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ABSTRACT

A number of behaviours influence health in a non-monotonic way. Physical activity and alcohol consumption, for instance, may be beneficial to one's health in moderate but detrimental in large quantities. We develop a demand-for-health framework that incorporates the feature of a physiologically optimal level. An individual may still choose a physiologically non-optimal level, because of the trade-off in his or her preferences for health versus other utility-affecting commodities. However, any deviation from the physiologically optimal level will be punished with respect to health. A set of steady-state comparative statics is derived regarding the effects on the demand for health and health-related behaviour, indicating that individuals react differently to exogenous changes, depending on the amount of the health-related behaviour they demand. We also show (a) that a steady-state equilibrium is a saddle-point and (b) that the physiologically optimal level may be a steady-state equilibrium for the individual. Our analysis suggests that general public-health policies may, to some extent, be counterproductive due to the responses induced in part of the population.

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1. INTRODUCTION

Most human behaviours are related to health. Individual health affects consumption patterns, but consumption patterns also affect individual health. While the sole intention of health-care utilisation is either to improve current health, whenever it has fallen below a certain illness-defining threshold value, or to prevent future illness, rather than to consume it for the sake of its direct utility (which might even be negative), the intention of many other behaviours may be twofold: both to gain direct consumption utility and to improve health (or to decrease the risk of illness). The latter category includes, for instance, physical exercise, certain consumption and composition of food, alcohol consumption and, as a matter of fact, any recreational activity (art, literature, music, etc). Obviously, health effects may be more or less intentional, and certain consumption patterns may also be detrimental to your health. Smoking is an unambiguous example of the latter.

Smoking is always bad for your health – and increasingly so with increased consumption (Doll et al, 1976; Doll et al, 1994; Colditz, 2000; Vineis et al, 2004). In contrast, there appears to be a physiologically determined, individually optimal level of activity (greater than zero) as regards, for instance, physical exercise, food intake, alcohol consumption, and sleep, implying that activity levels below or above that level would reduce the positive health effects of the activity. A consistently positive association between physical-activity level and health-related quality of life has been found (Bize et al, 2007). Certainly, too small amounts of physical exercise means that the human body atrophies and that the risks of several diseases, including coronary heart disease, hypertension, stroke, diabetes, depression, osteoporosis, and cancers of the breast and colon, increase (Garrett et al, 2004). However, too much or too intensive physical exercise means that the human body will wear down and/or that the risk of injury increases (Tisi and Shearman, 1998; Locke, 1999; Ji, 2001; Randolph, 2007; Howatson G and van Someren, 2008; Morton et al, 2009). A varied and balanced diet is emphasised in guidelines on healthy eating; see, for instance

(Swedish National Food Agency, 2012). Too little food or too one-sided diet lead to health problems (Steinhausen, 2002). Too much also creates health problems (Steinhausen and Weber, 2009), in particular in combination with too little physical exercise. Overweight ($25 \leq BMI < 30$)[1] and obesity ($BMI \geq 30$) increase the risks of asthma, coronary heart disease, hypertension, diabetes, osteoarthritis, and cancer, including cancers of the breast and colon (Colditz, 1999; Must et al., 1999); Dal Grande, 2009). Also underweight ($BMI < 18.5$) has been shown to be associated with health problems; for instance, coronary heart disease, diabetes, and gallbladder disease (Must et al., 1999). Light to moderate drinkers are at lower risk of coronary heart disease, stroke, diabetes, and gallstone disease than non-drinkers, while an increasing intake increases the risks of dementia, liver cirrhosis, pancreatitis, osteoporosis, and most cancers, including cancer of the oesophagus, breast, pancreas, colon, and rectum (Grönbaeck, 2009). Finally, both short and long sleep durations appear to be related to increased likelihood of obesity, diabetes, hypertension, and cardiovascular disease (Buxton and Marcelli, 2010; Sabanayagam and Shankar, 2010). It should be emphasised, though, that which level of physical exercise, food intake, alcohol consumption, and sleep is physiologically optimal differs among individuals, and if you have good genes and/or are lucky, you may suffer less from "unhealthy" behaviour than less advantaged people.

In general terms, these associations have been known for decades. Yet, there are no clear temporal trends worldwide towards healthier life-styles (Knuth and Hallai, 2009), and the population variance of these behaviours is large; for instance, many people do not perform any, or very little, physical exercise, others perform very large amounts. We will demonstrate that such polarization may be possible to explain, within a modified version of Grossman's demand-for-health model, assuming that there is a (strictly positive) physiologically optimal level of the corresponding health behaviour.

[1] BMI (body mass index) is calculated as weight in kilograms divided by height in meters.

The demand-for-health model extended the human capital theory by explicitly incorporating health and recognising that there are both consumption and investment motives for investing in health (Grossman, 1972a, b). It resulted in an economic theory of individual health-related behaviour. The basic features of the model are (1) that the individual demands health (a) for its utility enhancing effects (the consumption motive), and (b) for its effect on the amount of healthy time (the investments motive), (2) that the demand for investments in health is derived from the more fundamental demand for health, (3) that the investments in health are produced by the individual, and (4) that the stock of health depreciates at each point in time. The production aspect implies that the produced amount of investments in health has to be assimilated by the individual. Thus, the effects of, for instance, physical exercise, is assimilated and transformed into health by the individual at rates that differ between individuals. This goes beyond the effect of the depreciation of the stock of health at each point in time.

Although *some* variance in health-related behaviours may be readily understood within Grossman's original version of the demand-for-health model, the observed variance seems to be greater than what would be expected, solely taken the variability in physiologically determined, individually optimal level of activity into account. Furthermore, the observation that some health-related behaviours seem to be heavily distributed around two activity levels – high and low – cannot be explained within the original version of the model, except as a consequence of a corresponding distribution of behavioural-determining individual traits, which, in most cases, seems implausible. In this paper we develop a version of Grossman's demand-for-health model, modified in order to focus on individually optimal choices of health-related behaviour, distinguished by physiologically optimal activity levels.

Since its introduction, the demand-for-health model has been extended in various ways; incorporating uncertainty (Dardanoni and Wagstaff, 1987, 1990; Selden, 1993; Chang, 1996; Liljas, 1998, 2000), the family as producer of health (Jacobson, 2000; Bolin et al., 2001, 2002b), the employer as producer of health (Bolin et al., 2002c), social capital (Bolin et al., 2003), and healthy and unhealthy consumption (Forster, 2001). To our knowledge, however, the effect on the demand for health and health investments of the double-facetted nature of individual behaviours with physiologically determined optimal levels as regards health and negative or positive health effects, depending on the level of activity, has never been analysed.[2] While the emphasis of the paper is on extending theory, it is shown that such an analysis has important implications for the understanding of individual health-related behaviour and, hence, for designing and evaluating various health policies.

The structure of the rest of the paper is as follows. Next, we will present the model. After that, we will derive the optimality conditions. Following this, we will analyse the properties of the dynamic system in terms of steady-state and stability conditions. The final section contains a discussion and some conclusions.

2. THE MODEL

General structure

Our theoretical model takes its departure in the demand-for-health model developed by Michael Grossman (1972a, b). It differs from Grossman's original formulation mainly (1) by avoiding an

[2] Forster (2001) studied health-related effects of there being two types of consumption: good for health and bad for health. In both cases the effect on health is monotonic in consumption. In our case, the relationship between the amount of behaviour and its health effect is not monotonic. Grossman (1972a) examined the importance of joint production in the production of gross health investment. He argued that several goods are demanded as inputs into the production of commodities, other than health, that yield utility but may have adverse health effects.

explicit treatment of the individual's time allocation problem[3], and (2) by considering health-related behaviours that are not monotonic in their effect on health.

2.1 Preferences

We consider a version of the demand-for-health model, in continuous time, in which health at each point in time, H_t, is produced by the individual through a specific health behaviour, B_t, which influences health positively below a certain level, and negatively above that level; we assume that the smallest amount of the behaviour B_t is zero. The individual derives utility from his or her stock of health, H_t, from health-related behaviour, B_t, as well as from consumption unrelated to health, c_t. More specifically, we assume that preferences are additively separable, time additive, and concave in all arguments. In order to reduce complexity and, hence, to increase the capability of the model to generate unambiguous predictions, we also assume that the marginal utility of (the health-unrelated) consumption is constant. Formally, individual preferences are represented by the following quasi-linear utility function:[4]

$$U(H_t, B_t) = u^H(H_t) + u^B(B_t) + k \cdot c_t, (u_i^j > 0;\ u_{ii}^j < 0; u_{iii}^j = 0; i, j = H, B)^5 \qquad (1)$$

where k is the marginal utility of consumption.

The positive health effect produced by the behaviour, B_t, is partially offset by a natural depreciation – at rate $\delta\ (0 < \delta < 1)$ – of the existing stock of health capital.[6] For tractability of

[3] Instead, we utilise the individual's cost function pertaining to the allocation problem that he or she faces. This means that we – implicitly – assume that the individual has solved the time allocation problem.

[4] Quasi-linear utility functions have been extensively applied in economic analyses of the family, and related issues; see, for instance, Chang (2009, 2007); Chang and Weisman (2005); Konrad et al., (2002); Konrad and Lommerud (2000). Essentially, the quasi-linearity assumption means that the analysis is focused on the importance of relative prices, since there is no income effect for goods other than the linear-utility good.

[5] Throughout the paper, a subscript indicates a partial derivative (except when *t* indicates time dependence). Following established practice, the time derivative is denoted using a dot above the function or variable.

[6] Fundamentally, one may argue that the health-related behaviour is an input into the production of gross health investments and not the output of that production process. In an analysis of the composition of inputs into the production of gross health investments this distinction would be necessary. Here, however, our focus is on the effects of a specific behaviour having negatively U-shaped effect on health.

dynamic analysis we consider a model in which the rate of depreciation is time independent.[7] Further, we distinguish between the ability to produce (see below) the behaviour, B_t, and the rate at which the behaviour is transformed into gross health investments, I_t. Thus, the equation of motion for the stock of health capital is:

$$\dot{H} = B_t - \varphi \cdot (B_t - B^*)^2 - \delta \cdot H_t = I_t - \delta \cdot H_t, \qquad (2)$$

where B^* is the physiologically optimal amount of the behaviour ($B^* \geq 0$). Any deviation from the physiologically optimal level will reduce the positive impact or produce a negative contribution to health; the parameter φ captures the rate at which this negative contribution is dependent on the deviation from optimal behaviour ($\varphi > 1$).[8] Thus, a given level of the behaviour has both a direct effect on health and an indirect effect that results from deviating from the physiologically optimal level. Therefore, the maximum influence on health is $I_t^{max} = \frac{1}{4 \cdot \varphi} + B^*$, achieved when $B_t = \frac{1}{2 \cdot \varphi} + B^*$, since a small increase in B_t from B^*produces a smaller negative contribution to health than the positive marginal direct effect. The adverse health effect is bound below at $B_t = 0$, in which case the gross health investment is $-\varphi \cdot (B^*)^2$.

FIGURE 1 ABOUT HERE

Figure 1 illustrates the influence on gross health investment produced by each feasible amount of the behaviour. It facilitates our analysis to distinguish between two individual types: those that exert behaviour to the left and those to the right of $\frac{1}{2 \cdot \varphi} + B^*$. Notice, that a higher rate of punishment (higher φ) means that the marginal effect of each B_t on gross health investments for

[7] In this paper, we analyse time-paths and stability of equilibrium. This is considerably less difficult in autonomous systems, which require a time-independent rate of depreciation, or that total depreciation at each point in time is independent of the health stock. This is in contrast to Grossman (1972), Muurinen (1982), Wagstaff (1986), Liljas (1998), Jacobson (2000), and Bolin et al. (2001a), who all examined models with time-dependent rates of depreciation.

[8] This assumption is made for analytical convenience – it means that the steady-state loci can be constructed more decisively, without having to take into account behaviour when φ is close to zero.

the first type decreases, when $B_t \geq B^*$, and increases when $B_t < B^*$; while, for the latter, it always increases.

2.2 Production technology and cost function

For simplicity, we assume that the technology used for producing the behaviour is homogenous and exhibits constant returns to scale. Thus, the technology is formally represented by a production function that is homogenous, of degree 1 in the quantity of investment. Thus, the dual cost-of-behaviour function is homogenous of degree 1 with respect to the quantity of the behaviour, i.e., the cost of producing the behaviour is constant with respect to its quantity. Formally, the cost function is:

$$C(B_t) = \pi(p^B, w; E) \cdot B_t, \qquad (3)$$

where $\pi(p^B, w; E)$ is the one-unit cost of producing the behaviour, p^B is the composite price of market goods and services used in the production, w is the wage rate, and E is the level of knowledge. Thus, differences between individuals regarding their production efficiencies are comprised in w and E.

2.3 Constraints

For dynamic analysis tractability, we assume that there are no financial markets and, hence, that total spending at time t equals market income at time t.[9] Hence, with full income denoted y and the constant price of consumption p, the individual's budget constraint is:

$$p \cdot c_t = y(H_t) - \pi \cdot B_t. \qquad (4)$$

[9] In this way an autonomous dynamic model is obtained without introducing time as yet another state variable. Analysing properties of non-autonomous dynamic system is beyond what can be achieved using most economists' tool box. Autonomous systems are considerably more straightforward while allowing for important issues to be analysed. Dynamic models of health behaviour when there are no financial markets have been used by, for instance, Liljas (1998) and Forster (2001). Here, the absence of capital markets means that the only way that the individual can transfer resources between different points in time is by investing in health capital. We have made the assumption that preferences, prices, technology and the rate of depreciation are time invariant, which means that individual incentives for transferring resources between stages of the lifecycle do not comprise any timing-of-investment considerations. With time-varying prices for the inputs into production of health capital, this would be a major purpose for transferring resources in time.

We express full market income as a function of health. Time is allocated between three uses: sick time, τ_t^s, time allocated to the production of health-related behaviour, and time allocated to the market. At each point in time the individual chooses B_t, which completely determines H_t – via the health-capital equation of motion – and c_t as the difference between full income and cost of the health-related behaviour. Sick time is a function of health capital, $\tau^s(H_t)$ and, hence, full market income is:

$$y_t = w_t \cdot \left(\Omega - \tau^s(H_t)\right), \tag{5}$$

where Ω denotes total time. Time available for market work increases as the stock of health capital increases. This is manifested through the amount of time spent at being sick being inversely related to the stock of health capital, i.e., $\tau_H^s < 0$. We assume that the productivity of health in producing healthy time is diminishing, i.e., $\tau_{HH}^s > 0$. Thus, potential income is increasing and concave in health capital, since $y_H = -w \cdot \tau_H^s > 0$; and $y_{HH} = -w \cdot \tau_{HH}^s < 0$.

2.4 The individual's control problem

The intertemporal problem that faces the individual is to choose the time path of health capital in order to maximise his or her lifetime utility. We assume a fix end point for the individual's planning and, hence, if future utility is discounted at the rate ρ, the individual acts as if solving the following:[10]

$$\max_{B_t} \int_0^T e^{-\rho \cdot t} \cdot U(H_t, B_t, c_t)$$

subject to: $$\dot{H}_t = B_t - \varphi \cdot (B_t - B^*)^2 - \delta \cdot H_t.$$

[10] For convenience, we formulate the individual's optimisation problem as a vertical terminal line problem, which means that the terminal time is fixed, but the terminal state is free (Chiang, 1992, p.182). This is to be distinguished from a horizontal terminal line problem in which the terminal time, *T*, is free, and, hence, that the terminal state is restricted to $H_T \geq H_{min}$. The optimality conditions resulting from these two problems only differ regarding the transversality conditions. In the horizontal terminal line problem, optimal length of life is, implicitly, determined by the transversality conditions; see, for instance, Ehrlich and Chuma (1990). The individual's life-time optimisation problem was formulated as a vertical time line problem by, for instance, Bolin et al (2001; 2002b, c).

Transversality conditions are: $\lambda_T^H(T) \cdot (H_T - H_{min}) = 0;\ H_T > H_{min} > 0$, where H_{min} is the smallest permissible level of health.

3 RESULTS

3.1 Conditions for optimal paths of behaviour and health

The solution to the maximisation problem is achieved by applying optimal control theory. The maximum principle gives necessary and sufficient conditions for the optimal control of B_t, given the time path of the state variable H_t. The current-value Hamilton function for the maximisation problem is:

$$\boldsymbol{H} = U(H_t, B_t, c_t) + \lambda_t^H \cdot (B_t - \varphi \cdot (B_t - B^*)^2 - \delta \cdot H_t) \tag{6}$$

The maximum principle yields the following equations of motion.[11] First, for the stock of health:

$$\dot{\lambda}_t^H = -\frac{\partial \boldsymbol{H}}{\partial H_t} = -u_H^H + k \cdot \frac{w}{p} \cdot \tau_H^S + (\delta + \rho) \cdot \lambda_t^H = f(H_t) \tag{7}$$

For the optimal choice of the single control variable, B_t (consumption expenses equal the residual between income and health-behaviour expenses), the first-order condition is:

$$\frac{\partial \boldsymbol{H}}{\partial B_t} = u_B^B - k \cdot \frac{\pi}{p} + \lambda_t^H \cdot \big(1 - 2 \cdot \varphi(B_t - B^*)\big) = 0. \tag{8}$$

Interpretation

Let us begin by stating an immediate consequence: First, notice that since $u_H^H > 0$, we know that $\lambda_t^H > 0\ \forall\ t \in [0, T]$.[12] The implication of this – in our model – is that whether or not the amount of health-related behaviour that the individual chooses is below $\frac{1}{2 \cdot \varphi} + B^*$ is determined by equation (8): if $\frac{u_B^B}{k} > \frac{\pi}{p}$ (if the marginal rate of substitution between health behaviour and health-unrelated consumption is larger than the ratio between marginal costs) the individual will choose a B_t above $\frac{1}{2 \cdot \varphi} + B^*$ (and vice versa).

[11] The Hamiltonian function, $\boldsymbol{H}$, is jointly and strictly concave in (H, B). First, notice that $\boldsymbol{H}_{HB} = \boldsymbol{H}_{BH} = 0$. That leaves only the diagonal terms of the Hessian matrix. These are: $\boldsymbol{H}_{HH} = u_{HH}^H < 0$, and $\boldsymbol{H}_{BB} = u_{BB}^B - \lambda_t^H \cdot \varphi \cdot B_t < 0$, which means that $|\boldsymbol{H}| > 0$ and, hence, that the Hamiltonian is jointly and strictly concave in (H, B).

[12] See Caputo (2005), p 56.

After rearranging equation (7), the stock-of-health equilibrium condition reads:

$$u_H^H - k \cdot \frac{w}{p} \cdot \tau_H^S = (\delta + \rho - \frac{\dot{\lambda}_t^H}{\lambda_t^H}) \cdot \lambda_t^H. \qquad (7')$$

This condition requires that the current value of health capital (left-hand side), equals the instantaneous user cost of health capital (right-hand side). In order to derive an expression for optimal health-related behaviour, notice that the costate variable, λ_t^H, gives the value of health capital, which is independent of the health-related behaviour, B_t.

FIGURE 2 ABOUT HERE

From rearranging equation (8) we obtain:

$$v_t = \lambda_t^H = \frac{k \cdot \frac{\pi}{p} - u_B^B}{(1 - 2 \cdot \varphi(B_t - B^*))}. \qquad (8')$$

The right-hand side represents the net-marginal cost of the health-related behaviour B_t. In Figure 2, the value of health capital is drawn against health-behaviour, where the curves represent the net-marginal cost of health capital for two different values of λ_t^H.[13]

The intersections between the straight value-of-health-capital lines and the marginal-cost-of-health-investment schedule depict individually perceived optimal health-related behaviour. Since deviating positively or negatively, by equal amounts, from the physiologically optimal level results in equal amounts of health depreciation, there are two possible equilibria, symmetrically around the vertical line at $\frac{1}{2 \cdot \varphi} + B^*$. Individual preferences for the behaviour, B_t, determine at which side of the line the optimum is situated.

[13] It is straight-forward to check that the net marginal cost of health capital (MCH) curve, given by the right-hand side of (8'), is positively (negatively) inclined to the left (right) of $B_t = \frac{1}{2 \cdot \varphi} + B^*$, and that the slope is always increasing ($MCH_{BB} > 0$).

3.2 Steady states, dynamics and stability

In this section we will focus on the equilibrium properties. We begin by a description of possible steady states, defined as $\dot{H} = 0$ and $\dot{B} = 0$, followed by steady-state comparative statics. Then we describe the properties pertaining to the dynamics of the model.[14]

Steady state

The equation of motion of the stock of health is given by (2). Let:

$$\dot{H}_t = B_t - \varphi \cdot (B_t - B^*)^2 - \delta \cdot H_t = h(B_t, H_t). \quad (9)$$

In order to obtain the corresponding equation of motion for B_t, take the total time-derivatives of (8), which yields:

$$\dot{\lambda_t^H} \cdot \left(1 - 2 \cdot \varphi \cdot (B_t - B^*)\right) - (\lambda_t^H \cdot 2 \cdot \varphi - u_{BB}^B) \cdot \dot{B}_t = 0. \quad (10)$$

Solving for $\dot{B}_t$ yields:

$$\dot{B}_t = \frac{f \cdot \left(1 - 2 \cdot \varphi \cdot (B_t - B^*)\right)}{(\lambda_t^H \cdot 2 \cdot \varphi - u_{BB}^B)} = g(B_t, H_t), \quad (11)$$

where $f = \dot{\lambda_t^H} = -u_H^H + u_c^c \frac{w}{p} \cdot \tau_H^s + (\delta + \rho) \cdot \lambda_t^H$.

The dynamics of the system is described by equations (9) and (10), and the steady state loci can be found, using $h(B_t, H_t) = 0$ and $g(B_t, H_t) = 0$. Let us begin with the $\dot{H} = 0$ locus. Rearranging the equation $h(B_t, H_t) = 0$ yields:

$$H_t = \frac{1 + 4 \cdot B^* \cdot \varphi}{4 \cdot \delta \cdot \varphi} - \frac{\varphi}{\delta} \cdot \left(B_t - \frac{1/\varphi + 2B^*}{2}\right)^2. \quad (12)$$

FIGURE 3 ABOUT HERE

[14] Notice that with a steady-state concept that allows constant change in health or behaviour, we would obtain the same results.

Figure 3 illustrates the principal shapes of the steady-state loci. The graphs drawn in the figure are obtained as follows: equation (12) is a parabola having an inflexion point ($H^{max} = \frac{1+4\cdot B^*\cdot\varphi}{4\cdot\delta\cdot\varphi}$) at $B_t = \frac{1}{2\varphi} + B^*$, and intersections with the B-axis at $B_t = \frac{1}{2\varphi} + B^* \pm \sqrt{\frac{1+4B^*\varphi}{4\varphi^2}}$. Notice that if steady-state behaviour is $B_t = B^*$, equation (12) implies that the steady-state health stock is $H_t = \frac{B^*}{\delta}$. In the appendix, we show that the $\dot{B} = 0$ locus has one branch at each side of $B_t = \frac{1}{2\varphi} + B^*$, and that it is increasing in the left branch, decreasing in the right, and concave in both. From equation (11) it is clear that a third $\dot{B} = 0$ locus is $B_t = \frac{1}{2\varphi} + B^*$.

Steady-state comparative statics[15]

Steady-state comparative statics with respect to the four different variables φ, δ, E, and p^B can be derived, using the following equation system:[16]

$$h(B_t, H_t) = 0$$

$$g_s(B_t, H_t) = f \cdot \left(1 - 2 \cdot \varphi \cdot (B_t - B^*)\right) = 0$$

TABLE 1 ABOUT HERE

The comparative statics pertaining to H_t and B_t are derived in the appendix. Qualitative results are reported in Table 1. The table is partitioned into four subsections – one for the type of individual who exerts $B_t = \frac{1}{2\cdot\varphi} + B_t^*$ (type 1); two for the two types who exert less than $\frac{1}{2\cdot\varphi} + B_t^*$ of their health-related behaviour (types 2 and 3), and one for the type of individual who exerts $B_t > \frac{1}{2\cdot\varphi} + B_t^*$ (type 4). As regards the results for the type 1 individual, remember that the

[15] We do not consider the dynamics of the system in this section. Thus, we are not concerned with whether or not a small change in a parameter will result in a move from an initial steady state to a new one. Instead, we ask what steady-state values would have resulted, had a parameter been different.

[16] g_s is the numerator of equation (11).

inflexion-point value of health capital, at the $\dot{H} = 0$ locus, is $H^{max} = \frac{1+4\cdot B^*\cdot\varphi}{4\cdot\delta\cdot\varphi}$, which means that the amount of health capital is negatively related to the rate of depreciation (as expected).

The steady-state effects of changes in φ

An increase in the rate at which deviating from the physiologically optimal health-related behaviour is "punished" will have a negative impact on the demand for health for all types of individuals. However, this does not translate directly into health behaviour. The term $\lambda_t^H \cdot (1 - 2 \cdot \varphi(B_t - B^*))$ reflects the value of one unit of health behaviour. Now, for the type 2 individual ($B_t < \frac{1}{2\cdot\varphi} + B^*$) the change in this value, when the stock of health decreases, which increases the shadow price of health capital, and the rate at which deviating from $B^*$is punished increases, is ambiguous. This is so since the marginal effect on health which results from an additional unit of B decreases, making the total effect on $\lambda_t^H \cdot (1 - 2 \cdot \varphi(B_t - B^*))$ ambiguous, since $B_t^* < B_t < \frac{1}{2\cdot\varphi} + B_t^*$. For the type 3 individual, $B_t \leq B^*$, these forces work in the same direction, inducing an increase in the amount of B. For the fourth type, however, the value of one additional unit of the behaviour B decreases.

The steady-state effects of changes in δ

The effect on demand for health is negative for all types considered. The effect on health behaviour is ambiguous, however. This is so, since independently of whether B_t is smaller or larger than $\frac{1}{2\cdot\varphi} + B^*$ the reduction in demand for health and the effect of an increase in the rate of health capital depreciation, which decreases the supply of health capital, work in opposite directions.

The steady-state effects of changes in knowledge

The demand for health increases for types 2, 3 and 4, assuming that knowledge makes the individual a more efficient producer of the health-behaviour (and that the effect of the cost of own time is not dominating this efficiency effect). The increase in demand for health is met, however, differently by, on the one hand, types 2 and 3, and type 4 on the other: type 4 behaves $B_t > \frac{1}{2 \cdot \varphi} + B^*$ and will decrease his or her behaviour, while the other type will increase it. The reason for this is that type 2 and 3 have a positive effect on gross health investment of increasing the behaviour, B, while type 4 has to reduce the behaviour in order to increase gross health investments.

The steady-state effects of changes in price p^B

Increasing the price of the market good used for producing the health behavior leads to a reduction in the amount of health demanded, for type 2 and 3 individuals; while the demand for health increases for type 4 individuals. Thus, although the demand for the behavior, B_t, decreases for all types, this is beneficial for health for type 4 individuals only.

3.3 Stability and dynamics

In the previous section we examined how a steady state would change in response to a different value of an exogenous parameter. In this section we will ask: will the level of health and the health-related behavior ever reach an equilibrium? Moreover, what will happen, if an equilibrium is slightly disturbed – will the individual return to a steady state? These questions can be answered by examining the properties of the Jacobian matrix, i.e., the matrix of first-order derivatives of equations (10) and (11): $J = \begin{pmatrix} h_H & h_B \\ g_H & g_B \end{pmatrix}$.[17] We begin, below, by answering the latter question.

Stability of steady state

[17] This is achievable even though our dynamic system is not linear; see, for instance, Caputo (2005), p354.

In the appendix it is shown that a steady state for which $B_t \neq \frac{1}{2\cdot\varphi} + B_t^*$ is always a saddle point. This means that a small disturbance has a high probability of forcing the individual away from the steady state. More precisely, there is exactly one stable path (for each equilibrium) that leads to that particular steady-state equilibrium. All other paths lead away from it.

However, a steady state for which $B_t = \frac{1}{2\cdot\varphi} + B_t^*$ is (locally) stable (*viz.,* a sink; also shown in the appendix).

Dynamics

The dynamics of the model is expressed in equations $g(B_t, H_t)$ and $h(B_t, H_t)$. Taking the derivative of h and g, with respect to H, leaving out the arguments, results in $h_H = -\delta$, and $g_H = f_H \cdot \frac{(1-2\cdot\varphi\cdot(B-B^*))}{(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B)} > (<)\, 0$ when $B_t < (>)\, \frac{1}{2\cdot\varphi} + B_t^*$. This means that $\dot{H}_t > 0$ below the $\dot{H}_t = 0$ locus, and $\dot{H}_t < 0$, above it; and, further, that $\dot{B}_t > (<) 0$ below the $\dot{B}_t = 0$ locus when $B_t < (>)\, \frac{1}{2\cdot\varphi} + B^*$. Figure 4 illustrates the dynamic behaviour of the individual.

FIGURE 4 ABOUT HERE

Interpretation

Time paths in the two regions at each side of the vertical $\dot{B}_t = 0$ locus, bounded by the vertical locus, the non-vertical $\dot{B}_t = 0$ loci and the $\dot{H}_t = 0$ locus, will move closer to the sink equilibrium. This is so, since no integral curve can cross the non-vertical $\dot{B}_t = 0$ loci. Moreover, any initial position above the non-vertical $\dot{B}_t = 0$ loci but between the vertical locus and the stable-branch integral curves towards the saddle-point equilibrium, will move closer to the sink equilibrium. That is, for initial points sufficiently close to $\frac{1}{2\cdot\varphi} + B^*$, the movement will be towards the stable sink equilibrium.

For a point initially in one of the two regions bounded by the horizontal axis and the non-vertical $\dot{B}_t = 0$ loci and the $\dot{H}_t = 0$ locus, there are several possible time-paths. Health and behaviour may move towards the corresponding saddle-point equilibrium, or passing into one of the regions discussed above and, then, towards the stable equilibrium. It is also possible that health and behaviour will move towards the regions to the far left and right, respectively, in which case health will continue to deteriorate and health behaviour will continue to increase or decrease (no integral curve can cross the $\dot{H}_t = 0$ locus moving into either of the regions discussed, so, there is no return to a healthy trajectory).

4. DISCUSSION

By introducing – into the theoretical framework of the demand-for-health model – a non-monotonic influence of health-related behaviour on health as well as punishments with respect to health for deviations from the physiologically optimal level of behaviour, we were able to identify three types of equilibria, and characterise four types of individuals, using the relation between the exerted and the physiologically optimal levels of the health behaviour. For individuals in equilibrium – stationary health behaviour and health stock – our model predicts that individual health behaviour will be at one of three levels: low (type 3), high (type 4), or medium (type 1 and 2).

More specifically, the following predictions were derived for the different individual types: the type 1 individual exerts the level of health behaviour, which maximizes its influence on health. This level is not identical to the physiologically optimal one but slightly higher since a small deviation from the physiological optimal level will yield a positive marginal contribution to gross health investments. Changes in the rate of punishment, the depreciation rate, education, and commodity prices do not affect the health behaviour of the type 1 individual. Increases in the rate of punishment and in the depreciation rate will decrease the optimal health level, whereas

changes in education or commodity prices will leave the individually perceived optimal health level unchanged. The type 2 individual exerts a level of health behaviour, which is strictly below the level that maximizes its influence on health but strictly above the physiologically optimal level, while the type 3 individual exerts a level, which is below the physiologically optimal level. The results of changes in relevant parameters as above are qualitatively similar, except that a higher rate of punishment will decrease health behaviour for the type 2 individual and increase it for type 3. The type 4 individual prefers a level, which is strictly above the level, which maximizes its influence on health. Increases in the rate of punishment, the level of education, and commodity prices decreases the optimal levels of the health behaviour. Increases in the rate of punishment, the depreciation rate, and the level of education will decrease the optimal level of health.

Moreover, the dynamic analyses show that the "low" and "high" equilibria are saddle-point stable. This means that an individual, who is initially in one of these equilibria, and who is forced into disequilibrium by a small distrurbance, is not likely to return to the initial equilibrium. Instead, exerted levels of health behaviour will either diverge further away from equilibrium or approach the health-maximization level (type 1) of health behaviour. Notice, the model rules out any movement between "low" and "high" equilibria.

Building on the fundaments of the demand-for-health model, we developed a theoretical model that incorporates the double facetted nature of much health-related behaviour. Our results can be summarized as follows: (1) one important insight produced by the model is that individuals who differ regarding their valuation of health-related behaviour, but are otherwise identical, may still hold the same amounts of health capital, due to the non-monotonic health effect of the behaviour; (2) the non-monotonic effect of the health-related behaviour highlights that changes in exogenous parameters potentially affect different individuals not only quantitatively differently

but also *qualitatively* differently. This suggests that public policy efforts that do not take into account individual health-related behaviour may be contra productive; and (3) individuals who exert the health-related behaviour in a steady-state amount that equals the physiologically optimal level plus an amount inversely proportionate to φ (the rate at which a deviation in behaviour from the physiologically optimal level adversely influence health) are likely to not vary their behaviour over time. Further, this suggests that for health behaviours with a high corresponding φ, this steady state is close to the physiologically optimal level.

The preceding analysis implies that, in general, health-related public policy has to take into account that many behaviours may not be consistently bad or good for one's health but rather that the beneficial or detrimental effects depend on the individually chosen activity level of the behaviour in question. Thus, a first-best health policy would distinguish between individuals who exert low amounts and individuals who exert high amounts of a specific health-related behaviour. An illustrative and, perhaps, provocative, example is alcohol consumption. Given that there is a physiologically optimal level (strictly greater than zero) of alcohol consumption, taxing individuals that consume above a certain threshold level, and maybe *subsidizing* those who consume below the threshold, would increase population health, ceteris paribus. Obviously, the same qualitative conclusion can be made concerning all health-related behaviours that may influence health both positively and negatively. Thus, in order to affect such behaviours in a health-promoting way, policy-makers would, in principle, have to incorporate not only knowledge about the relationship between health and the target behaviour, but also a mechanism that is dependent on the type of individual that will be affected.

In practice, a "perfect" discrimination of individuals depending on type of preferences for health-related behaviours would not be possible due to information problems. Nevertheless, there seems to be sufficient information both for taking also undesired health effects into account,

when designing and evaluating various health policies. Thus, the lesson for policy-makers of this theoretical exercise would be to rely less on general public-health policies and more on tailor-made measures for specific target groups.

APPENDIX

We need to establish the value of f and the sign of $f's$ partial derivatives. In order to determine the value of f, solve for λ_t^H in equation (10), and take the time derivative. We have, first, $\lambda_t^H = \frac{k\cdot\frac{\pi}{p}-u_B^B}{\left(1-2\cdot\varphi(B_t-B^*)\right)}$, and then: $\dot{\lambda_t^H} = \frac{-u_{BB}^B\cdot\frac{dB}{dt}}{(1-2\cdot\varphi\cdot(\mathrm{B}-\mathrm{B}^*))} + \frac{(k\cdot\frac{\pi}{p}-u_B^B)\cdot 2\cdot\varphi\cdot\frac{dB}{dt}}{(1-2\cdot\varphi\cdot(B-B^*))^2} = f$. For a steady state we have $f = 0$ (which also follows from equation 14). Moreover, $f = 0$ along the $\dot{B} = 0$ locus.

In order to determine the sign of $f's$ partial derivatives we use $f = -u_H^H + u_c^c\frac{w}{p}\cdot\tau_H^s + (\delta+\rho)\cdot\lambda_t^H$:

$f_H = -u_{HH}^H + k\cdot\frac{w}{p}\cdot\tau_{HH}^s > 0$

$f_B = (\delta+\rho)\cdot\frac{-u_{BB}^B}{\left(1-2\cdot\varphi(B_t-B^*)\right)} + 2\cdot\varphi\cdot(\delta+\rho)\cdot\frac{k\cdot\frac{\pi}{p}-u_B^B}{\left(1-2\cdot\varphi(B_t-B^*)\right)^2} >< 0.$

$f_{BB} > 0$ (show calculations – immediate from the above)

Notice, that $f_B >< 0$ but $f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right) > 0$.

$f_\delta = \lambda_t^H$.

The steady state loci

The $\dot{B} = 0$ *loci*

In order to obtain the $\dot{B} = 0$ loci, notice that $g(H_t, B_t) = 0$ implies that:

$$g(H_t, B_t) = \dot{\lambda_t^H}\cdot\left(1-2\cdot\varphi\cdot(B_t-B^*)\right) = 0.$$

First, when $\left(1-2\cdot\varphi\cdot(B_t-B^*)\right)\neq 0$, applying the implicit function theorem on $g_s(H_t,B_t)$ yields:[18]

$$\frac{dH}{dB}=-\frac{f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)-f\cdot 2\cdot\varphi}{f_H\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)}=-\frac{f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)}{f_H\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)}=-\frac{f_B}{f_H}.$$

In order to infer the curvature of the $\dot{B}=0$ loci in this case, take the implicit derivative of $\frac{dH}{dB}$, which is simply:

$$\frac{d^2H}{dB^2}=-\frac{\left(f_{BB}+f_{BH}\cdot\frac{dH}{dB}\right)\cdot f_H-\left(f_{HH}\cdot\frac{dH}{dB}+f_{HB}\right)\cdot f_B}{{f_H}^2}=-\frac{f_{BB}}{f_H}<0,$$[19]

which means that the $\dot{B}$- locus is increasing in the left branch, and decreasing in the right, and concave. In the case when $\left(1-2\cdot\varphi\cdot(B_t-B^*)\right)=0$ the locus is the vertical line at $B_t=\frac{1}{2\cdot\varphi}+B^*$. The shape of the the $\dot{B}=0$ loci, together with the previously shown shape of the $\dot{H}=0$ locus, establish that there are three separate steady states.

Comparative statics

For a change in a parameter, x, the effects on steady-state levels of H_t and B_t can be obtained applying Cramer's rule to the following system:

$$\begin{pmatrix}h_H & h_B\\ g_{s_H} & g_{s_B}\end{pmatrix}\cdot\begin{pmatrix}\frac{dH}{dx}\\ \frac{dB}{dx}\end{pmatrix}=\begin{pmatrix}-h_x\\ -g_{s_x}\end{pmatrix}.$$

$J_s=\begin{pmatrix}h_H & h_B\\ g_{s_H} & g_{s_B}\end{pmatrix}$ is the Jacobian matrix of equations (9) and (11) evaluated in steady state.

Applying Cramer's rule in the case of a change in φ, we have: $\begin{pmatrix}\frac{dH}{d\varphi}\\ \frac{dB}{d\varphi}\end{pmatrix}=\begin{pmatrix}\frac{\begin{vmatrix}-h_\varphi & h_B\\ -g_{s_\varphi} & g_{s_B}\end{vmatrix}}{|J_s|}\\ \frac{\begin{vmatrix}-h_\varphi & h_B\\ -g_{s_\varphi} & g_{s_B}\end{vmatrix}}{|J_s|}\end{pmatrix}$.

The sign of $|J_s|$

$$|J_s|=\begin{vmatrix}-\delta & \left(1-2\cdot\varphi\cdot(B-B^*)\right)\\ f_H\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right) & f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)\end{vmatrix}$$

[18] $f=0$ along the $\dot{B}=0$ locus.
[19] $f_{HH}=f_{HB}=f_{BH}=0$.

Since $f_H > 0;\ \ f_B \cdot \left(1 - 2 \cdot \varphi \cdot (B - B^*)\right) > 0$, the determinant is always negative.

The steady state effects of φ

Applying Cramer's rule to the equations system above gives:

$$\frac{dH_t}{d\varphi} = \frac{(B_t-B^*)^2 \cdot g_{s_B} + (f_\varphi) \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) \cdot h_B}{|J_s|} = \frac{(B_t-B^*)^2 \cdot f_B \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) + f_\varphi \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right)^2}{|J_s|} =$$

$$\frac{(B_t-B^*)^2 \cdot f_B \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) + (\delta+\rho) \cdot \frac{2 \cdot (B_t-B^*)}{\left(1-2 \cdot \varphi (B_t-B^*)\right)} \cdot \lambda_t^H \cdot \left(1-2 \cdot \varphi (B_t-B^*)\right)^2}{|J_s|} =$$

$$\frac{(B_t-B^*)^2 \cdot f_B \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) + (\delta+\rho) \cdot 2 \cdot (B_t-B^*) \cdot (k \cdot \frac{\pi}{p} - u_B^B)}{|J_s|} < 0.$$

In order to see that $\frac{dH_t}{d\varphi} < 0$, rewriting the numerator, using the second term in f_B, which yields the condition: $(B_t - B^*)^2 \cdot 2 \cdot \varphi \cdot (\delta + \rho) \cdot \lambda_t^H > (\delta + \rho) \cdot 2 \cdot (B_t - B^*) \cdot \lambda_t^H \cdot \left(1 - 2 \cdot \varphi (B_t - B^*)\right) \implies (B_t - B^*) \cdot \varphi > \left(1 - 2 \cdot \varphi (B_t - B^*)\right) \implies B_t > \frac{1}{3 \cdot \varphi} + B^*$. This condition will always be fulfilled when $B_t > \frac{1}{2 \cdot \varphi} + B^*$. For $B^* \leq B_t \leq \frac{1}{2 \cdot \varphi} + B^*$, the conclusion follows immediately. For $B_t < B^*$, notice that $k \cdot \frac{\pi}{p} - u_B^B < 0$ (follows from equation 8), and the numerator is positive.

$$\frac{dB_t}{d\varphi} = \frac{-(B_t-B^*)^2 \cdot g_{s_H} - (f_\varphi) \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) \cdot h_H}{|J_s|} = \frac{-(B_t-B^*)^2 \cdot f_H \cdot \left(1-2 \cdot \varphi \cdot (B_t-B^*)\right) + \delta \cdot (\delta+\rho) \cdot 2 \cdot (B_t-B^*) \cdot \lambda_t^H}{|J_s|},$$

which is < 0 if $1 - 2 \cdot \varphi \cdot (B_t - B^*) < 0$, and > 0 if $B_t < B^*$.

Steady state effects of δ

Again, applying Cramer's rule to the equations system above (φ has been substituted for δ):

$$\frac{dH_t}{d\delta} = \frac{H_t \cdot f_B \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) + \lambda_t^H \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right)^2}{|J_s|} < 0$$

$$\frac{dB_t}{d\delta} = \frac{-H_t \cdot f_H \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right) + \delta \cdot \lambda_t^H \cdot \left(1-2 \cdot \varphi \cdot (B-B^*)\right)}{|J_s|} >< 0$$

Steady state effects of E

Proceeding as before gives:

$\frac{dH_t}{dE} = \frac{(\delta+\rho)\cdot\frac{k}{p}\cdot\pi_E}{|J_s|} \cdot \left(1 - 2 \cdot \varphi \cdot (B_t - B^*)\right) > (<)0$ if $1 - 2 \cdot \varphi \cdot (B_t - B^*) > (<)0$, and $\pi_E < (>)0$.

$\frac{dB_t}{dE} = \frac{(\delta+\rho)\cdot\frac{k}{p}\cdot\pi_E}{|J_s|} \cdot \delta < 0$ if $\pi_E < 0$.

Steady state effects of p^B

Finally, applying Cramer's rule the equations system above (substituting φ for p^B), gives:

$\frac{dH_t}{dp^B} = \frac{(\delta+\rho)\cdot\frac{k}{p}\cdot\pi_{p^B}}{|J_s|} \cdot \left(1 - 2 \cdot \varphi \cdot (B_t - B^*)\right) < (>)\ 0$ if $1 - 2 \cdot \varphi \cdot (B_t - B^*) > (<)0$.

$\frac{dB_t}{dp^B} = \frac{(\delta+\rho)\cdot\frac{k}{p}\cdot\pi_{p^B}}{|J_s|} \cdot \delta <\ 0$.

Dynamic analysis

The cases when $B_t \neq \frac{1}{2\cdot\varphi} + B^*$

The terms g_H, g_B of the Jacobian matrix J are obtained from $g(B_t, H_t) = \frac{f\cdot\left(1-2\cdot\varphi\cdot(B_t-B^*)\right)}{(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)}$.

Taking the derivative with respect to H gives:

$$g_H = \frac{\left(f_H\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)}{0}{(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)^2}.$$

Similarly, taking the derivative of $g(B_t, H_t)$ with respect to B yields:

$$g_B = \frac{\left(-f\cdot 2\cdot\varphi + f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)}{0}{(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)^2}.$$

For a steady state we have:

$$g_B = \frac{\left(f_B\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)}{0}{(\lambda_t^H\cdot 2\cdot\varphi - u_{BB}^B)^2}.$$

Together with h_H and h_B we are able to formulate:

$$|J| =$$

$$\delta \cdot \frac{\left(f_B \cdot \left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot\left(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B - u_{cc}^c \cdot \frac{\pi^2}{p}\right)}{0} - (1 - 2 \cdot \varphi \cdot (B_t - B^*)) \cdot$$

$$\frac{\left(f_H \cdot \left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot\left(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B\right)}{0} \Longrightarrow$$

$$|J| = -\delta \cdot \frac{f_B \cdot \left(\cdot\left(1-2\cdot\varphi\cdot(B-B^*)\right)\right)\cdot\left(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B\right)}{\left(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B\right)^2} - \frac{\left(f_H \cdot \left(1-2\cdot\varphi\cdot(B-B^*)\right)^2\right)\cdot\left(\lambda_t^H \cdot 2\cdot\varphi - u_{BB}^B\right)}{0} < 0.$$

$|J| < 0$ is a necessary and sufficient condition for a saddle point and, hence, the result follows.

The cases when $B_t = \frac{1}{2\cdot\varphi} + B^*$

In this case the determinant of the Jacobian matrix is zero, and the trace is strictly negative, i.e., one eigenvalue is zero and the other is strictly negative. This implies a sink.

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Figure 1. Illustration of the relationship between behaviour, *B*, and the amount of health investment, *I*. The parabola is the graph of the function $I_t = B_t - \varphi \cdot (B_t - B^*)^2$. The maximum influence on health attainable through the behaviour, *B*, is at the inflexion point of the parabola.

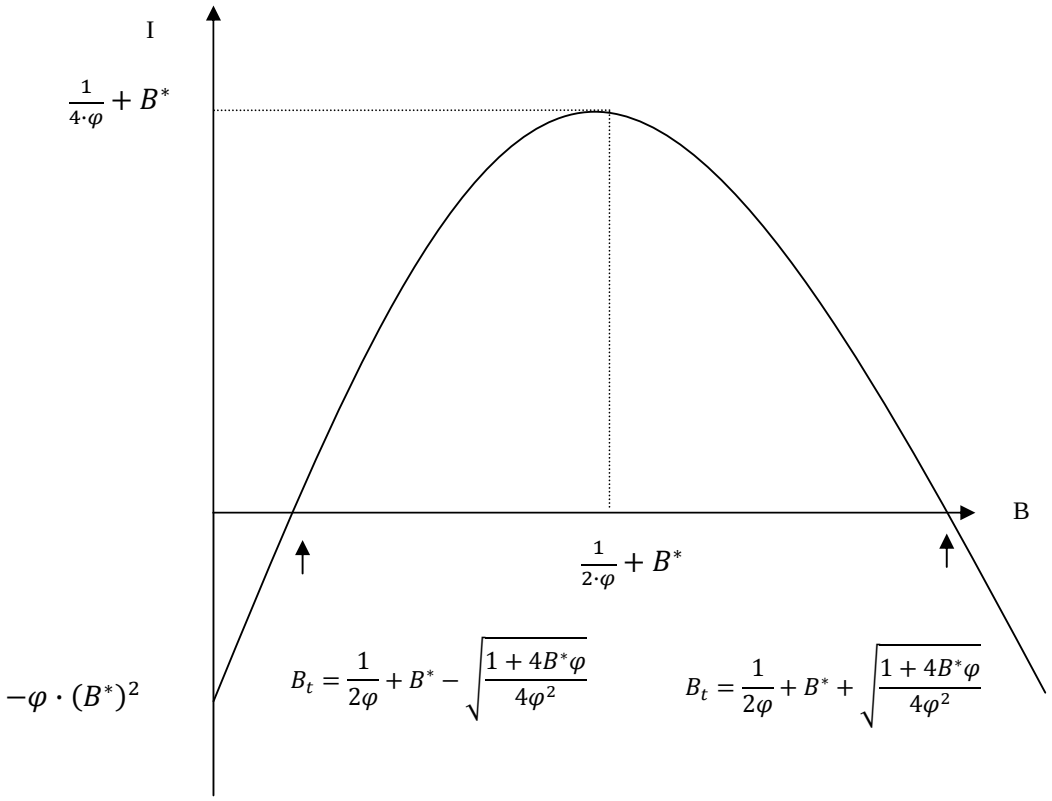

Figure 2. Illustration of the shadow price of health capital (the horizontal straight lines) and the marginal cost of health capital (the curves).

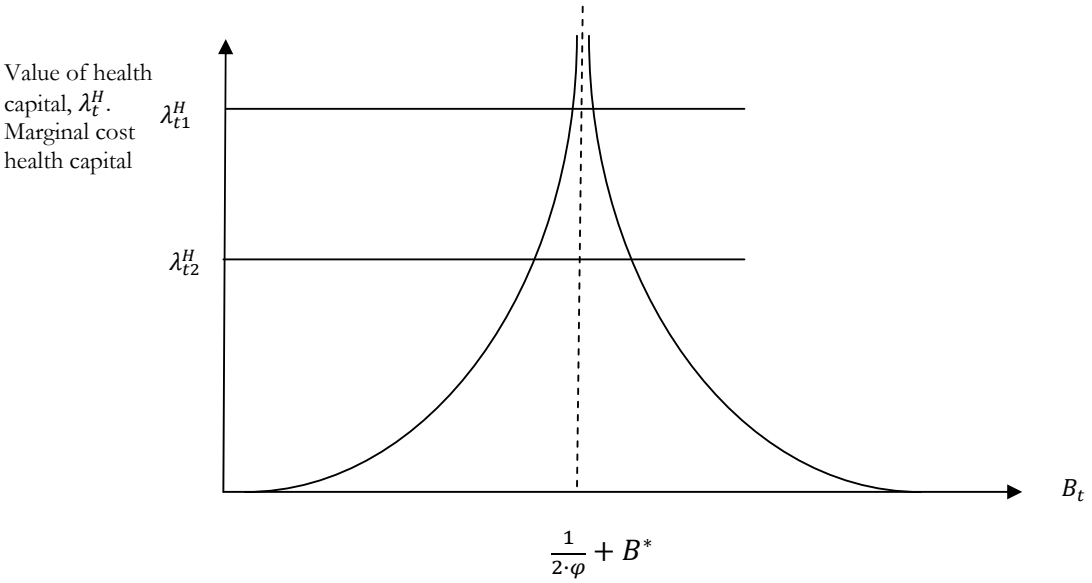

Figure 3. Illustration of the shapes of the steady-state loci and possible equilibria. The parabola shows the $\dot{H} = 0$ loci, the hyperbolas the $\dot{B} = 0$ loci.

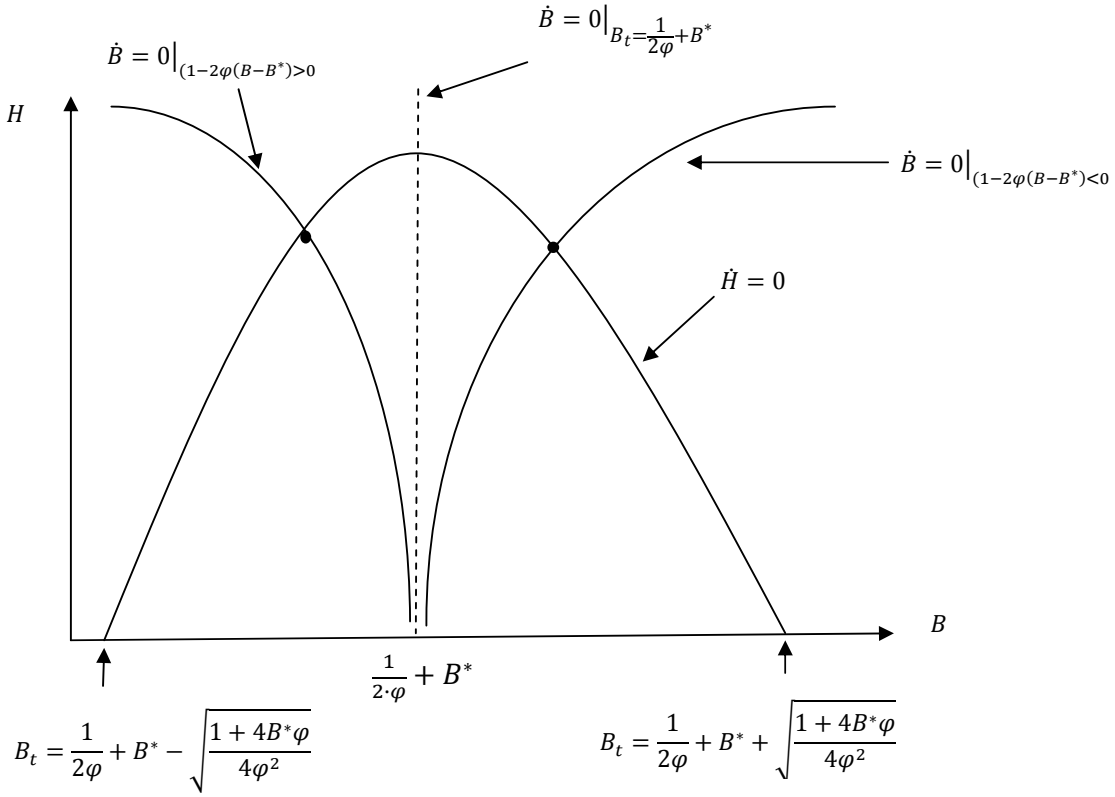

Table 1. Comparative static results regarding steady-state levels of health and behaviour. We assume that the effect of knowledge, E, on the one-unit cost, π, is negative. If the effect had been positive the results in the third raw would be reversed.

	Type 1: $B_t = \frac{1}{2 \cdot \varphi} + B_t^*$	Type 2: $B_t^* < B_t < \frac{1}{2 \cdot \varphi} + B_t^*$	Type 3: $B_t \leq B_t^*$	Type 4: $B_t > \frac{1}{2 \cdot \varphi} + B_t^*$
$(\frac{dH}{d\varphi}, \frac{dB}{d\varphi})$	$-,0$	$-\,,-/+$	$-\,,+$	$-\,,-$
$(\frac{dH}{d\delta}, \frac{dB}{d\delta})$	$-,0$	$-\,,-/+$	$-\,,-/+$	$-\,,-/+$
$(\frac{dH}{dE}, \frac{dB}{dE})$	$0,0$	$+\,,+$	$+\,,+$	$-\,,-$
$(\frac{dH}{dp^B}, \frac{dB}{dp^B})$	$0,0$	$-\,,-$	$-\,,-$	$+\,,-$

Figure 4. Illustration of time paths of health capital and health-related behaviour. Any integral curve that passes across a steady-state locus must indicate a zero rate of change in the locus variable. The quadrant is divided into 4 different section at each side of the vertical line. Each saddle-point equilibrium has one stable branch. The sink-equilibrium is locally stable.