\varphi} - \tilde{\varphi} \right)}{\delta} \tag{7}$$

where q^i is the proportion of agents of type i.[13] Note that the model exhibits here what Bisin and Verdier (2001) call the "cultural substitution" property: a higher proportion of agents of type i in the population of reference makes parents of type i choose a *lower* socialization effort. Thus, parents who belong to a larger group tend to rely more on the society for the socialization of their children, and thus make fewer socialization efforts *ceteris paribus*. Hence there is a "substitution" at work here: parents use socialization by means of the society *instead* of socialization by means of private efforts (the former being cheaper, and, given the level of q^i, sufficiently effective).[14]

Note also that the optimal socialization effort level depends positively on the life expectancy π^i. The total welfare gain from coexisting with a child is determined by the lifetime horizon, so that a higher life expectancy yields a higher socialization effort. As a consequence of that "lifetime horizon effect", type-H agents, who have higher longevity prospects, tend also to invest more in

[11] It should be stressed, however, that this transmission technology is far from neutral for the long-run dynamics of lifestyles transmission. See Bisin and Verdier (2001) on the study of various transmission technologies.

[12] Agents are here good at anticipating their life expectancy, in conformity with Hamermesh (1985).

[13] To have $0 < e^i_t \leq 1$, we impose: $\frac{\beta \pi^i (\hat{\varphi} - \tilde{\varphi})}{\delta} \leq 1$.

[14] Note that this property follows from the particular transmission technology (and cost functions) assumed, but would not necessarily prevail under other technologies (see Bisin and Verdier 2001). The cultural substitution property is also far from neutral for long-run dynamics of cultural transmission. Under transmission technologies allowing for cultural complementarity, there could exist stable equilibria without heterogeneity, unlike here.

socialization than type-U agents. Hence the healthy lifestyle has some form of evolutionary advantage, in the sense that the agents who adopt it tend, *ceteris paribus*, to invest more in the socialization of their own children, which leads to a higher proportion of that type of agents in the long-run population.

3 Long-run dynamics

In the economy under study, the partition of the population q_t determines all other variables: socialization efforts and longevity outcomes. Hence, the constancy of q_t over time brings the constancy of all other variables.

Therefore, examining the existence of a stationary equilibrium (i.e. an equilibrium with constant population composition) amounts to studying whether there exist some values of q_t that can be maintained constant over time. Substituting for $\rho_{t+1}^H = e_t^H$ and $\rho_{t+1}^U = e_t^U$ in the expression

$$q_{t+1} = \left[\rho_{t+1}^H + \left(1 - \rho_{t+1}^H\right) q_t\right] q_t + \left[\left(1 - \rho_{t+1}^U\right) q_t\right] (1 - q_t)$$

yields the following transition function $q_{t+1} \equiv G(q_t)$:

$$q_{t+1} \equiv G(q_t) = q_t + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\left[\pi^H q_t (1 - q_t)^2 - \pi^U q_t^2 (1 - q_t)\right]$$

Thus, looking for a stationary equilibrium amounts to find a fixed point for $G(q_t)$, that is, a level of q_t such that $G(q_t) = q_t$. As it is shown in the Appendix, there exist three stationary equilibria in the economy under study. Two equilibria involve a perfect homogeneity of the population, whereas one equilibrium involves some heterogeneity. Proposition 1 summarizes our results.

Proposition 1 *There exist three stationary equilibria:*

$$q^1 = 0; \;\; q^2 = \frac{\pi^H}{\pi^H + \pi^U} > \frac{1}{2}; \;\; q^3 = 1$$

Only q^2 is locally stable, whereas q^1 and q^3 are unstable.

Proof. See the Appendix. ■

Thus, there exist three stationary equilibria: one in which no one has a healthy lifestyle, as there exist only agents of type U (i.e. $q = 0$); one in which everyone follows a healthy lifestyle, as there exist only agents of type H (i.e. $q = 1$). Finally, there is also an equilibrium with a mixed population. Note that the level of that equilibrium depends only on the longevity differential between agents of types H and U. Given that the healthy agents have a higher life expectancy (i.e. $\pi^H > \pi^U$), the intermediate equilibrium involves necessarily a majority of healthy people. Thus, under $0 < q_0 < 1$, the model predicts that the long-run population must involve a majority of healthy people, on the grounds that a healthy lifestyle, by leading to better survival prospects, implies also a higher socialization effort *ceteris paribus*, which explains why the healthy lifestyle finally dominates the unhealthy lifestyle at the stationary equilibrium. Hence, in this basic model, the healthy lifestyle H exhibits an evolutionary advantage, and this has nothing to do with parental benevolence: on the contrary, the evolutionary advantage of the healthy lifestyle is uniquely

due to the difference in life expectancy between the two lifestyles, which affects how much egoistic parents invest in the socialization of their children.

Whereas a formal proof of Proposition 1 is provided in the Appendix, we can here give some graphical intuition behind that result. For that purpose, Figure 1 represents the transition function $G(q_t)$ in the (q_t, q_{t+1}) space.[15] Clearly, the transition function goes through 0 and 1, as a perfectly homogeneous society can only reproduce itself over time in the present framework. Moreover, the transition function is above the 45° line in the neighbourhood of 0, and below the 45° line around 1. As a consequence, $G(q_t)$ must necessarily intersect the 45° line somewhere for $0 < q_t < 1$. From the argument discussed above we know also that this intermediate equilibrium must involve a majority of healthy agents.

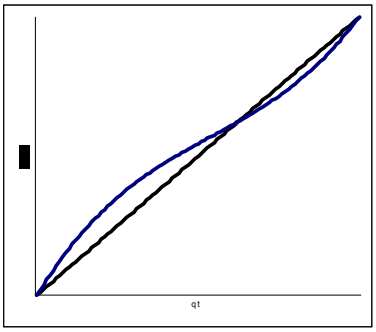

Figure 1: Multiplicity of equilibria

Regarding the stability of equilibria, there is, here again, a formal discussion in the Appendix. However, we can use Figure 1 to give some geometrical intuitions. The two extreme equilibria are unstable, as the transition function has a slope higher than unity at those equilibria, so that small departures from the equilibria lead to large changes in the composition of the population. However, the intermediate equilibrium is locally stable, as the transition function intersects the 45° line at a place where its slope is smaller than unity. Hence as long as the initial population is heterogeneous (i.e. $0 < q_0 < 1$), the economy will converge towards that equilibrium.

Finally, a few words should be added here regarding the dynamics of aggregate life expectancy, equal here to $2+q_t\pi^H+(1-q_t)\pi^U$. Clearly, if the economy starts from a situation where the healthy lifestyle is dominated by the unhealthy lifestyle (i.e. $0 < q_0 < 1/2$), it follows from Proposition 1 that, over time, the proportion of healthy agents will go up (since the equilibrium $q > 1/2$), which will have the effect of raising the aggregate life expectancy. Thus this model describes the secular rise of life expectancy as caused by a rise in the proportion of individuals adhering to a healthy lifestyle (or, equivalently, by a decline of the proportion of the population having an unhealthy lifestyle). Once the long-run equilibrium is reached, the life expectancy stabilizes, but still hides significant inequalities between the two subgroups.

[15] Figure 1 relies on the following parameters values: $\beta = 0.6$; $\delta = 0.5$; $\hat{\varphi}-\tilde{\varphi} = 1.5$; $\pi^H = 0.5$ and $\pi^U = 0.3$.

Regarding the long-run level of aggregate life expectancy, it should also be noted that, quite paradoxically, an exogenous rise in the survival probability π^U thanks, for instance, to an exogenous medical technology shock, will not necessarily raise the long-run aggregate life expectancy. Indeed, a rise in π^U has also the effect to reduce the long-run proportion of healthy agents in the population, so that a rise in π^U may reduce - rather than raise - the aggregate life expectancy.[16] Hence a positive exogenous medical shock reducing the dangerousness of some lifestyles can have a negative effect on long-run aggregate life expectancy, since this modifies the whole evolutionary dynamics of lifestyles in the economy, by favouring the expansion of the unhealthy lifestyle.

4 A model with parental altruism

While the basic model developed in the previous sections has the virtue of simplicity, and can easily explain the improvement of survival prospects over time thanks to the spreading of the healthy lifestyle, it relies, however, on some strong assumptions. In particular, one may think that parents, although they prefer, *ceteris paribus*, having a child of their type, may nonetheless be divided as to what they should do in a context where the lifestyle affects the survival prospects of their children. Actually, it could be argued that parents are divided between two desires. On the one hand, the desire to coexist with a child like themselves, that is, a child with whom they share the same lifestyle. On the other hand, the desire to a have a child that is as healthy as possible.

For instance, parents who smoke may not want their children to smoke too, even though such parents would feel quite isolated in their unhealthy lifestyle if their children did not smoke. The same can be said of alcoholic behaviour: alcoholic parents do not want their children to be like them. Note, however, that in many other cases, the balance between the two desires is less obvious. For instance, the accommodation choice may be a source of tensions between the two desires: if parents live in a highly polluted urban area, they will probably want their children to live also in the same area, and not in a distant, unpolluted rural area far in the countryside, even if this would be better for the health of their children. Similarly, workaholic parents are likely to prefer to have quite active children, even though this is not good for their health. Thus in many cases there may be conflicts between the willingness to share something with children and the willingness to "have the best" for their children.

The basic model only accounted for the first of those motives, but did not allow parents to be concerned with the health of their children. In this section, we extend the model, to allow for the second kind of parental desire. For that purpose, we will assume that parents derive also some utility from the expected health of the child. As we shall see, this will affect the dynamics of heterogeneity in the population.

[16] Given that $q^2 = \frac{\pi^H}{\pi^H + \pi^U}$, the long-run aggregate life expectancy Λ is $2 + \frac{(\pi^H)^2 + (\pi^U)^2}{\pi^H + \pi^U}$. Hence the impact of a marginal rise in π^U is: $\frac{\partial \Lambda}{\partial \pi^U} = \frac{2\pi^H \pi^U - (\pi^H)^2 + (\pi^U)^2}{(\pi^H + \pi^U)^2}$. Hence this is negative if and only if $(\pi^U)^2 < (\pi^H)^2 - 2\pi^H \pi^U$.

4.1 A modified framework

If parents derive some utility from the health status of their children, independently from whether their children are like them or not, the objective function of a parent of type $i \in \{H, U\}$ can be written as:

$$U^i = -\delta \frac{\left(e^i\right)^2}{2} + \beta \pi^i \left[p_{t+1}^{ii} \hat{\varphi} + p_{t+1}^{ij} \tilde{\varphi} \right] + \alpha \left(E(\pi^{child}) \right) \tag{8}$$

where α reflects health-oriented altruism towards the child (not full altruism, as there is no care for socialization), while $E(\pi^{child})$ is the expected health status of the child.[17] The additional term introduced here is not weighted by the probability of survival of the parent, as we assume that this altruism is not conditional on coexistence with the child. The parent wants, in any case, his child to have a good health. Finally, note also that the additional term does not need to be interpreted in terms of altruism: it could be the case that parents just care about having healthy children, without any benevolence behind.[18]

This objective function can be rewritten as

$$U^i = -\delta \frac{\left(e^i\right)^2}{2} + \beta \pi^i \left[p_{t+1}^{ii} \hat{\varphi} + p_{t+1}^{ij} \tilde{\varphi} \right] + \alpha \left(p_{t+1}^{ii} \pi^i + p_{t+1}^{ij} \pi^j \right) \tag{9}$$

Assuming an interior solution, the optimal socialization effort is now:

$$e_t^i = \frac{\beta \pi^i (1 - q^i) \left(\hat{\varphi} - \tilde{\varphi} \right) + \alpha (1 - q^i) \left(\pi^i - \pi^j \right)}{\delta} \tag{10}$$

If α equals 0, we are back to basic model. Otherwise, there is now an additional term at the numerator of the RHS.[19] For healthy parents, the factor $\left(\pi^i - \pi^j\right)$ is positive, which reinforces the socialization effort in comparison with self-oriented parents. On the contrary, for unhealthy parents, this additional term is negative, and thus pushes the socialization effort at a lower level. However, given the transmission technology $\rho_{t+1}^i = e_t^i$, the socialization effort must be non-negative. Thus, if the altruistic motive is large, or if the longevity differential is strong, the optimal socialization effort chosen by unhealthy parents is a corner solution, i.e. $e_t^U = 0$.

Actually, the socialization efforts of healthy and unhealthy parents are given by

$$\begin{aligned}
e_t^H &= \frac{\beta \pi^H (1 - q^H) \left(\hat{\varphi} - \tilde{\varphi} \right) + \alpha (1 - q^H) \left(\pi^H - \pi^U \right)}{\delta} > 0 \\
e_t^U &= \frac{\beta \pi^U (1 - q^U) \left(\hat{\varphi} - \tilde{\varphi} \right) + \alpha (1 - q^U) \left(\pi^U - \pi^H \right)}{\delta} > 0 \text{ if } \beta \pi^U \left(\hat{\varphi} - \tilde{\varphi} \right) > \alpha \left(\pi^H - \pi^U \right) \\
&= 0 \qquad \text{if } \beta \pi^U \left(\hat{\varphi} - \tilde{\varphi} \right) \leq \alpha \left(\pi^H - \pi^U \right)
\end{aligned}$$

In the latter case, the unhealthy parent does not socialize his child, in the sense that there will be no investment to make him share the parent's lifestyle.

[17] See Ponthiere (2007) on empirical studies estimating intrafamily altruistic weights.

[18] Nonetheless, the time structure of this model is such that the parental concern for the child's life expectancy cannot be caused by selfish coexistence concerns, as the survival of children concerns the old age, at which their parents are necessarily dead.

[19] Hence, to have $e_t^i \leq 1$, we can now impose $\frac{\beta \pi^i (\hat{\varphi} - \tilde{\varphi}) + \alpha \left(\pi^i - \pi^j \right)}{\delta} \leq 1$.

To sum up, in this extended model, there are *two forces* driving parental socialization efforts. On the one hand, parents would like their children to have the *same* lifestyle as theirs; on the other hand, parents would like to *prevent* their children from *early death*. It is important to notice that those two forces do not contradict each other for healthy parents, for whom the promotion of their own lifestyle H coincides with the promotion of best survival prospects for their children. However, for unhealthy parents, there is a conflict between those two forces: the former recommends an effort yo transmit the U-type, whereas the latter does not. Hence, when the latter force exceeds the former one, unhealthy parents prefer not investing in the socialization of their children at all. That case happens when parental altruism is sufficiently strong, and when the life expectancy gap between the two lifestyles is sufficiently large, that is, when we have $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H-\pi^U\right)$.

4.2 Long-run dynamics

Substituting for $\rho_{t+1}^H = e_t^H$ and $\rho_{t+1}^U = e_t^U$ in the expression

$$q_{t+1} = \left[\rho_{t+1}^H + \left(1-\rho_{t+1}^H\right)q_t\right]q_t + \left[\left(1-\rho_{t+1}^U\right)q_t\right](1-q_t)$$

yields the following transition functions $q_{t+1} \equiv G_1(q_t)$ and $q_{t+1} \equiv G_2(q_t)$:

$$G_1(q_t) = q_t + q_t(1-q_t)\frac{\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi})+\alpha\left(\pi^H-\pi^U\right)\right](1-q_t)-\left[\beta\pi^U(\hat{\varphi}-\tilde{\varphi})+\alpha\left(\pi^U-\pi^H\right)\right]q_t}{\delta}$$
$$\text{if } \beta\pi^U(\hat{\varphi}-\tilde{\varphi}) > \alpha\left(\pi^H-\pi^U\right)$$

$$G_2(q_t) = q_t + (1-q_t)q_t\frac{\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi})+\alpha\left(\pi^H-\pi^U\right)\right](1-q_t)}{\delta}$$
$$\text{if } \beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H-\pi^U\right)$$

As in the previous section, the existence and uniqueness of a stationary equilibrium can be discussed by exploring whether the transition function admits a fixed point. For that purpose, we will here distinguish between two cases, depending on the transition function. Proposition 2 summarizes our results.

Proposition 2 *(1) If* $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) > \alpha\left(\pi^H-\pi^U\right)$*, there exist three stationary equilibria:*

$$\begin{aligned} q^1 &= 0\ ;\ q^3 = 1 \\ q^2 &= \frac{\beta\pi^H(\hat{\varphi}-\tilde{\varphi})+\alpha\left(\pi^H-\pi^U\right)}{\beta\pi^U(\hat{\varphi}-\tilde{\varphi})+\beta\pi^H(\hat{\varphi}-\tilde{\varphi})} > \frac{1}{2} \end{aligned}$$

where only q^2 *is locally stable.*
(2) If $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H-\pi^U\right)$*, there exist two stationary equilibria:*

$$q^1 = 0\ ;\ q^2 = 1$$

where only q^2 *is locally stable.*

Proof. See the Appendix. ■

Thus, if the strength of altruism is limited, and/or if the longevity differential between healthy and unhealthy agents is small, there still exist three stationary equilibria, as in the basic model. Among those equilibria, two of them involve

a homogeneous population, while the intermediate equilibrium, which is the unique locally stable one, involves a heterogeneous population. Note, however, that the intermediate equilibrium has here a different form in comparison with the basic model. Clearly, it involves a larger proportion of healthy persons than in the basic model.[20] Hence, the introduction of parental altruistic concerns for children's health tends to raise the long-run proportion of healthy agents, and, thus, the long-run aggregate life expectancy.

If altruism is strong enough, and/or if the longevity differential is large, the intermediate equilibrium does not exist, and the equilibrium with a population made exclusively of healthy agents is locally stable. The same result holds if agents strongly discount the future (i.e. β is low) or if the welfare gains from having a child of one's type are low (i.e. $\hat{\varphi} - \tilde{\varphi}$ is close to zero). Hence, any population with at least some agents adhering to a healthy lifestyle will end up being fully made of agents having a healthy lifestyle and, hence, a high life expectancy. Figures 2 and 3 below illustrate the two cases.[21]

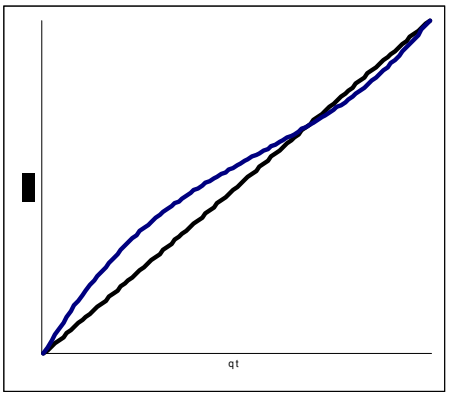

Figure 2: The altruistic model: case (1)

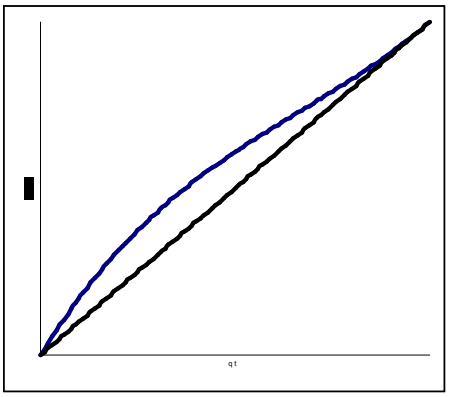

Figure 3: The altruistic model: case (2)

Thus, the introduction of a parental altruistic motive may affect the long-run dynamics of lifestyles and longevity. Clearly, if the parental altruism is sufficiently large and/or the longevity differential induced by lifestyles is sufficiently wide, only the healthy lifestyle must prevail in the long-run: the equilibrium population is necessarily homogeneous. Hence Proposition 2 suggests that the survival of unhealthy lifestyles at the equilibrium can only be due to (1) a low parental altruism (in comparison with the egoistic welfare gains from socialization); and (2) the fact that the unhealthy lifestyle does not yield a too big reduction of life expectancy in comparison with the healthy lifestyle.

Therefore, despite parental altruism, some - relatively benign - unhealthy lifestyles will survive in the long-run, but not more dangerous lifestyles. Lifestyles yielding a large mortality differential will mechanically disappear, as parents will

[20] This is so because a positive constant is added to the numerator of the intermediate equilibria under the basic model.

[21] Figure 2 relies on the following parameters values: $\alpha = 0.2$; $\beta = 0.6$; $\delta = 0.5$; $\hat{\varphi} - \tilde{\varphi} = 1.5$; $\pi^H = 0.5$ and $\pi^U = 0.3$. Figure 3 relies on the parameters: $\alpha = 0.4$; $\beta = 0.6$; $\delta = 0.5$; $\hat{\varphi} - \tilde{\varphi} = 0.5$; $\pi^H = 0.8$ and $\pi^U = 0.3$.

not invest in the transmission of those lifestyles *even if* they adopted these when being younger.[22]

Finally, regarding the dynamics of the aggregate life expectancy, we can make here the same observation as above: starting from a population where the healthy lifestyle is dominated by the unhealthy lifestyle (i.e. $0 < q_0 < 1/2$), the economy will, over time, exhibit a rise in the aggregate life expectancy, as the proportion of healthy agents in the population goes up during the transition towards the long-run equilibrium. Nevertheless, a significant difference here is that the long-run aggregate life expectancy is, provided $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) > \alpha\left(\pi^H-\pi^U\right)$, a function not only of group-specific life expectancies π^H and π^U, but, also, of preference parameters such as β (time preferences) and α (parental altruism), unlike in the basic model. Furthermore, if $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) \leq \alpha\left(\pi^H-\pi^U\right)$, the proportion of healthy persons will tend towards 1, so that the aggregate life expectancy will be here merely $2+\pi^H$.

Regarding the effect of an exogenous rise in π^U on aggregate life expectancy, this will, under $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) > \alpha\left(\pi^H-\pi^U\right)$, reduce the long-run level of q, and may thus also, quite paradoxically, reduce long-run aggregate life expectancy, as in the basic model. On the contrary, as long as $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) \leq \alpha\left(\pi^H-\pi^U\right)$, a rise in π^U will not affect the long-run level of aggregate life expectancy, equal, in that case, to $2+\pi^H$. Indeed, as long as $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) \leq \alpha\left(\pi^H-\pi^U\right)$, unhealthy parents do not invest in the transmission of the unhealthy lifestyle, and so a change in their life expectancy does not have any effect on the socialization process. This constitutes another significant departure from the standard model. Therefore, the introduction of parental altruistic concerns for children's health influences also the long-run dynamics of aggregate life expectancy.

5 A model with asymmetric socialization costs

The present analysis, by highlighting the evolutionary advantage of healthy lifestyles, tends to provide a quite optimistic message. Actually, the basic model developed in Sections 2 and 3 emphasized that, thanks to the larger life expectancy associated with a healthy lifestyle, healthy parents must, *ceteris paribus*, invest more in the socialization of their children, so that the healthy lifestyle must be majoritary in the long-run. Moreover, the introduction of parental altruism (Section 4) tends to reinforce that optimistic message.

However, it should be stressed that our analysis relied so far on some significant simplifications, which may be far from neutral for the issue at stake. In particular, we assumed, throughout our study, that the costs of socialization for parents are equal *whatever* the lifestyle that they want to promote. More precisely, the welfare cost from socialization efforts are equal for parents who want to transmit a healthy lifestyle and for parents who want to transmit an unhealthy lifestyle. That assumption is quite strong, as it may be more difficult to transmit a healthy lifestyle rather than an unhealthy lifestyle. Take, for instance, the case of eating habits. It is probably more difficult for a parent to transmit to his children the lifestyle "non-snacking between meals", rather than to transmit the lifestyle "snacking between meals". Thus assuming equal socialization costs across types seems to simplify the picture significantly.

[22] In some sense, for those lifetyles, the altruistic concern will overcome the will to have children like oneself.

5.1 Unequal socialization costs

To explore the robustness of our conclusions to that aspect of the socialization process, let us now develop another extension of our model, where the cost of socialization varies across the parents, depending on whether the lifestyle to be transmitted is healthy or unhealthy.

Introducing asymmetric socialization costs in our model can be made by assuming that the disutility of socialization varies across parents, depending on the lifestyle to which they adhere. Thus we have now

$$C(e_t^i) = \delta^i \frac{\left(e_t^i\right)^2}{2} \tag{11}$$

where δ^i, which accounts for the disutility of socialization efforts ($\delta^i > 0$), is now type-specific. In the example developed above (eating habits), it is likely that $\delta^H > \delta^U$, since the healthy lifestyle is more difficult to transmit than the unhealthy lifestyle. Note, however, that this may not be the case. One could imagine some healthy lifestyles that are easier to transmit. For instance, the (unhealthy) lifestyle "workaholic" may be more difficult to transmit than the (healthy) lifestyle "non-workaholic", leading to $\delta^H < \delta^U$. We will thus, for the sake of generality, not restrict ourselves to a particular assumption.

The objective function for a parent of type $i \in \{H, U\}$ is now

$$U^i = -\delta^i \frac{\left(e^i\right)^2}{2} + \beta\pi^i \left[p_{t+1}^{ii}\hat{\varphi} + p_{t+1}^{ij}\tilde{\varphi}\right] + \alpha\left(p_{t+1}^{ii}\pi^i + p_{t+1}^{ij}\pi^j\right) \tag{12}$$

Assuming an interior solution, the optimal socialization effort for a parent of type $i \in \{H, U\}$ is now:[23]

$$e_t^i = \frac{\beta\pi^i(1-q^i)\left(\hat{\varphi}-\tilde{\varphi}\right) + \alpha(1-q^i)\left(\pi^i - \pi^j\right)}{\delta^i} \tag{13}$$

As above, the transmission technology $\rho_{t+1}^i = e_t^i$ presupposes that the socialization effort must be non-negative. Hence we have

$$\begin{aligned}
e_t^H &= \frac{\beta\pi^H(1-q^H)\left(\hat{\varphi}-\tilde{\varphi}\right) + \alpha(1-q^H)\left(\pi^H - \pi^U\right)}{\delta^H} > 0 \\
e_t^U &= \frac{\beta\pi^U(1-q^U)\left(\hat{\varphi}-\tilde{\varphi}\right) + \alpha(1-q^U)\left(\pi^U - \pi^H\right)}{\delta^U} > 0 \text{ if } \beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) > \alpha\left(\pi^H - \pi^U\right) \\
&= 0 \qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad \text{if } \beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right) \leq \alpha\left(\pi^H - \pi^U\right)
\end{aligned}$$

When the welfare costs from socialization are equal across parents with distinct lifestyles (i.e. $\delta^H = \delta^U = \delta$), whether healthy parents invest more or less in the socialization of their children than unhealthy parents depends on the life expectancy gap between the two lifestyles (i.e. π^H *versus* π^U), on the relative proportions of the two lifestyles in the population (i.e. q^H *versus* q^U), and on degree of altruism α, but is invariant to the level of δ, as this plays symmetrically for the two types of parents. However, once $\delta^H \neq \delta^U$, there is an additional factor explaining differences in socialization efforts. If the healthy lifestyle is

[23] To have $e_t^i \leq 1$, we now impose $\frac{\beta\pi^i(\hat{\varphi}-\tilde{\varphi})+\alpha\left(\pi^i-\pi^j\right)}{\delta^i} \leq 1$.

more difficult to transmit than the unhealthy one (i.e. $\delta^H > \delta^U$), this pushes the socialization effort of the healthy parent downwards. On the contrary, if the healthy lifestyle is less difficult to transmit (i.e. $\delta^H < \delta^U$), this pushes the socialization effort of the healthy parent upwards.

5.2 Long-run dynamics

Substituting for $\rho_{t+1}^H = e_t^H$ and $\rho_{t+1}^U = e_t^U$ in the expression

$$q_{t+1} = \left[\rho_{t+1}^H + \left(1 - \rho_{t+1}^H\right) q_t\right] q_t + \left[\left(1 - \rho_{t+1}^U\right) q_t\right] (1 - q_t)$$

yields the following transition functions $q_{t+1} \equiv G_1(q_t)$ and $q_{t+1} \equiv G_2(q_t)$:

$$G_1(q_t) = q_t + (1 - q_t) q_t \left[\frac{\left[\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right](1 - q_t)}{\delta^H} - \frac{\left[\beta\pi^U(\hat{\varphi} - \tilde{\varphi}) + \alpha\left(\pi^U - \pi^H\right)\right] q_t}{\delta^U}\right]$$
$$\text{if } \beta\pi^U(\hat{\varphi} - \tilde{\varphi}) > \alpha\left(\pi^H - \pi^U\right)$$

$$G_2(q_t) = q_t + (1 - q_t) q_t \frac{\left[\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right](1 - q_t)}{\delta^H}$$
$$\text{if } \beta\pi^U(\hat{\varphi} - \tilde{\varphi}) \leq \alpha\left(\pi^H - \pi^U\right)$$

As in the previous sections, the existence and uniqueness of a stationary equilibrium can be studied by exploring whether the transition function admits a fixed point. Proposition 3 summarizes our results.

Proposition 3 *(1) If* $\beta\pi^U(\hat{\varphi} - \tilde{\varphi}) > \alpha\left(\pi^H - \pi^U\right)$*, there exist three stationary equilibria:*

$$q^1 = 0 \; ; \; q^3 = 1$$
$$q^2 = \frac{\frac{\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)}{\delta^H}}{\frac{\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \alpha(\pi^H - \pi^U)}{\delta^H} + \frac{\beta\pi^U(\hat{\varphi} - \tilde{\varphi}) + \alpha(\pi^U - \pi^H)}{\delta^U}}$$

where only q^2 *is locally stable.*
(2) If $\beta\pi^U(\hat{\varphi} - \tilde{\varphi}) \leq \alpha\left(\pi^H - \pi^U\right)$*, there exist two stationary equilibria:*

$$q^1 = 0 \; ; \; q^2 = 1$$

where only q^2 *is locally stable.*

Proof. See the Appendix. ■

In comparison with Proposition 2, Proposition 3 differs regarding the level of the intermediate long-run equilibrium q^2, which, here again, is the unique locally stable equilibrium when $\beta\pi^U(\hat{\varphi} - \tilde{\varphi}) > \alpha\left(\pi^H - \pi^U\right)$. It is straightforward to see that, provided the welfare costs from socialization were equal, i.e. $\delta^H = \delta^U = \delta$, then the expression for the intermediate equilibrium q^2 would collapse to

$$q^2 = \frac{\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)}{\beta\pi^H(\hat{\varphi} - \tilde{\varphi}) + \beta\pi^U(\hat{\varphi} - \tilde{\varphi})}$$

which is independent from the socialization cost parameter δ. As discussed above, we know that, under equal socialization costs, the healthy lifestyle has an evolutionary advantage: $q^2 > 1/2$. However, this is no longer necessarily the

case under unequal socialization costs, as the healthy lifestyle may be dominated in the long-run:

$$q^2 < \frac{1}{2} \iff \frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)} < \frac{\delta^H}{\delta^U}$$

Under equal socialization costs, the RHS equals 1, and the LHS is necessarily larger than 1 (given $\pi^H > \pi^U$), so that the condition is never satisfied. However, if the healthy lifestyle is much more costly to transmit than the unhealthy lifestyle, i.e. $\delta^H >> \delta^U$, than the above condition may be satisfied, provided the life expectancy gap between the two lifestyles is not too large, and provided parental altruism is not too strong.

In sum, this section shows that, despite the lifetime horizon effect and the presence of parental altruism, which both favour the transmission of the healthy lifestyle, the mere introduction of asymmetric socialization costs suffices to qualify the optimistic result according to which healthy lifestyles must dominate unhealthy lifestyles in the long-run. How costly the socialization is to parents is a key ingredient for understanding the long-run dynamics of healthy and unhealthy habits and lifestyles, and, from that, the dynamics of life expectancy.

6 Concluding remarks

The starting point of this paper was the large empirical literature on the intergenerational transmission of health and survival outcomes, and on the significant impact of lifestyles on these. While those studies suggest that the transmission of lifestyles across generations may be a significant phenomenon, these tell us little on the equilibrium prevalence of (un)healthy lifestyles in the long-run.

To answer that question, we set up a three-period OLG model with a socialization process following Bisin and Verdier (2001), and where the socialization concerns the adherence to a healthy or an unhealthy lifestyle, which yield distinct life expectancies. We show that, if parents are only motivated by the desire to share their lifestyle with their children, there exist three long-run equilibria in that economy, each of these coincides with a constant partition of the population in the different lifestyles: two homogeneous equilibrium populations, and one heterogeneous one, which involves a majority of agents with the healthy lifestyle. This evolutionary advantage of the healthy lifestyle comes from the fact that the longer time horizon associated to it leads to a higher socialization effort of the healthy parents *ceteris paribus* in comparison with the socialization effort of the unhealthy parents. Note, however, that, given that the intermediate equilibrium is the only one that is locally stable, unhealthy lifestyles are, under such a framework, likely to survive, at least to some extent, in the long-run, despite their negative effects on life expectancy.

Then, we proposed another framework, where parents care not only about sharing their lifestyle with their children, but care also altruistically about the health of their children.[24] We show that, if parents are strongly altruistic, and if the longevity gap induced by lifestyles is sufficiently large, only healthy

[24] The postulated form of concern for the child's health can be, in principle, regarded as egoistic. However, given that it cannot be driven by coexistence (as parents are dead at the old age, whatever their children are long-lived or not), it is more plausible to regard that concern as altruistic.

lifestyles can survive in the long-run, as strongly altruistic parents do not invest in the transmission of a lifestyle that causes much damage to the health of their children. Otherwise, if the unhealthy lifestyle is not too damageable, there still exists an equilibrium with a heterogeneous population dominated by the healthy lifestyle. It follows from this that, under parental altruism, only *some* - but not all - unhealthy lifestyles will survive in the long-run.

Finally, we developed an expanded version of the model inclusive of asymmetric socialization costs, varying across the lifestyles to be transmitted by parents. The intuition underlying that theoretical generalization is that healthy lifestyles may be more difficult to transmit by parents than unhealthy lifestyles. The introduction of unequal socialization costs affects our results significantly, since the healthy lifestyle may not exhibit an evolutionary advantage. The healthy lifestyle may be dominated in the long-run, provided it is much more costly to transmit than the unhealthy lifestyle, if the life expectancy gap between the two lifestyles and the parental altruistic concerns are small.

In the light of those findings, one can try to extrapolate the future of some unhealthy lifestyles: what about the future of smoking, alcoholism, and unhealthy eating habits? The answer was shown to depend on (1) the mortality gap induced by those practices; (2) the degree of altruism of parents (in comparison with the welfare gains from coexisting with a child like them); (3) the socialization costs associated with the healthy and unhealthy lifestyles. Depending on those factors, unhealthy lifestyles will survive to a more or less large extent. Given the difficulties to estimate preference parameters in general (including altruistic concerns and disutility from socialization efforts), no answer can be given for the long-run prevalence of a particular lifestyle. Nevertheless, some qualitative predictions can be made on the basis of observed mortality differentials induced by lifestyles. According to the estimates in Kaplan et al. (1987), smoking, which leads to a larger mortality gap than alcoholism or overweight, is likely to survive to a smaller extent than those two other unhealthy lifestyles in the long-run (everything else being constant).

Note, however, that the present study, which focused on the dynamics of (un)healthy lifestyles transmission through a vertical or oblique (decentralized) socialization process, ignored a major agent: the State. Given that governments may affect the transmission of lifestyles through various channels (e.g. information campaigns, education programs), the absence of a government is a major simplification in our model.[25] Hence it makes sense to add a few words here on the impact of governments on the dynamics of lifestyles transmission, and, in a second stage, to try to draw policy conclusions.

Regarding the former point, it should be stressed that governments can have a large influence on the dynamics of lifestyles transmission. However, it is hard, in the light of the model, to draw a precise conclusion on the sign of that influence, since various effects play in opposite directions. A government could, by information campaigns, support the parents's attempts to transmit a healthy lifestyle, which would, by reducing δ^H, *raise* the long-run prevalence of the healthy lifestyle.[26] But, at the same time, government-funded medical

[25] On the effectiveness of public health programs (and its measurement of spillovers arising from these), see Chaudhuri (2009).

[26] Indeed, teachers could, by supporting the healthy parents' arguments, make the transmission of the healthy lifestyle easier. Note, however, that this effect is far from certain, as teaching programs may, if inadequate, be counterproductive, especially with teenagers.

research against diseases related to unhealthy lifestyles raises π^U, which *reduces* the long-run prevalence of the healthy lifestyle *ceteris paribus*. Hence it is not obvious to draw clear conclusions on the actual role of governments.

Moreover, it is even more difficult to characterize the optimal public policy, since this would depend on another aspect of the problem, which was not treated here: the temporal welfare levels associated with the different lifestyles. Clearly, unhealthy lifestyles are also a source of gains in terms of temporal welfare (see, for instance, sin goods consumptions, such as alcohol, tobacco, etc.). That aspect should also be taken into account when discussing the optimal public policy. Thus one should be cautious before drawing normative conclusions from the present positive dynamic analysis.[27]

To conclude, it should also be reminded that the present theoretical framework, despite its generality, may not fully capture all aspects of socialization. Our model relies on a particular lifestyles transmission technology, whereas other, more general, candidate technologies should also be examined. Moreover, socialization is here vertical or oblique, but not horizontal (i.e. intracohort). This constitutes another limitation of the paper, as some lifestyles and behaviours may be transmitted within cohorts rather than between cohorts. Furthermore, the role of the family in the socialization process is here limited to the choice of a socialization effort, which is also a simplification, as other family choices matter (education, fertility, food, etc.).[28] Finally, this model considers decentralized socialization through parents or role models, but does not study the role of centralized education or information programs, which may also influence lifestyles dynamics. Those non-trivial generalizations are left for future research.

7 References

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[27] See Ponthiere (2010) for a study of that issue in the case of a particular longevity-affecting lifestyle: the working time. The optimal policy is shown there to depend on individuals' myopia, and on intergenerational externalities due to the transmission of lifestyles.

[28] The family plays also a major role regarding food (in)security, as studied in Coleman-Jensen (2010) and Guo (2010).

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8 Appendix

Basic model: existence and uniqueness The transition function is such that $G(0) = 0$ and $G(1) = 1$. Hence, $G(q_t)$ meets the 45° line at the two extremities, which are stationary equilibria. Note also that the derivative of $G(q_t)$ with respect to q_t is

$$G'(q_t) = 1 + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\left[\pi^H(1-q_t)^2 - \pi^H q_t 2(1-q_t) - \pi^U 2q_t(1-q_t) + \pi^U q_t^2\right]$$

We have

$$\begin{aligned} G'(0) &= 1 + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\pi^H > 1 \\ G'(1) &= 1 + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\pi^U > 1 \end{aligned}$$

Thus, given that the slope of $G(q_t)$ is higher than 1 at q_t equal to 0 and 1, it must be the case that $G(q_t)$ is above the 45° line in the neighbourhood of 0, and below the 45° line in the neighbourhood of 1. Hence, by continuity, $G(q_t)$ must intersect the 45° line somewhere, for a level of q_t between 0 and 1. Regarding the uniqueness of that intermediate equilibrium, substituting for $q_{t+1} = q_t = q$ in the transition function shows that this intermediate equilibrium takes a single value: $q = \frac{\pi^H}{\pi^H + \pi^U} > \frac{1}{2}$.

Basic model: stability Stability requires $\left|\frac{\partial G}{\partial q_t}\right| < 1$. We have

$$\begin{aligned} G'(0) &= 1 + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\pi^H > 1 \\ G'(1) &= 1 + \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\pi^U > 1 \end{aligned}$$

Thus $q = 0$ and $q = 1$ are not stable.

Finally, at $q_t = \frac{\pi^H}{\pi^H + \pi^U}$, we have:

$$G'(q_t) = 1 - \frac{\beta\left(\hat{\varphi} - \tilde{\varphi}\right)}{\delta}\frac{\pi^H \pi^U}{\pi^H + \pi^U} < 1$$

Given $\frac{\beta \pi^H (\hat{\varphi} - \tilde{\varphi})}{\delta} < 1$, that expression is smaller than 1 in absolute value, so that the equilibrium is locally stable.

Altruistic model: existence and uniqueness Note that $G_1(q_t)$ is such that: $G_1(0) = 0$ and $G_1(1) = 1$. Hence, $G_1(q_t)$ meets the 45° line at the two extremities, which are stationary equilibria. The derivative of $G_1(q_t)$ with respect to q_t is

$$\begin{aligned} G_1'(q_t) &= 1 + [1-2q_t]\frac{\left[\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right](1-q_t) - \left[\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^U - \pi^H\right)\right]q_t\right]}{\delta} \\ &\quad + q_t(1-q_t)\frac{\left[-\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right] - \left[\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^U - \pi^H\right)\right]\right]}{\delta} \end{aligned}$$

We have

$$\begin{aligned} G_1'(0) &= 1 + \frac{\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)}{\delta} > 1 \\ G_1'(1) &= 1 + \frac{\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^U - \pi^H\right)}{\delta} > 1 \end{aligned}$$

Thus, under $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) > \alpha\left(\pi^H - \pi^U\right)$, the transition function lies above the 45° line in the neighbourhood of 0, and below the 45° line in the neighbourhood of 1. Hence, the existence of an intermediate equilibrium can be proved as in the basic model. Fixing $q_{t+1} = q_t = q$ in the transition function yields the intermediate equilibrium:

$$q = \frac{\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)}{\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) + \beta\pi^H(\hat{\varphi}-\tilde{\varphi})} > \frac{1}{2}$$

In the case where $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H - \pi^U\right)$, the transition function $G_2(q_t)$ is such that $G_2(0) = 0$ and $G_2(1) = 1$. Hence, $G_2(q_t)$ meets the 45° line at the two extremities, which are stationary equilibria. Note also that

$$G_2'(q_t) = 1 + \left[-2(1-q_t)q_t + (1-q_t)^2\right]\frac{\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right]}{\delta}$$

We have

$$\begin{aligned} G_2'(0) &= 1 + \frac{\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)}{\delta} > 1 \\ G_2'(1) &= 1 \end{aligned}$$

Thus, under $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H - \pi^U\right)$, the transition function lies above the 45° line in the neighbourhood of 0, but does not lie below the 45° line in the neighbourhood of 1. Actually, it is not difficult to show that there cannot be an intermediate equilibrium in this model. Indeed fixing $q_{t+1} = q_t = q$ in the transition function yields:

$$q = q + (1-q)^2 q\frac{\left[\beta\pi^H(\hat{\varphi}-\tilde{\varphi}) + \alpha\left(\pi^H - \pi^U\right)\right]}{\delta}$$

which cannot be true for $0 < q < 1$. Thus there exists no intermediate equilibrium under $\beta\pi^U(\hat{\varphi}-\tilde{\varphi}) \leq \alpha\left(\pi^H - \pi^U\right)$.

Altruistic model: stability In the case where $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)>\alpha\left(\pi^H-\pi^U\right)$, stability requires $\left|\frac{\partial G_1}{\partial q_t}\right|<1$. We have

$$\begin{aligned}G_1'(q_t) &= 1+[1-2q_t]\,\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right](1-q_t)-\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]q_t}{\delta}\\ &\quad +q_t(1-q_t)\frac{-\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]-\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]}{\delta}\end{aligned}$$

Hence

$$\begin{aligned}G_1'(0) &= 1+\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta}>1\\ G_1'(1) &= 1+\frac{\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)}{\delta}>1\end{aligned}$$

Thus, given $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)>\alpha\left(\pi^H-\pi^U\right)$, neither $q=0$ nor $q=1$ are stable.

Finally, at $q_t=\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)}$, we have:

$$G_1'(q_t)=1-\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]}{\delta\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)\right]}<1$$

Hence, given that socialization efforts are between 0 and 1, the intermediate equilibrium must be locally stable.

When $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)\leq\alpha\left(\pi^H-\pi^U\right)$, stability requires $\left|\frac{\partial G_2}{\partial q_t}\right|<1$. We have

$$G_2'(q_t)=1+\left[-2(1-q_t)q_t+(1-q_t)^2\right]\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]}{\delta}$$

Hence

$$\begin{aligned}G_2'(0) &= 1+\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]}{\delta}>1\\ G_2'(1) &= 1\end{aligned}$$

so that $q=0$ is not stable. Regarding $q=1$, the equality $G_2'(1)=1$ does not exactly coincide with what insures local stability (a strict inequality). However, it is clear that the transition function crosses the 45° line from above at 1, and thus that equilibrium can be regarded as locally stable.

Asymmetric costs: existence and uniqueness Note first that the transition function $G_1(q_t)$ is such that: $G_1(0)=0$ and $G_1(1)=1$. Hence, $G_1\left(q_t\right)$ meets the 45° line at the two extremities, which are stationary equilibria. Note also that:

$$\begin{aligned}G_1'(q_t) &= 1+(1-2q_t)\left[\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right](1-q_t)}{\delta^H}-\frac{\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]q_t}{\delta^U}\right]\\ &\quad +(1-q_t)q_t\left[-\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]}{\delta^H}-\frac{\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]}{\delta^U}\right]\end{aligned}$$

We have

$$\begin{aligned} G_1'(0) &= 1+\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta^H}>1 \\ G_1'(1) &= 1+\frac{\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)}{\delta^U}>1 \end{aligned}$$

Thus, under $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)>\alpha\left(\pi^H-\pi^U\right)$, the transition function lies above the 45° line in the neighbourhood of 0, and below the 45° line in the neighbourhood of 1. Hence, the existence of an intermediate equilibrium can be proved as in the basic model. Fixing $q_{t+1}=q_t=q$ in $q_{t+1}=G(q_t)$ allows us to derive the intermediate equilibrium:

$$q=\frac{\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta^H}}{\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta^H}+\frac{\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)}{\delta^U}}$$

When $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)\leq\alpha\left(\pi^H-\pi^U\right)$, $G_2(q_t)$ is such that: $G_2(0)=0$ and $G_2(1)=1$. Hence, $G_2(q_t)$ meets the 45° line at the two extremities, which are stationary equilibria. Note also that

$$G_2'(q_t)=1+\left[-2(1-q_t)q_t+(1-q_t)^2\right]\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta^H}$$

We have

$$\begin{aligned} G_2'(0) &= 1+\frac{\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)}{\delta}>1 \\ G_2'(1) &= 1 \end{aligned}$$

Thus, under $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)\leq\alpha\left(\pi^H-\pi^U\right)$, the transition function lies above the 45° line in the neighbourhood of 0, but does not lie below the 45° line in the neighbourhood of 1. Actually, it is not difficult to show that there cannot be an intermediate equilibrium in this model. Indeed fixing $q_{t+1}=q_t=q$ in the transition function yields:

$$q=q+(1-q)^2q\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]}{\delta^H}$$

which cannot be true for $0<q<1$. Thus there exists no intermediate equilibrium under $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)\leq\alpha\left(\pi^H-\pi^U\right)$.

Asymmetric costs: stability If $\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)>\alpha\left(\pi^H-\pi^U\right)$, stability requires $\left|\frac{\partial G_1}{\partial q_t}\right|<1$. We have

$$\begin{aligned} G_1'(q_t) &= 1+(1-2q_t)\left[\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right](1-q_t)}{\delta^H}-\frac{\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]q_t}{\delta^U}\right] \\ &\quad +(1-q_t)q_t\left[-\frac{\left[\beta\pi^H\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^H-\pi^U\right)\right]}{\delta^H}-\frac{\left[\beta\pi^U\left(\hat{\varphi}-\tilde{\varphi}\right)+\alpha\left(\pi^U-\pi^H\right)\right]}{\delta^U}\right] \end{aligned}$$

We have

$$\begin{aligned} G_1'(0) &= 1 + \frac{\beta\pi^H\left(\hat{\varphi} - \tilde{\varphi}\right) + \alpha\left(\pi^H - \pi^U\right)}{\delta^H} > 1 \\ G_1'(1) &= 1 + \frac{\beta\pi^U\left(\hat{\varphi} - \tilde{\varphi}\right) + \alpha\left(\pi^U - \pi^H\right)}{\delta^U} > 1 \end{aligned}$$

Thus, given $\beta\pi^U\left(\hat{\varphi} - \tilde{\varphi}\right) > \alpha\left(\pi^H - \pi^U\right)$, $q = 0$ and $q = 1$ are not stable.

Finally, at $q_t = q^2$, we have: $G_1'(q_t) < 1$, so that, given that individual socialization efforts are between 0 and 1, the intermediate equilibrium, if it exists, must be locally stable.

If $\beta\pi^U\left(\hat{\varphi} - \tilde{\varphi}\right) \leq \alpha\left(\pi^H - \pi^U\right)$, stability requires $\left|\frac{\partial G_2}{\partial q_t}\right| < 1$. We have

$$G_2'(q_t) = 1 + \left[-2(1-q_t)q_t + (1-q_t)^2\right]\frac{\beta\pi^H\left(\hat{\varphi} - \tilde{\varphi}\right) + \alpha\left(\pi^H - \pi^U\right)}{\delta}$$

Hence

$$\begin{aligned} G_2'(0) &= 1 + \frac{\beta\pi^H\left(\hat{\varphi} - \tilde{\varphi}\right) + \alpha\left(\pi^H - \pi^U\right)}{\delta} > 1 \\ G_2'(1) &= 1 \end{aligned}$$

so that $q = 0$ is not stable. Regarding $q = 1$, the equality $G_2'(1) = 1$ does not exactly coincide with what insures local stability (a strict inequality). However, it is clear that the transition function crosses the 45° line from above at 1, and, thus that equilibrium can be regarded as locally stable.