AN ECONOMIC THEORY OF DEPRESSION AND ITS IMPACT ON HEALTH BEHAVIOR AND LONGEVITY

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An Economic Theory of Depression and its Impact on Health Behavior and Longevity[*]

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Abstract. In this paper, I introduce depression to the economics of human health and aging. Based on studies from happiness research, depression is conceptualized as a drastic loss of utility and value of life (life satisfaction) for unchanged fundamentals. The model is used to explain how untreated depression leads to unhealthy behavior and adverse health outcomes: depressed individuals are predicted to save less, invest less in their health, consume more unhealthy goods, and exercise less. As a result, they age faster and die earlier than non-depressed individuals. I calibrate the model for an average American and discus the socioeconomic gradient of health and depression as well as the hump-shaped association of antidepressant use with age. Delays in treatment for depression in young adulthood are predicted to have significant repercussions on late-life health outcomes and longevity.

Keywords: depression, depression therapy, health behavior, aging, longevity.
JEL: D15, D91, I10, I12.

The first thing that goes is happiness. You cannot gain pleasure from

anything. That's famously the cardinal symptom of major depression.

(Solomon, 2015).

1. Introduction

In health economic theory, depression has received relatively little attention. This is perhaps

surprising given the seriousness of the disease. According to the WHO (2017), more than 300

million people worldwide are living with depression, which makes depression the leading cause

of ill health and disability. Depressive disorders are estimated to be responsible of 50 million

years lived with disability, which accounts for 7.5% of the total number of years lived with

disability (WHO, 2017). In the US, 6.7 percent of all adults above age 18 have had at least

one major depressive episode in the past year (NSDUH, 2016). Similar population shares are

observed in other developed countries. In many developing countries, the prevalence is higher

since depression is associated with poverty (Lorant et al., 2003; Strulik, 2018b).

The most notable feature of depression is its strong and independent influence on life satis-

faction, even when controlling for poverty, unemployment, and ill health (Clark et al., 2017).

In fact, mental illness has been identified as the single largest determinant of misery, i.e. of

being in the bottom quarter of the population in terms of life satisfaction, more important than

physical health, income, and unemployment (Layard, 2013). The present research is inspired by

these observations from happiness studies. Building on this literature, I consider the empirical

observation of life satisfaction from surveys as a reasonable approximation of lifetime utility

(Frey and Stutzer, 2002; Clark et al., 2008; Stevenson and Wolfers, 2008).[1] I then conceptualize

depression as an exogenous drastic and sustained drop in experienced instantaneous utility and

thus also in lifetime utility (life satisfaction).[2]

Depression can be treated with drugs or psychotherapy but the efficacy of treatment is imper-

fect and individual-specific. For major depression, it has been estimated that pharmacological

treatment leads to recovery within four months in over 50% of cases. The probability of relapse

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[1]Most of the economics literature uses the terms happiness and life satisfaction interchangeably but it has been argued that the term happiness better describes the instantaneous component of subjective well-being while life satisfaction is the more appropriate measure of its evaluative, long-term component (Deaton, 2008; Stevenson and Wolfers, 2008).

[2]Actually, depression may be triggered by a life event and then persists after the cause has gone. In this paper, I ignore this refinement in the sequence of events and treat the loss of utility as being fully exogenous. This is a useful device in order to identify causality. In Strulik (2018b), I considered childbirth in poverty as one particular trigger of (maternal) depression.

into depression, however, remains high unless drugs are continued to be taken (Layard, 2013). Here, I model depression treatment as medical expenditure that dampens the loss of utility. Historically, treatment of mental health conditions had been covered less generously than physical illness by private insurance, a feature, which perhaps contributed to the under-treatment of mental health problems. In the U.S., it is estimated that one-third of the depressed population remains untreated (SAMHSA, 2002).

This paper focusses on the impact of depression on health behavior, aging, and longevity. Depression is associated with less healthy life styles in many dimensions. The rate of daily cigarette use, for example, is almost twice as high among adults with major depression compared to the general population (29.7 percent vs. 16 percent; NSDUH, 2006). Depressed individuals are also more likely to drink heavily and to use other addictive substances (NSDUH, 2006). Aside from increased alcohol and cigarette consumption, depression is also associated with inactivity, eating and sleeping problems, and a lack of treatment adherence for medical problems (Schulz et al., 2002). According to the model proposed in this paper, these behavioral changes are explained as a direct consequence of reduced life satisfaction. Facing low lifetime utility (life satisfaction), depressed individuals have less incentive to reduce unhealthy consumption, to save for health care expenditure in old age, and to invest in their health in monetary terms and physical exercise. As a result, their bodies age faster and their life ends prematurely. With respect to unhealthy consumption, for example, the model captures the notion that the high rate of smoking among depressed people does not reflect a particular preference for nicotine but a "general self-destructiveness among people for whom the future is only bleak" (Solomon, 2015).

It is well-known that (severe) depression is associated with an increased risk of suicide (ASS, 2014). In this paper, we assume that depression is mild enough such that depressed individuals do not kill themselves. Formally, we assume that the value of life of depressed individuals is low but still positive. This allows us to ignore mortality from suicide and to focus on the impact of depression on mortality through increased morbidity and the speed of aging. Many studies have documented an association between health and depression. One of the most robust findings is the relation between depression and cardiovascular disease (Schulz et al., 2002). With regard to the health deficit index, which is the measure of health used in the present study, Lohman et al. (2015) document a positive association between depression and the health deficit index

in late life, and Rao et al. (2016) find that depression significantly predicts increased health deficits, disability, and mortality among the elderly.[3] A rare study that estimated the impact of depression on life expectancy is provided by Zivin et al. (2012) who found that depressed male patients died about 4.8 years earlier than non-depressed patients (71.1 versus 75.9 years). Saint Onge et al. (2014) also documented an association of major depression and (non-suicide) mortality risk in the noninstitutionalized U.S. population and found that adjusting for health behaviors reduces the hazard rate between major depression and mortality by 17%.

In order to investigate health behavior and health outcomes, I integrate the theory of depression into the context of the health deficit model of Dalgaard and Strulik (2014) and extend the model with unhealthy consumption and physical exercise. The health deficit model has a foundation in gerontology and predicts that the pace of aging increases as people get older. Most importantly, health deficits are easily measured by a straightforward metric, the health deficit index (or frailty index; Mitnitski et al., 2002a,b, 2005; Rockwood et al., 2007; Harttgen et al., 2013). This allows for a calibration of the model with real data such that the model can be used to quantitatively address lifetime health issues of depression. This feature distinguishes the health deficit approach from the earlier paradigm in health economics, the accumulation of health capital (Grossman, 1972), which considered a latent variable that remained unknown in medical science. Conceptually, the main distinctive feature of the health deficit approach is the implication that unhealthy people age faster. The health capital model, in contrast, assumes that healthy people age faster (lose more health capital through depreciation). Among other things, this counterfactual assumption implies that the health capital model predicts that early-life health shocks matter little for late-life health outcomes (for a critique, see Almond and Currie, 2011) whereas the health deficit model predicts the opposite (Dalgaard et al., 2017). In the current context, this distinction matters because the health deficit model highlights the importance of early diagnosis and treatment of mental illness, a feature that would be impossible to observe within the health capital paradigm.[4]

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[3]The health deficit index (or frailty index) computes the percentage of health deficits present in a person from a long list of potential health deficits, see Mitnitski et al. (2002a,b; 2005); and Rockwood and Mitnitski (2006, 2007).

[4] Earlier studies using the health deficit model were concerned with the long-term evolution of the age at retirement (Dalgaard and Strulik, 2017), adaptation to poor health (Schuenemann et al., 2017a), the gender gap in mortality (Schuenemann et al., 2017b), and the education gradient (Strulik, 2018a).

The modeling of depression treatment implements (for the first time) the suggestion to analytically discuss the use of antidepressants as a new form of consumption that lies in the currently grey area between medication and consumer goods. (Katolik and Oswald, 2017, Blanchflower and Oswald, 2016). It is related to a small literature arguing in favor of a backward causality from happiness to (economic) behavior. Guven (2012) identifies the backward causality by instrumenting happiness with unexpected sunshine and finds that happier people save more, have a lower marginal propensity to consume, and expect a longer life. Similarly, De Neve et al. (2013) find that high subjective wellbeing is associated with healthier eating, reduced likelihood of smoking, more exercise, and a longer life.

So far, two alternative and potentially complementing theories of depression have been suggested. De Quidt and Haushofer (2016) propose to model depression symptoms as the consequence of downward shocks in the belief about the return to effort in a model where a decision-maker chooses labor effort, non-food consumption, food consumption, and sleep. Health issues and their consequences on life cycle-health and longevity are not addressed. Strulik (2018b) proposes an overlapping generations model to address the intergenerational transmission of depression and shows that this gene-environment interaction may generate a poverty trap. The study focused on maternal depression modeled in reduced-form as increased present-bias, which leads to less child investments of depressed mothers. Health behavior and its life cycle implications are not addressed.

This paper is organized as follows. In the next section, I set up a life-cycle model of depression, health behavior, health outcomes, and longevity; and, using comparative statics, I derive analytical results for the use of antidepressants at the intensive and extensive margins. The full dynamic model can only be analyzed numerically. In Section 3, I calibrate the model for a reference American (a 20-year-old male in the year 2000) and in Section 4, I assess the quantitative impact of depression and depression treatment on life-cycle behavior, health, and longevity. In Section 5, I consider applications and extensions of the basic model with regard to depression and the income gradient of health and longevity, feedback effects of depression on labor market outcomes (retirement and lifetime earnings), the impact of delayed diagnosis and treatment on health outcomes, and a u-shaped age-pattern of depression in a population with idiosyncratic susceptibility to depression. Section 6 concludes with suggestions for future research.

2. A Theory of Depression, Health Behavior, and Health Outcomes

2.1. **Utility.** Consider an individual who derives utility from the consumption of health-neutral goods c and unhealthy goods u and whose utility declines in the number of health deficits D. The (sub-)utility function $U(c, u, D)$ exhibits, as usual, declining marginal utility from consumption, $\partial U / \partial j > 0$, $\partial^2 / \partial j^2 < 0$ for $j = c, u$. Health deficits reduce utility and, following Finkelstein et al. (2013), we assume that health deficits reduce the marginal utility from consumption, $\partial^2 U / (\partial c \partial D) < 0$. Additionally, individuals may engage in healthy behavior and the exerted health effort x, which could be thought of as physical exercise, has a utility cost (e.g. in terms of leisure). The disutility from effort increases in the number of health deficits present in a person. For simplicity, we assume that the subutility function for the cost of health effort $f(x, D)$ is separable from the utility from consumption and has positive first derivatives and non-positive second derivatives.

As motivated in the Introduction, depression can be best conceptualized as a downward-shifter of the utility function. The severity of the depression is measured by $\delta \geq 0$. In this paper, we focus on depression that is mild enough such that utility remains positive and depressed individuals are not induced to kill themselves. Instead, we investigate the effects of depression on longevity through induced unhealthy behavior and (in the extended model) through the side-effects of medication and addiction. When the depression is treated, the negative utility shock becomes less severe. A unit of treatment m could be conceptualized as the use of prescription antidepressants or therapy. The efficacy of the treatment is denoted by $\eta \geq 0$. Together, this means that the utility of depressed individuals is reduced by $\delta g(m, \eta)$ with $\partial g / \partial \eta < 0$ and with declining returns of treatment intensity, $\partial g / \partial m < 0$, $\partial^2 g / \partial m^2 > 0$. Moreover, an extra unit of treatment reduces depression to a larger degree when treatment is effective, i.e. $\partial^2 g / \partial \eta \partial m < 0$. Reasonable albeit not decisive further assumptions are that $g(0, \eta) = 1$ and $\lim g_{m \to \infty} = 0$. The first assumption means that untreated individuals experience the full power of depression δ. The second assumption rules out that non-depressed individuals take antidepressants to further boost their happiness. Altogether, this means that lifetime utility is given by

$$V = \int_0^T \left[U(D, c, u) - f(x, D) - \delta g(m, \eta) \right] \mathrm{e}^{-\rho t} \mathrm{d}t, \tag{1}$$

in which t is age, T is the age at death, and ρ is the time preference rate. Here, we focus on a deterministic model in which death occurs when an upper limit of health deficits $\bar{D}$ has

been accumulated. As shown in Strulik (2015a) and Schuenemann et al. (2017b), stochastic versions of the health deficit model in which the probability of death depends positively on health deficits add more realism at the price of higher complexity but provide little extra insight into life-cycle choices and outcomes. Since the present model is already quite complex, for the sake of simplicity, we abstract from stochastic death.

2.2. **Health.** The state of health is measured by a health deficit index D, defined as the share of health deficits present in a person (from a long list of potential health deficits). As shown by Mitnitski et al. (2002), Harttgen et al. (2013), and Abeliansky and Strulik (2017), health deficits are accumulated in a quasi-exponential way with increasing age. As in Dalgaard and Strulik (2014) and Schuenemann et al. (2017a), we assume that the "natural" increase of health deficits can be slowed down by health investment h and accelerated by unhealthy consumption u. Additionally, we assume that health effort x slows down the accumulation of health deficits. In short, health deficits accumulate as

$$\dot{D} = \mu\left[D - Ah^{\gamma} + Bu^{\omega} - Ex^{\kappa} - a\right],\qquad(2)$$

in which μ is the "natural" force of aging. The parameters A and γ measure the efficacy of health technology, the parameters B and ω measure the degree of unhealthy consumption, and the parameters E and κ measure the impact of health effort. We assume decreasing returns of health investment and health effort, $0 < \gamma, \kappa < 1$ while marginal health costs are increasing in unhealthy consumption, $\omega > 1$.

2.3. **Wealth.** Individuals receive an income flow w, conceptualized as net wage income until retirement, and as pension income afterwards. Income is used for consumption, saving at interest rate r, and health expenditure. In order to introduce a primitive health care system, we assume that a share ϕ of health investments and a share ϕ_m of depression treatments are paid out of pocket. Let the prices of unhealthy consumption, health investment, and depression treatment be denoted by q, p, and p_m. Then, wealth k accumulates as

$$\dot{k} = w + rk - c - qu - p\phi h - p_m\phi_m m.\qquad(3)$$

2.4. **Solution.** Individuals chose consumption (c and u), health expenditure h, health effort x, and depression treatment m in order to maximize lifetime utility (1) subject to constraints (2)

and (3), the initial states of health $D(0)$ and wealth $k(0)$, the boundary conditions $D(T) = \bar{D}$ and $k(T) = \bar{k}$, and non-negativity constraints on all choice variables. However, it will turn out that only for unhealthy consumption and depression treatment, the non-negativity constraint becomes occasionally binding whereas the other choices are always positive. The associated Hamiltonian is given by

$$H = U(D, c, u) - f(x, D) - \delta g(m, \eta)$$
$$+ \lambda_D \mu \left[D - Ah^\gamma + Bu^\omega - Ex^\kappa - a \right] + \lambda_k \left[w + rk - c - p\phi h - p_m \phi_m m \right]. \tag{4}$$

The first order conditions for a maximum are:

$$\frac{\partial U(D, c, u)}{\partial c} = \lambda_k \tag{5}$$

$$\frac{\partial U(D, c, u)}{\partial u} \leq \lambda_k q - \lambda_D \mu B \omega u^{\omega-1} \quad \text{with} = \text{for } u > 0 \tag{6}$$

$$-\lambda_D \mu \gamma A^{\gamma-1} = \lambda_k \phi p \tag{7}$$

$$-\lambda_D \mu \kappa E x^{\kappa-1} = \frac{\partial f}{\partial x} \tag{8}$$

$$-\delta \frac{\partial g}{\partial m} \leq \lambda_k \phi_m p_m \quad \text{with} = \text{for } m > 0. \tag{9}$$

I have aligned the first order conditions such that the left-hand side always shows the marginal benefit and the right-hand side shows the marginal cost. Condition (5) requires that the marginal utility from consumption of health neutral goods equals the marginal cost in terms of foregone future utility, measured by the shadow price of capital. Condition (6) requires that the marginal utility from unhealthy consumption is not larger than the marginal cost, which consists of implied forgone future utility $\lambda_k q$ and the marginal impact of unhealthy consumption on health $\mu B \omega u^{\omega-1}$, evaluated by the shadow price of health deficits. Notice that future health deficits are bad such that λ_D is negative. Condition (7) requires that the marginal return from health investment $\mu \gamma A^{\gamma-1}$ evaluated with the shadow price of health deficits equals the marginal cost in terms of foregone marginal utility from consumption. Condition (8) requires that the marginal return from health effort equals the marginal utility cost of health effort.

Finally, condition (9) requires that the marginal return of depression treatment is not larger than the marginal cost of treatment. To observe this, recall that treatment reduces depression, i.e. that $\partial g / \partial m < 0$. For the treated, marginal benefits equal marginal costs. When marginal

costs exceed marginal benefits, individuals remain untreated. Inserting (5) into (9), we obtain the depression treatment constraint:

$$0 \leq G \equiv \frac{\partial U(D, c, u)}{\partial c} \phi_m p_m + \delta \frac{\partial g(m, eta)}{\partial m}. \tag{10}$$

From the inspection of (10), we obtain a first result:

PROPOSITION 1 (Intensive Margin). *The treatment intensity of depression m is increasing in the level of consumption c (and thus income) of the patient, and in the number of health deficits of the patient. It declines in the price p_m of the treatment and the out-of-pocket share ϕ_m.*

For the proof, I apply the implicit function theorem on (10) evaluated with equality. For example, we have for price p_m, $\mathrm{d}m/\mathrm{d}p_m = -\left(\phi_m \partial U(D, c, u)/\partial c\right) / \left(\delta \partial^2 g/\partial m^2\right) < 0$, and likewise for ϕ_m. The comparative static for health deficits uses the assumption $\partial^2 U/(\partial c \partial D) < 0$ and the comparative static for consumption uses the assumption of declining marginal utility from consumption $\partial^2 U/(\partial c^2) < 0$. The intuition for the result with respect to consumption (income) is that individuals who experience little extra utility from spending an extra dollar on consumption are more inclined to spend income on depression treatment. The marginal utility from consumption is low when the level of consumption is high or when many health deficits are present. As long as consumption goods are normal (which we assume), richer individuals will consume more. In other words, poor individuals have other pressing needs and are therefore less inclined to spend much money on depression treatment, in particular, if treatment prices are high and the out-of-pocket share is large (i.e. if they are uninsured).

PROPOSITION 2 (Extensive Margin). *Depression remains untreated if the treatment is sufficiently inefficient (η is low), if the out-of-pocket share ϕ_m is sufficiently large, or if the level of consumption (income) of the patient is sufficiently low.*

The proof with respect to η shows that G is increasing in η since $\partial^2 g/\partial m \partial \eta > 0$, which makes it less likely that (10) binds with equality when η is low. The other comparative statics are obtained similarly. These comparative statics, however, do not provide any information about the impact of depression on health behavior and longevity. For this, we need the full solution of the model, which also requires the costate equations and boundary conditions to be fulfilled.

The costate equations are given by

$$\lambda_k r = \lambda_k \rho - \dot{\lambda}_k \qquad (11)$$

$$\frac{\partial U(c,u,D)}{\partial D} - \frac{\partial f(x,D)}{\partial D} + \lambda_D \mu = \lambda_D \rho - \dot{\lambda}_D \qquad (12)$$

Aside from the boundary conditions on k and D, the optimal solution of the free terminal time problem also fulfills $H(T) = 0$. Inspection of (4) shows that the Hamiltonian assumes a large value, ceteris paribus, when utility is large, i.e. for individuals who are healthy, wealthy, and not depressed. The fact that instantaneous utility is strictly concave in the level of consumption while lifetime utility is (aside from the health effects) linear in instantaneous utility motivates the pursuit for a long life. Individuals, unconditionally prefer to extend their consumption over a longer time period against consuming more right now. The pursuit for a long life, however, comes also at a cost in terms of health investments, health effort, and the eschewal of unhealthy consumption. Rich individuals experience lower marginal utility from (even more) consumption and have more funds to finance a long life. They are thus predicted to live longer. These mechanism were the main subject of investigation in Dalgaard and Strulik (2014). The novelty here is that depression drives down the level of instantaneous utility (experienced happiness). This means that it reduces the desire for a long life which in turn reduces the incentive to invest time and money in health and increases the incentive to indulge in unhealthy consumption.

In order to explore the impact of depression on health and health behavior we need the full solution of the model, i.e. the dynamic life cycle trajectories implied by the first order conditions and boundary conditions. This can be achieved only numerically and the first step is to assume functional forms for the sub-utility functions. For simplicity, we conceptualize total consumption $\tilde{c}$ as a convex combination of the consumption of health neutral goods and unhealthy goods, $\tilde{c} = \theta c + (1-\theta) u$, $0 < \theta < 1$. One advantage of such a simple additive sub-utility function is that it allows for a preemptively high price at which households abstain from unhealthy consumption (see below). We assume that utility from total consumption is iso-elastic. Controlling for health deficits, utility is given by $(\tilde{c}^{1-\sigma} - 1)/(1-\sigma)$, in which $1/\sigma$ is the elasticity of intertemporal substitution. Following Finkelstein et al. (2013), we treat the state of health as a shifter of the utility function of consumption such that both utility and marginal utility of consumption are negatively affected by bad health. Specifically, we assume that health adjusted utility is given by $(D/D_0)^{-\epsilon} \cdot (c^{1-\sigma} - 1)/(1-\sigma)$. The parameter ϵ controls the amount by which an additional

health deficit will shift the utility function downwards. The normalizing constant D_0 implies that at the state of best health (which is the initial level of health deficits), utility is unaffected by health.

The disutility from health effort (physical exercise) is formalized as $f(x, D) = (D/D_0)^\chi \tau x$, with $\tau > 0$ and $\chi > 1$. In order to obtain a closed form solution, we assume that utility is linear in health effort x. Disutility from effort is potentially increasing in health deficits ($\chi \geq 0$). Since $D(t) \geq D_0$, health effort becomes increasingly painful with the accumulation of health deficits for $\chi > 1$. The parameter τ controls how much the individual, in general, dislikes health effort.

Finally, we assume a simple exponential function for depression treatment $g(m, \eta) = e^{-\eta m}$. This function fulfills the assumption of decreasing returns. It implies that non-depressed individuals will not demand antidepressants and that non-treated individuals experience the full power of depression. In short, lifetime utility (1) is parameterized as

$$V = \int_0^\infty \left(\frac{D}{D_0}\right)^{-\epsilon} \frac{[\theta c + (1-\theta)u]^{1-\sigma} - 1}{1 - \sigma} - \left(\frac{D}{D_0}\right)^\chi \tau x - \delta e^{-\eta m} dt. \tag{13}$$

From the first order conditions, we obtain closed form solutions for u, x, and m (see the Appendix for details on the derivation of these results). The solution for unhealthy consumption is displayed in (14).

$$u = \begin{cases} \left\{ \left[\frac{1-\theta}{\theta} - q\right] \frac{A\gamma h^{\gamma-1}}{B\omega\phi p} \right\}^{\frac{1}{\omega-1}} & \text{for } q < \frac{1-\theta}{\theta} \\ 0 & \text{otherwise.} \end{cases} \tag{14}$$

For unhealthy consumption to prevail, the relative utility weight of unhealthy goods, $(1-\theta)/\theta$, has to exceed the price q. If unhealthy consumption exists, then condition (14) predicts that its extent is large if the resulting health damage is low (B is low), if medical efficiency in repairing damage is large (A is large), or if the price of health goods p is low.

The solution for health effort is displayed in (15).

$$x = \left[\left(\frac{D}{D_0}\right)^{-(\epsilon+\chi)} \frac{(\tilde{c})^{-\sigma} \theta \phi p \kappa E}{\tau \mu A \gamma h^{\gamma-1}} \right]^{\frac{1}{1-\kappa}}. \tag{15}$$

Health effort (physical exercise) is high for healthy persons (with low D) and when the health gain E is large. It is low when τ is large, i.e. when exercising provides much disutility. Physical exercise is also high when the marginal return of health investments $\gamma A h^{\gamma-1}/p$ is low. This outcome is intuitive because it makes sense to exercise more in order to reduce the accumulation

of health deficits when there is little return to further monetary investments on the state of health.

The solution for depression treatment is displayed in (16). The comparative statics stated in general form in Proposition 1 and 2 are easily verified by inspection of (16).

$$m = \max \left\{ 0, \ \frac{1}{\eta} \log \left[\frac{\delta \eta (D/D_0)^\epsilon \tilde{c}^\sigma}{p_m \phi_m \theta} \right] \right\}. \tag{16}$$

Finally, the optimal solution is characterized by two dynamic equations for consumption and health expenditure (see Appendix for details on the derivation):

$$\frac{\dot{\tilde{c}}}{\tilde{c}} = \frac{1}{\sigma} \left[r - \rho - \epsilon \left(\frac{\dot{D}}{D} \right) \right], \tag{17}$$

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma} \left\{ r - \mu - \left[\epsilon \frac{\tilde{c}^{1-\sigma} - 1}{1-\sigma} + x\tau \left(\frac{D}{D_0} \right)^{\chi+\epsilon} \right] \frac{\mu A \gamma h^{\gamma-1} \tilde{c}^\sigma}{p \phi D} \right\}. \tag{18}$$

Equation (17) shows the standard Euler equation for consumption adjusted by health deficits, Intuitively, the accumulation of health deficits drives down the incentive to spend on consumption in old age, and thus lowers the slope of the lifetime consumption path (which may become hump-shaped; see Strulik, 2015b). Equation (18) shows the health-Euler equation. When health does not show up in the utility function ($\epsilon = \tau = 0$), health expenditure becomes steeper with rising return on wealth and declining force of mortality (i.e. with greater incentive to save for late-life health expenditure). The presence of health deficits in the utility function decreases the slope of the health expenditure path through its impact on the utility from consumption and on the disutility from exerting health effort.

3. CALIBRATION

The model is calibrated, as in Dalgaard and Strulik (2014), for an average white 20-year-old American male in the year 2000. From Mitnitski et al. (2002a), we take the estimate of for the rate of aging, $\mu = 0.043$, and from Dalgaard and Strulik (2014), we take $r = \rho = 0.06$. From age 20 to 65, we set $w = 35,320$, which is the average annual pay for workers in the year 2000 (BLS, 2011). For older individuals, we set $w = 0.45 \cdot 35,320$ using an average replacement rate of 0.45 from the OECD (2016). We assume that individuals neither bequeaths nor inherit any wealth, $k_0 = \bar{k} = 0$. We set the out-of-pocket expenditure share to $\phi = 0.28$ for all ages. This proxy somewhat overestimates the out-of-pocket share of the elderly and underestimates it

somewhat for the young (Machlin and Carper, 2014). For the benchmark run, we set $\phi_m = \phi$, assuming no discrimination of payments for depression treatment. Furthermore, we normalize prices $p = q = 1$. This is an interesting benchmark case because it eliminates any price channel through which poor individuals may have an incentive to consume more unhealthy goods or spend less on general health care.

The calibration of the effect of health on utility is related to Finkelstein et al. (2013) who estimate that a one-standard-deviation increase in the number of chronic diseases is associated with a 11% decline in the marginal utility of consumption (with a 95% confidence band from 2.7% to 16.8%). Finkelstein et al. focus on individuals above 50 and apply a smaller set of more severe health deficits compared to this study. Here, we calibrate the frailty index of Mitnitski et al. (2002a), which also contains relatively mild health deficits like farsightedness and incontinence. We thus consider, for the benchmark case, a smaller impact of health deficits by setting $\epsilon = 0.06$. This means that an unexpected increase in health deficits from D_0 by one standard deviation reduces the marginal utility from consumption by 4 percent.

Since most of the available empirical literature on consumption of unhealthy goods is on cigarettes and tobacco, we conceptualize u as smoking. On average, Americans spent \$ 319 on cigarettes in the year 2000 (BLS, 2002). The Smoking intensity declines with age, at least from middle age onwards (Holford et al., 2014). Empirically, there is some variation in the estimates of years of life lost due to smoking, ranging from 2.5 years (Preston et al., 2010) to 10 years (Jha et al., 2013). Here, we try to fit an intermediate value of 4.3 years, estimated by a study that takes selection into smoking into account, see Darden et al., 2015.

To measure physical exercise, we use the metric of metabolic equivalents (METs) defined as the energy cost of a given physical activity divided by energy expenditure at rest. This metric allows for the aggregation of different physical activities like walking, playing sports, gardening, etc., and to compare them across individuals and ages. The average American in Moore et al.'s (2012) sample spends about 1.14 MET per day (8 MET per week) on physical exercise, an equivalent of about 23 minutes of brisk walking per day. Moore et al. estimate that this exercise increases life expectancy by about 3 years. They also document strongly decreasing returns from physical exercise, i.e. large gains for departure from inactivity and very small gains for excessive exercise. Studies from the UK (Townsend et al., 2015) and Canada (Statistics Canada, 2007) suggest that the intensity of exercise declines by about a factor of 2 from age 35 to age 70.

Assuming that British and Canadian men are, in this regard, sufficiently similar to Americans, we try to match their age gradient of physical activity.

To summarize, the remaining parameters of the model for a non-depressed individual, that is A, a, B, E, γ, χ, θ, σ, τ, and ω, are estimated to fit the following stylized facts: a 20-year-old U.S. American male in the year 2000 has a life expectancy of 55.5 years (dying at age 75.5; NVSS, 2012), total spending on health, on average, equals 13 percent of his lifetime income (the health expenditure share of GDP in the U.S. in the year 2000; World Bank, 2015); as the individual ages, health expenditure increases by about two percent per year (Dalgaard and Strulik, 2014); the individual spends, on average, \$ 300 per year on smoking; smoking expenditure declines by 50 percent from age 30 to age 60; smoking costs 4.3 years of life; the individual spends $x = 1.14$ METs per day on exercise, which allows him to live 3 years longer than he would in total inactivity; and such that we map – with successive out-of-sample predictions – the age gradient as well as the marginal return of physical exercise for alternative x, as estimated by Moore et al. (2012). This leads to the estimates $A = .00013$, $a = 0.0182$, $B = 9.5 \cdot 10^{-7}$, $E = 1.06 \cdot 10^{-3}$, $\gamma = .32$, $\chi = 1.15$, $\kappa = 0.1$, $\theta = .073$, $\sigma = 1.08$, $\tau = 0.0085$, and $\omega = 1.20$. While most of these parameters are latent, there exist many studies on the size of σ. The estimated value accords well with studies suggesting that the intertemporal elasticity of substitution is close to unity (Chetty, 2006).

As another plausibility check of the calibration, we compute the value of life (VOL) of the Reference American and compare it with previous estimates. The VOL provides a monetary expression of aggregate utility experienced during life until its end, that is, utility is converted by the unit value of an "util", $u'(c)$. The VOL at the initial age is obtained by applying the formula $VOL = V/[\partial u(c(0), u(0), D_0)/\partial c(0)]$. The benchmark calibration predicts a VOL of about \$ 6.7 million at age 20. In terms of order of magnitude, this value corresponds well to Murphy and Topel's (2006, Fig. 3) estimate of a VOL of about \$ 6.5 million for American men at age 20.

Most studies on the toll of depression on longevity compute mortality hazard rates for small samples because computation of effects on life expectancy needs large sample sizes. Such a study is provided by Zivin et al. (2012) who use data from the U.S. department of Veteran Affairs and the National Death Index and find that depressed individuals died, on average, at age 71.1 while average non-depressed individuals died at age 75.9. While the sample is not representative, the

leading causes of death were similar to those of the general U.S. population. It is also useful for the calibration that the life expectancy of non-depressed individuals in the sample is close to the life expectancy of the Reference American. We thus model depression such that it reduces the length of life by 1-71.1/75.9= 6 percent, implying a life expectancy of 71.0 years of the depressed individual. In the benchmark run, we assume that depression is untreated, present from the start (i.e. at age 20), and permanent. The evidence suggests that after its first onset, depression stays at least as a threat and re-occurring illness throughout life. In a sensitivity analysis we consider alternative onsets and progressions of depression. For the benchmark case, we get the estimate $\delta = 1.74$ for the average severity of depression.

The literature has not yet converged on a general view about the efficacy of depression treatment and its impact on health and longevity. As a benchmark, we begin with a case where treatment is quite effective such that it reduces the impact of depression on life expectancy from 4.5 years to 1 year (by 75%). In order to pin down p_m, we take into account that, in the year 2000, expenditure for mental health contributes to about 6% of all health care expenditure (SAMHSA, 2016), that 7.3% of Americans suffered from (severe) depression, and that one third of this population remained untreated (SAMHSA, 2002). Let the average expenditure for health care and mental health over the life cycle be denoted by $\bar{h}$ and $\bar{m}$. We then back out p_m by solving $0.073 \cdot 2/3 p_m \bar{m}/(0.073 \cdot 2/3 p_m \bar{m} + p\bar{h})$. This provides the estimates $p_m = 0.46$ and $\eta = 0.0003$. We provide a sensitivity analysis with respect to the efficacy of treatment.
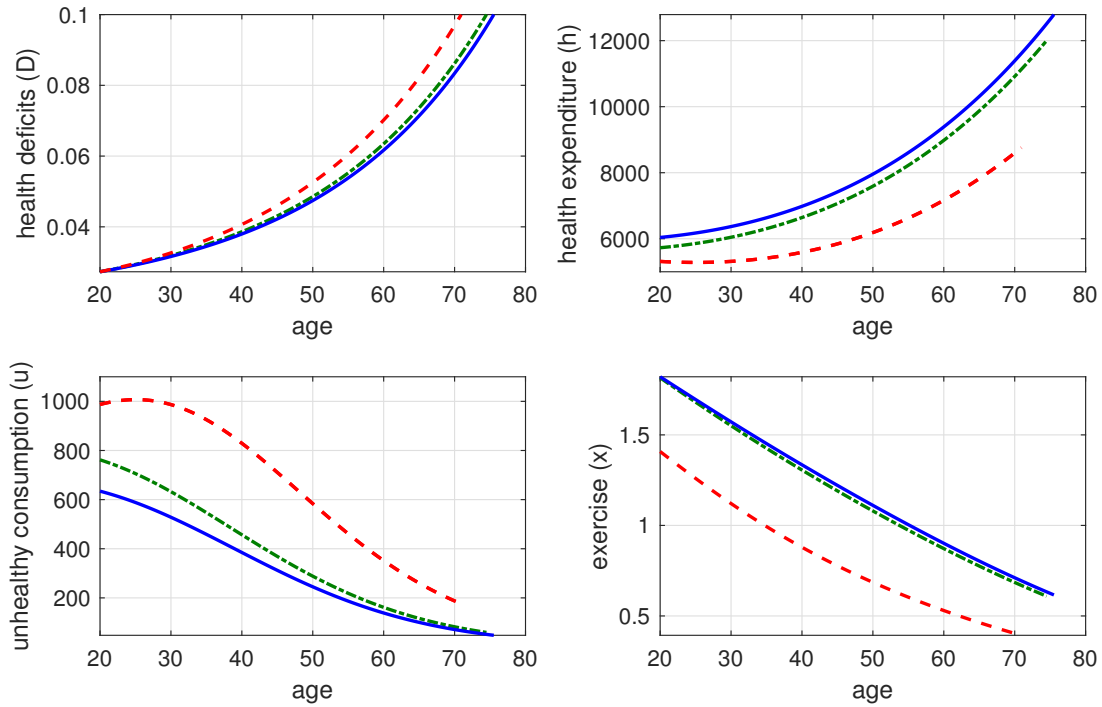
4. RESULTS: DEPRESSION, LIFE-CYCLE HEALTH BEHAVIOR, AND LONGEVITY

We solve model by applying the relaxation algorithm of Trimborn et al. (2008). We begin with the life cycle choices of the non-depressed benchmark American, which are shown in Figure 1 by blue (solid) lines. With advancing age, the individual develops more health deficits, spends more on health, consumes less unhealthy goods, and exercises less. At age 75.5, the individual dies when the terminal condition for health deficits $\bar{D}$ is reached. The life-cycle choices of non-depressed individuals are discussed in detail by Dalgaard and Strulik (2014) and Schuenemann et al. (2017a). The new choice variable here is physical exercise, which declines with age since exercise becomes increasingly painful as the individual develops more health deficits.

The life-cycle choices of the depressed individual are shown by red (dashed) lines in Figure 1. The relative change of behavior and age at death, compared to the non-depressed benchmark

FIGURE 1: DEPRESSION, HEALTH BEHAVIOR, AND HEALTH OUTCOMES

Blue (solid) lines: Reference American. Red (dashed lines): untreated depressed individual ($\delta = 1.74$, $\eta = 0$). Green (dash-dotted) lines: treated depressed individual ($\delta = 1.74$, $\eta = 0.0003$).

are shown as case 1 in Table 1. The depressed individual is predicted to spend much more on unhealthy consumption. Particularly, when young, unhealthy consumption is not declining with advancing age. Averaged over the lifetime, unhealthy consumption increases by 118 percent, compared to the benchmark case. The depressed individual invests less in health at all ages (24% less over the lifetime). The depressed individual also exercises less intensely (31% less effort in physical exercise). The fact that the depressed individual cares less about the future is also expressed in less savings. The average wealth (k) accumulated over the lifetime declines by 18%. The depressed individual has a relatively low regard for a long life because depression reduces the value of life quite dramatically. Compared to the benchmark, the VOL declines by 30.0%, implying that the model predicts that depression contributes about 30% to life satisfaction. In order of magnitude, this prediction accords with the estimate that mental illness explains 21 percent of the variation in adult life satisfaction in the US (and 32 percent in the UK), see Clark et al. (2017).

The prediction that depression motivates more unhealthy consumption, less savings, less health care, and less physical exercise are in line with the empirical observations cited in the Introduction. In order to elicit these behavioral changes, we need not to appeal on preference

changes of depressed individuals. Instead, the reason for the more unhealthy behavior is the depression itself, which reduces the value of life and makes the strife for a long life less worthwhile. Notice that a life-cycle model with endogenous health and longevity is necessary in order to obtain these result. A model that treats life length as given (and perhaps infinitely long) would require the manipulation of the utility functions $U(c, u, D)$ and $f(x, D)$ in order to elicit these behavioral changes. Here, the introduction of an exogenous drop in utility and life satisfaction is sufficient in explaining the change in behavior.

Table 1: Comparative Dynamics: Sensitivity Analysis and Model Extensions

case	parameter change	ΔT	$\Delta u/u$	$\Delta h/h$	$\Delta x/x$	$\Delta k/k$
1	$\delta = 1.74$	-4.5	1.18	-0.24	-0.31	-0.18
2	$\eta = 0.0003$	-1.0	0.20	-0.05	-0.01	0.00
3	$\eta = 0.0001$	-2.5	0.53	-0.12	-0.03	0.03
4	$\phi_m = 0.1$	-0.4	0.08	-0.02	0.00	0.00
5	$\phi_m = 0.5$	-1.6	0.33	-0.08	-0.02	0.01
6	$\phi_m = 1$	-2.9	0.62	-0.14	-0.04	0.02
7	$\phi_m = \phi = 0.5$	-2.6	0.29	-0.60	-0.02	-0.07
8	$\delta = 2.50$	-7.7	2.26	-0.34	-0.40	-0.47
9	$\delta = 2.50, \eta = 0.0003$	-1.1	0.22	-0.06	-0.01	0.04
10	$\nu = 1$	-4.6	1.19	-0.25	-0.31	-0.08
11	$\nu = 3$	-4.8	1.24	-0.25	-0.31	0.10
12	$\nu = 1, \eta = 0.0003$	-1.0	0.20	-0.05	-0.01	-0.01
13	$\nu = 3, \eta = 0.0003$	-1.0	0.20	-0.05	-0.01	-0.01
14	treatment delay 10 yrs	-1.7	0.20	-0.01	-0.05	-0.63
15	treatment delay 20 yrs	-3.3	0.25	-0.03	-0.14	-0.51
16	$\delta_0 = 1.29$	-4.5	1.16	-0.24	-0.30	-0.18
17	$\delta_0 = 1.10$	-3.6	0.89	-0.20	-0.26	-0.15
18	$\delta_0 = 1.29, \eta = 0.0003$	-1.0	0.20	-0.05	-0.01	0.01

The table shows the impact of self-control by reducing ω to zero; ΔT is measured in years, $\Delta u/u$ and $\Delta h/h$ are measured in percent.

The life cycle choices of the treated depressed individual are shown by green (dash-dotted) lines in Figure 1 and summarized as case 2 in Table 1. By assumption the treatment is very effective. The lifetime trajectories for physical exercise of the treated individual and the non-depressed individual basically coincide. There is also a pronounced increase in health expenditure (to 5 percent below non-depressed expenditure). Here, the improvement is less than complete because the out-of-pocket expenditure for depression treatment is partly financed by reduced out-of-pocket expenditure for general health. With lower health expenditure (compared to the benchmark), the incentive to spend more on unhealthy consumption increases, see the discussion

of equation (14). The treatment almost eliminates the entire impact of depression on happiness. The value of life of the treated individual is 1% below the benchmark (compared to 30% of the non-treated). As a result, aggregate savings are now even higher than for the non-depressed individual. The treatment cured the low valuation of a long life and raised the need for savings to finance the out-of-pocket share of mental health care in old age.

Case 3 in Table 1 considers outcomes when depression treatment is less effective (η declines to 0.0001). As a result, treatment is not able to sufficiently restore the value of life. Depressed individuals still exhibit less healthy behavior than the non-depressed, and treatment saves only 2 years of longevity (T is reduced by 2.5 years instead of 4.5 when untreated).

Case 4 to 6 consider different out-of-pocket shares for depression treatment. A larger out-of-pocket share reduces the budget that remains for other expenditures. In particular, general health investment declines with increasing out-of-pocket share for depression treatment. This increases the marginal productivity of health care and induces more unhealthy consumption (see (14)). Physical exercise, in contrast, is almost restored to the non-depressed level. This indicates that depression is still (almost) cured, as in case 3. The difference in health deficit accumulation and age of death between these cases operates through the budget constraint. This can also be seen in the response of savings. A higher out-of-pocket share induces more savings for depression treatment in old age, which, among other things, leaves less income to be spent on current health investments. For completeness, case 7 considers a simultaneous increase in the out-of-pocket share for both types of health expenditure. As expected, this pariculary harms investments in general health care.

Case 8 considers a more severe depression: δ increases to 2.5, which means that, untreated, depression can be attributed to the loss of 7.7 years of lifetime. Interestingly, when treated, the health outcomes and behavioral changes are almost the same as in the case of milder depression, as the comparison between cases 9 and 2 shows. The biggest difference is in terms of savings since more severe forms of depression require more expenditure for treatment in old age.

5. Applications and Extensions

5.1. **Depression and Labor Supply.** So far, we have ignored the additional indirect effects of depression on life-cycle outcomes through reduced productivity and labor income. The recent study of Peng et al. (2016) suggests that the effects on income may indeed be small. Depression
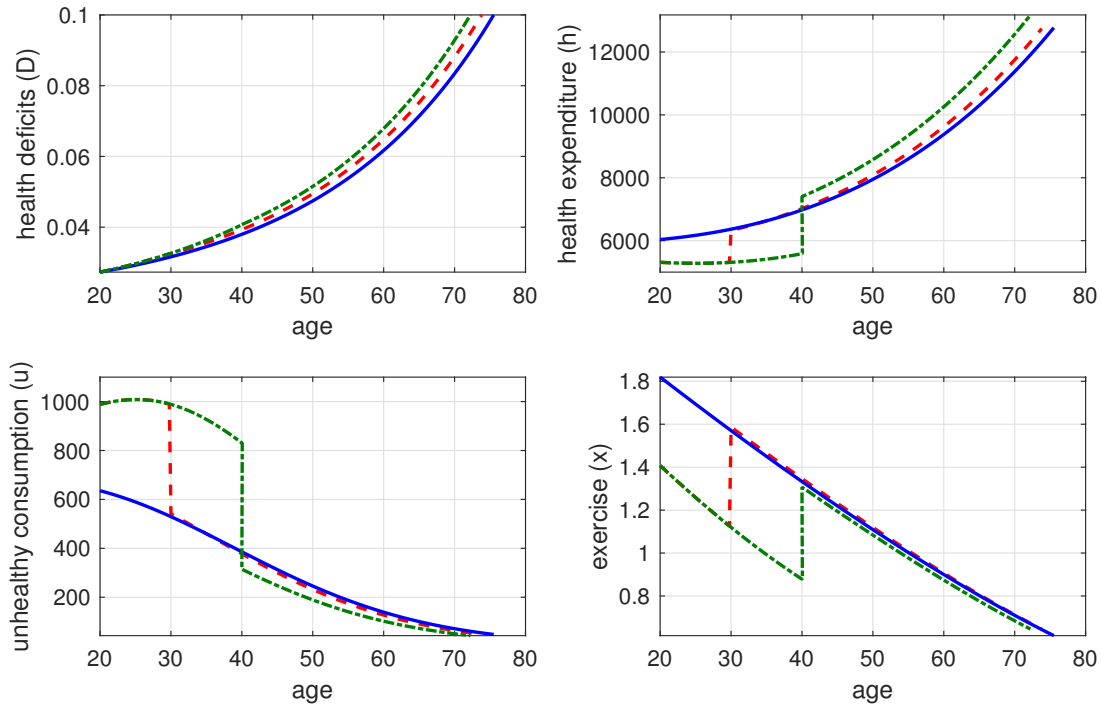
is estimated to reduce the probability of employment by 2.6 percentage points and to increase annual work loss days by 1.4 days. Earlier studies suggested higher effects on labor market outcomes (e.g. Chatterji et al., 2011). In the model, a convenient way to acknowledge labor market effects is through early retirement. Subsequently, we assume that the previous age of retirement ($R = 65$) is reduced to $R - \nu \delta \mathrm{e}^{m\eta}$. In cases 10 and 11 of Table 1, we consider relatively strong effects of depression on retirement and lifetime income by setting $\nu = 1$ and $\nu = 3$. This means that, in case 10, the individual receives 1.74 times (1-0.45) times w, i.e. \$ 33,800 less lifetime income. In case 11, it implies that depression reduces lifetime income by \$ 101,400.

Comparing results of case 10 and 11 with case 1 shows that outcomes for untreated depression are, perhaps surprisingly, only marginally affected by the income feedback although the impact on income is large. In other words, the direct depression effect on life satisfaction is much stronger than any indirect effect through income, as suggested in related empirical studies (Layard et al. 2013). Health behavior, in turn, responds more sensitively to depression than to income. Case 12 and 13 further confirm this assessment by showing that the treatment outcomes in case of feedback effects also deviate only insignificantly from the benchmark treatment (Case 2).

5.2. **Delayed Treatment.** As discussed in the Introduction, a large share of depression cases remains untreated. One reason for a delay in treatment could be that individuals and their relatives fail to self-diagnose the disease and confound and perhaps rationalize it as (justified) sadness "with a reason". In Table 1, case 14, we see the health outcomes when depression is treated with a delay of 10 years. The individual loses 1.7 years of lifetime, compared to 1.0 year if depression is treated immediately. The life-cycle behavior shown in Figure 2 explains why. Blue (solid) lines reiterate the life-cycle trajectories of the benchmark non-depressed individual. Red (dashed) lines show the trajectories when treatment is delayed by 10 years. They coincide with those of the untreated individual from Figure 1 until treatment sets in. Then, the treated depressed individual actually behaves in a more healthy manner than the non-depressed individual: he invests more in health, consumes less unhealthy goods, and engages in somewhat more physical exercise. But it is too late to fully compensate for the faster deteriorating health during early adulthood. Health deficits continue to accumulate faster after treatment and lead to an earlier death.

Case 15 in Table 1 and the green (dash-dotted) trajectories in Figure 2 show the outcome when treatment is delayed by 20 years. The reduction of longevity now more than triples compared
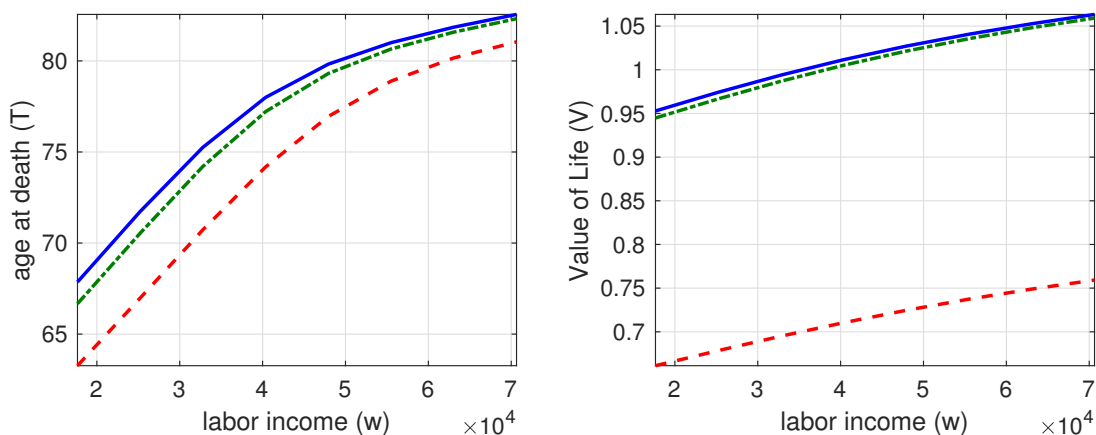
FIGURE 2: DELAYED TREATMENT

Blue (solid lines): non-depressed individual. Red (dashed lines): benchmark depressed individual with treatment delay of 10 years. Green (dashed-dotted lines): benchmark depressed individual with treatment delay of 20 years.

to benchmark treatment (case 2). Aggregate behavioral outcomes are in fact more similar to completely untreated depression than to instantly treated depression. Significantly increased health expenditure after treatment and reduced unhealthy consumption fail to compensate for the lost health during the depression phase. These outcomes are a manifestation of the quasi-exponential nature of the health deficit accumulation, which leads to the amplification of early-life health shocks over time. The health capital model (Grossman, 1972), in contrast, would predict that initial differences in health shocks – or, in the current case, the consequences of delayed treatment – depreciate as the individual ages (see Almond and Currie, 2011 and Dalgaard et al., 2017 for a critique). The health deficit model, in contrast, captures the cumulative character of health deficit accumulation emphasized in gerontology (e.g. Arking, 2006). The damage done in young adulthood by unhealthy consumption, little health investment, and little physical exercise is never fully repaired after the onset of depression treatment. These outcomes highlight the importance of diagnosing and treating the disease early.

5.3. **Income Gradient.** We next look at the income gradients of longevity and the value of life and how they are transformed by depression. To do so, we feed different values of labor

income ranging from half to double the benchmark income into the calibrated model. Results are shown in Figure 3. The value of life is scaled relative to the benchmark. Blue (solid) lines show the gradient for non-depressed individuals. The predicted age at death is steeply increasing in income, in particular, at low income levels (as explained in detail in Dalgaard and Strulik, 2014). The value of life (life satisfaction) increases less steeply. It is about 10 percent higher for the richest individuals compared to the poorest, reflecting strongly decreasing returns of income on utility, as emphasized by many happiness studies (e.g. Layard et al., 2013, 2017).
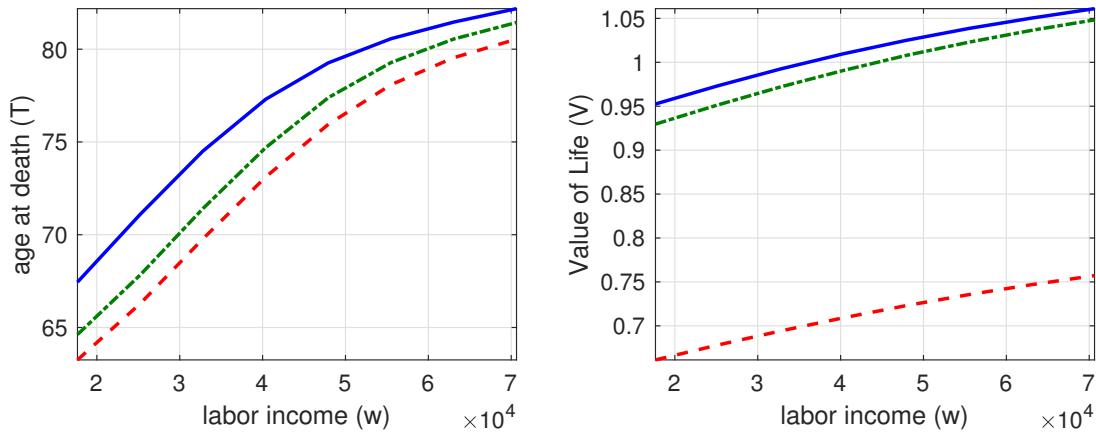
FIGURE 3: INCOME GRADIENT

The value of life (at age 20) is computed relative to the value of life of the benchmark case (Table 1). Blue (solid) lines: non-depressed individual. Red (dashed lines): untreated depressed individual ($\delta = 1.74$, $\eta = 0$). Green (dash-dotted) lines: treated depressed individual ($\delta = 1.74$, $\eta = 0.0003$). Other parameters as for the benchmark case.

Non-treated depression, shown by red (dashed) lines, implies a considerable shift downwards of the value of life, which declines by about 30 percent across all income levels. In terms of longevity, however, the poor are particularly harmed by depression. The income gradient shifts down by about 5 years at low income levels and "only" by about 2 years at high income levels. Poor people realize a relatively low level of consumption, and thus experience relatively high marginal utility from current consumption, which drives down the incentives to invest in health, to save, and to abstain from unhealthy goods. These mechanisms further drive down the longevity of poor depressed individuals.

When depression is treated, shown by green (dash-dotted) lines, the value of life (life satisfaction) is almost restored to its original level for all income strata. Rich individuals are able to almost completely restore their longevity compared to non-depressed individuals. Treatment also greatly benefits the longevity of the poor. The shift towards the non-depressed gradient,

however, is incomplete. Treatment costs matter more for the poor and drive down investment in general health and savings (for health expenditure in old age).

FIGURE 4: INCOME GRADIENT ($\phi_m = 1$)

The value of life (at age 20) is computed relative to the value of life of the benchmark case (Table 1). Blue (solid) lines: non-depressed individual. Red (dashed lines): untreated depressed individual ($\delta = 1.74$, $\eta = 0$). Green (dash-dotted) lines: treated depressed individual ($\delta = 1.74$, $\eta = 0.0003$). Other parameters are the same as in the benchmark case.

In order to highlight the last comparative dynamics, we set the out-of-pocket share for depression $\phi_m = 1$ and redo the sensitivity analysis with respect to income. Results are shown in Figure 4. Blue and red lines coincide with Figure 3. The green line shows outcomes when the treatment costs of depression are borne privately. The opportunity cost of treatment is now larger, in particular ,for poor people due to declining marginal utility from consumption. Without access to insurance, depression treatment remains incomplete and individuals have less disposable funds to spend on health investment and savings for health investments in old age. This fact is visible by the incomplete upward shift of longevity for treated individuals. The loss of longevity is particulary strong for poor individuals who lose about 3 years of life with treatment (instead of about 1 year as in the case of Figure 3). Notice that we imposed equal (non-) access to insurance for all social strata. Taking into account that access to insurance (i.e. the size of ϕ_m) is positively correlated with income would further increase the income gradient for depressed individuals.

5.4. **Hump-Shaped Depression.** In principle, depression could appear and vanish at any given age. These lifetime trajectories can be constructed by matching non-depressed and depressed lifetime outcomes (health deficits, capital stock) at the time when depression occurs or vanishes. Here, we consider another interesting case where the intensity of depression varies over
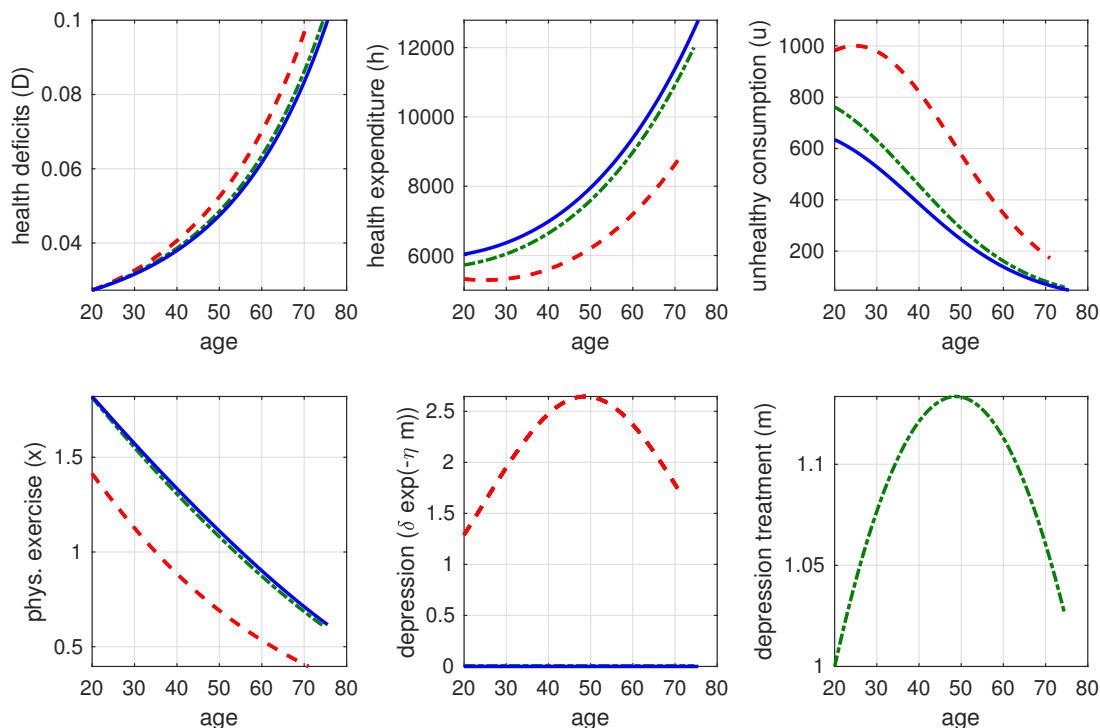
the lifetime (and may only at its peak, be experienced as unbearable). This scenario relates to Blanchflower and Oswald (2008, 2016), who argue that life satisfaction is u-shaped across the life cycle and that this pattern is partly explained by an invertedly u-shaped age pattern of the prevalence or intensity of depression. As corroborating evidence, they show that the prevalence of antidepressant use is invertedly u-shaped in age with a peak in the late forties.

In order to conveniently represent these ideas we replace the constant δ with a depression function for age t:

$$\delta(t) = \max\left\{0, \ \delta_0 \exp(\psi_1 t - \psi_2 t^2) - \xi\right\}. \tag{19}$$

The ψ-parameters control the age pattern, δ_0 controls the severity of depression, and ξ is a perception threshold. With ξ, we can determine at which ages depression is perceived as salient such that it motivates a desire for treatment. To begin with, we set $\xi = 0$ and adjust the other parameters such that depression reaches a maximum at age 49 and that, if untreated, leads to the loss of 4.5 life years. This provides the estimates $\psi_1 = 0.05$, $\psi_2 = 0.00087$, and $\delta_0 = 1.29$.

FIGURE 5: HUMP-SHAPED DEPRESSION

Blue (solid) lines: Reference American. Red (dashed lines): untreated age-dependent depression.
Green (dash-dotted) lines: treated age-dependent depression.

Results are shown in Figure 5 and case 16 in Table 2. Blue (solid) lines reiterate the life cycle trajectories for the non-depressed individual. Red (dashed) lines show the trajectories for the
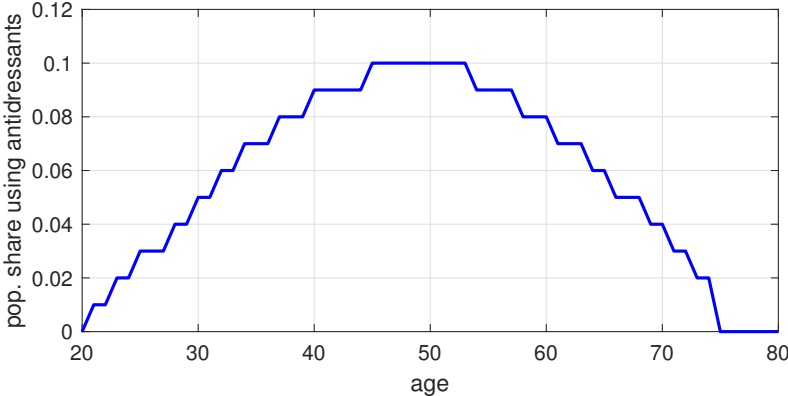
non-treated individual. Unsurprisingly, the results for health behavior and outcomes look very similar to those for the benchmark case from Figure 1. Depression, shown at the center panel at the bottom of Figure 3, is (by design) invertedly u-shaped in its intensity. Remarkably, the impact of depression on the value of life (life satisfaction) is higher than for the benchmark run. The VOL declines by 34.2 % instead of 30.0%. The reason is that for young individuals (whose instantaneous utility is not much discounted to the present) depression is on the rise while for old individuals (whose instantaneous utility is heavily discounted to the present) depression declines.

Alternatively, we could match the impact of depression on the value of life to be equal to the benchmark depression case (i.e. 30%). This leads to the estimate $\delta = 1.10$. Results are shown as case 17 in Table 2. The impact on health outcomes is now smaller because depression in young age has a particularly severe impact on health outcomes through reduced health investments and increased unhealthy behavior and the caused cumulative damage. Finally, case 18 in Table 1 shows that health outcomes for treated u-shaped depression do not differ significantly from those for constant depression (case 2).

We next explore sensitivity with respect to ξ, the threshold of depression perception. Keeping the other parameter values as specified above, a value of $\xi = 1.3$ implies that (perceptible) depression sets in shortly after the 20th birthday (at model age $t = 0.02$) and then gradually rises in severity until age 49 after which it declines again to a low positive value before death. A value of 2.6 implies that depression is perceived only as a "midlife low" between ages 45 and 53.

FIGURE 6: SHARE OF POPULATION USING ANTI-DEPRESSANTS

Ten percent of the population is susceptible to depression. For this subpopulation the perception threshold ξ is uniformly distributed in (1.3, 2.6). The figure shows the predicted prevalence of depression at any given age. Parameters are kept the same as in the benchmark run.

Suppose that 10 percent of the population are susceptible to depression and that among these individuals ξ is uniformly distributed in $(1.3, 2.6)$. In order to make inferences about the association of age and aggregate demand for antidepressants, we run the model (with otherwise benchmark parameters) for 10 equally-spaced draws from $(1.3, 2.6)$ and compute for any age (in the discrete space of natural numbers) whether antidepressants are used. Figure 6 shows the predicted population share using antidepressants. The inverted u replicates the empirical association between age and antidepressant use estimated by Blanchflower and Oswald (2015).

6. Conclusion

This paper introduces depression into health economic theory and evaluates its impact on life-cycle health behavior and longevity. Inspired by happiness research, depression has been conceptualized as a potentially large drop in utility (happiness) and the value of life (life satisfaction). Successful therapy (partly) restores original utility and life satisfaction. These elements have been integrated into a life-cycle model of health deficit accumulation, augmented by utility-enhancing unhealthy consumption and utility-reducing but health-enhancing physical exercise.

The model explains why depressed people consume more unhealthy goods, save less, invest less in their health, and exert less effort in physical exercise. These responses are elicited without transforming the utility function for consumption and exercise. They are an expression of the depression itself in a model that considers longevity as endogenous and at least partly malleable by health behavior. Since depressed people experience less life satisfaction, they strive less to extend life and are less motivated to exhibit healthy behavior.

The model has been calibrated to predict the life-cycle trajectories of health behavior and health outcomes for a non-depressed Reference American. Introducing depression as a 30% drop in life satisfaction rationalizes changes in health behavior that reduce the length of life by about 4 years. Extensions of the model have shown that these results are obtained largely independently from the size of (reasonable) feedback effects of depression on labor supply and lifetime income. The timing of treatment, however, is predicted to be crucial. Delays in diagnosis or treatment lead to severe losses in health that are not fully recovered in the treatment period. The reason is that the (gerontologically founded) health deficit model amplifies the late-life consequences of health shocks in early life. In the present context, this means that individuals do not manage

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to compensate fully for the health destroyed by unhealthy behavior during untreated depression in early adulthood.

Since this study is a first attempt to discuss depression in the context of health and longevity, there are ample possibilities to further extend and refine the theory. For example, it could also be taken into account that depression has a direct biological impact on health through, for example, blood pressure, inflammation, or immune function. Another possibility is to analyze inferior and potentially health-damaging self-treatment of depression symptoms, for example, by excessive alcohol consumption. This could be a particularly interesting endeavor in the context of the income gradient of longevity and limited access to insurance-covering clinical depression treatment. Such an analysis could also consider the addictive potential of the applied self-treatment, investigate the repercussions of unhealthy addiction on longevity (Strulik, 2017), and explain the positive association between depression and addiction (Solomon, 2015).

The assumed exogeneity of depression is a reasonable first approximation of reality and a useful device in order to identify causality. An extension, however, could discuss depression triggered by bad health and investigate the interdependence of both phenomena. In particular, chronic pain seems to be associated with depression (DePaulo and Horvitz, 2002). This fact could establish a link between depression, use of antidepressants and painkiller consumption, and contribute to the literature on the opioid epidemic .

APPENDIX

6.1. **Derivation of (14) to (18).** We first write the first order conditions (5)–(9) and the costate equations (11)–(12) for the parameterized utility function.

$$\left(\frac{D}{D_0}\right)^{-\epsilon} [\theta c + (1-\theta)u]^{-\sigma}\,\theta = \lambda_k \tag{A.1}$$

$$\left(\frac{D}{D_0}\right)^{-\epsilon} [\theta c + (1-\theta)u]^{-\sigma}(1-\theta) \le \lambda_k q - \lambda_D \mu B \omega u^{\omega-1} \quad \text{with = for } u > 0 \tag{A.2}$$

$$-\lambda_D \mu \gamma A^{\gamma-1} = \lambda_k \phi p \tag{A.3}$$

$$-\lambda_D \mu \kappa E x^{\kappa-1} = \tau \left(\frac{D}{D_0}\right)^{\chi} \tag{A.4}$$

$$-\delta\eta e^{-\eta m} \le \lambda_k \phi_m p_m \quad \text{with = for } m > 0. \tag{A.5}$$

$$\lambda r = \lambda_k \rho - \dot{\lambda}_k \tag{A.6}$$

$$-\epsilon \left(\frac{D}{D_0}\right)^{-\epsilon-1} \frac{[\theta c + (1-\theta)u]^{1-\sigma}-1}{1-\sigma} - \chi \left(\frac{D}{D_0}\right)^{\chi-1}\tau x + \mu\lambda_D = \lambda_D \rho \dot{\lambda}_D \tag{A.7}$$

Let $\tilde{c} \equiv \theta c + (1-\theta)u$ define a weighted measure of total consumption. From (A.1) and (A.2) we obtain

$$\frac{1-\theta}{\theta}\lambda_k - q\lambda_k + \lambda_D \mu B \omega u^{\omega-1} \le 0.$$

Using (A.3) to eliminate λ_D and and dividing by λ_k we obtain

$$\frac{1-\theta}{\theta} - q \le \frac{\phi p}{\gamma A h^{\gamma-1}}B\omega u^{\omega-1}.$$

Solving for u provides (14) in the text. Inserting (A.3) into (A.4) provides:

$$\frac{\lambda_k \phi p}{\gamma A h^{\gamma-1}}\kappa E x^{\kappa-1} = \left(\frac{D}{D_0}\right)^{\chi}\tau.$$

Inserting λ_k from (A.1) we get

$$\left(\frac{D}{D_0}\right)^{-(\epsilon+\chi)}\frac{\phi p}{\gamma A h^{\gamma-1}}\theta\tilde{c}^{-\sigma}\kappa E = \tau x^{1-\kappa}.$$

Solving for x provides (15) in the text. Inserting (A.1) into (A.5) we obtain

$$\frac{\delta\eta(\tilde{c}^{\sigma}(D/D_0)^{\epsilon}}{\phi_m p_m} \le e^{\eta m}.$$

Solving for m provides (16) in the text. Log-differentiating (A.1) provides

$$-\epsilon\frac{\dot{D}}{D} - \sigma\frac{\dot{\tilde{c}}}{\tilde{c}} = \frac{\dot{\lambda}_k}{\lambda_k}.$$

Eliminating $\dot{\lambda}_k/\lambda_k$ using (A.6) provides (17) in the text. Log-differentiating (A.3) and eliminating $\dot{\lambda}_k/\lambda_k$ using (A.6) provides

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left(\rho - r - \frac{\dot{\lambda}_D}{\lambda_D}\right). \tag{A.8}$$

26

Using (A.3), equation (A.7) can be written as

$$-\frac{\lambda_D}{\lambda_D} = \mu - \rho - \left[\epsilon \left(\frac{D}{D_0} \right)^{-\epsilon-1} \frac{\tilde{c}^{1-\sigma} - 1}{1 - \sigma} + \chi \left(\frac{D}{D_0} \right)^{\chi-1} \tau x \right] \frac{\mu \gamma A h^{\gamma-1}}{\lambda_k \phi p}.$$

Using (A.1) this can be expressed as

$$-\frac{\lambda_D}{\lambda_D} = \mu - \rho - \left[\epsilon \frac{\tilde{c}^{1-\sigma} - 1}{1 - \sigma} + \chi \left(\frac{D}{D_0} \right)^{\chi+\epsilon} \tau x \right] \frac{\mu \gamma A h^{\gamma-1} \tilde{c}^{\sigma}}{\lambda_k \phi p D}. \tag{A.9}$$

Inserting (A.9) into (A.8) provides (18) in the text.

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