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**I SHOULDN'T EAT THIS DONUT: SELF-  
CONTROL, BODY WEIGHT, AND  
HEALTH IN A LIFE CYCLE MODEL**

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# I Shouldn't Eat this Donut: Self-Control, Body Weight, and Health in a Life Cycle Model \*

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**Abstract.** In this paper I discuss overweight and obesity and their repercussions on health deficit accumulation and longevity in a life cycle model. Individual decisions are conceptualized as the partial control of impulsive desires of a short-run self (the limbic system) by a rationally forward-looking long-run self (the prefrontal cortex). The short-run self strives for immediate gratification through consumption of food and other goods. The long-run self reflects the consequences of eating behavior on weight gain and health, exercises to lose weight, invests money to improve health and saves for health expenditure in old age. Not conceding to short-run desires, however, entails an idiosyncratic utility cost of self-control. The model is calibrated to match food expenditure, exercise, and other choices of an average U.S. American. The results suggests that imperfect self-control reduces average lifetime by up to five years. I use the model to analyze the role of self-control, income, food prices, energy density, and medical progress in explaining obesity and to develop a test on whether obesity is driven by excessive desire for food or lack of self-control.

*Keywords:* self-control, overweight, obesity, physical exercise, health investments, aging, longevity.

*JEL:* D11, D91, E21, I10, I12.

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## 1. INTRODUCTION

In the year 2016, worldwide, 39 percent of adults were overweight and 13 percent were obese. In developed countries, the median citizen is typically overweight and between 20 percent and more than 40 percent are obese. U.S. Americans are particularly big (67.9 percent overweight and 36.2 percent obese), yet other countries are catching up quickly (WHO, 2018).<sup>1</sup>

Elevated body mass promotes the development of health deficits and premature death. Overweight and obesity increase the risk of heart disease, ischemic stroke, type 2 diabetes, and some cancers (including endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon cancer) as well as osteoarthritis and other musculoskeletal disorders. The WHO estimates that, worldwide, at least 2.8 million people die each year as a result of being overweight or obese.

The unhealthy built-up of excess body mass could, in principle, be avoided by consuming less calories or burning more calories (through physical exercise and other activities). The fact that it is seemingly so straightforward to avoid overweight makes the phenomenon a particularly fascinating object of investigation for economists who believe in rational decision making and long-term planning. The present paper aims to contribute to this literature by developing a life cycle model in which individuals understand the consequences of food consumption and exercise on health deficit accumulation, longevity, and lifetime utility.<sup>2</sup>

A straightforward way to explain the diversity of BMI-phenotypes is to focus on differences in preferences for food or physical (in-) activity and maintain the conventional assumption of individuals as fully rational planners. I argue below that this line of reasoning is not entirely convincing in the context of a life cycle model when health outcomes and longevity are endogenous. It would imply the prediction that heavier individuals exercise more when the argument is based on varying food preferences or that heavier individuals eat less when the argument is based on varying preferences for (in-) activity. At the minimum, it would thus beg the question for why food preferences and activity preferences are correlated.

Here, I suggest an alternative explanation, based on present-biased preferences, that motivates varying body sizes without resorting to differences in preferences for food or physical exercise.

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<sup>1</sup>Overweight is defined as having a body mass index (BMI) above 25 and obesity as a BMI above 30. BMI is defined as weight in kilogram divided by the square of height in meters. The optimal healthy BMI lies in the range of 21 to 23 (WHO, 2018).

<sup>2</sup>Alternative and complementing economic models of obesity are proposed by, among others, Levy (2002), Cutler et al. (2003), Lakdawalla et al. (2005), Philipson and Posner (2009), Dragone and Savorelli (2011), Strulik (2014), and Dragone and Ziebarth (2017).

Specifically, food consumption and exercise and their effects on health outcomes and longevity are discussed in a life cycle model of imperfect self-control based on Thaler and Shefrin (1981) and Fudenberg and Levine (2006). This approach formalizes the notion that humans are neither mere “cold” long-run planners nor “hot” affective persons by considering a dual-self consisting of a rational long-run self who partly controls the impulsive actions of a short-run self. Self-control is costly in terms of utility, reflecting the pain from not conceding to the cravings of the short-run self for, for example, another icecream or a leisurely evening on the couch. The cost of self-control is increasing in the deviation of the constrained optimal solution from the optimal solution preferred by the short-run self.

The dual-self model provides a particular, psychological view on the role of impatience and present bias for human decision making. In economics, these phenomena are usually addressed by the modeling of a discount rate that individuals apply to utility experienced in the future. In health economics, several studies have found that individuals who heavily discount the future are more likely to be obese, to exercise less, and to perform fewer health maintenance activities (Komlos, 2004; Bradford, 2010; Lawless et al., 2013; Bradford et al., 2014; Courtemanche et al., 2015). The conventional model of exponential discounting is a useful tool for analyzing the impact of (rational) impatience for health behavior. It is, however, unsuitable for addressing present bias stemming from affective (irrational) behavior. For that purpose, it has been proposed to use hyperbolically declining discount rates.

While it is undisputed that individual decisions are subject to a strong present bias (for surveys, see Frederick et al., 2002; DellaVigna, 2009), it is less clear whether hyperbolic discounting is the best way to describe affective behavior and self-control problems. According to conventional wisdom, hyperbolic discounting necessarily involves time-inconsistent decision making (see e.g. Angeletos et al., 2001., p.53; Cawley and Ruhm, 2012, p. 139). It is possible, however, to propose empirically plausible forms of hyperbolic discounting that support time-consistent decisions.<sup>3</sup> Strulik and Trimborn (2018) have recently integrated time-consistent hyperbolic discounting into a standard life cycle model. They showed that hyperbolically discounting individuals invest more in their health, spend less on unhealthy goods, and live longer than they

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<sup>3</sup>Formally, Strotz’s (1956) fundamental theorem states that only exponential discounting leads to time-consistent decisions if the discount factor is a function of the algebraic distance between planning time and payoff time. The “if”-clause, however, seems to sometimes be forgotten in the following literature. In fact, any form of discounting that is separable in planning time and payoff time implies time-consistency, see Theorem 1 in Burness (1976) and Drouhin (2015).

would if they had a constant time preference rate. This suggests that, if hyperbolic discounting is associated with inferior health behavior, the cause is not the present-bias as such but the entailed inconsistency of decision making. These considerations may be helpful for an assessment of the sometimes inconclusive studies on the impact of hyperbolic discounting on health behavior (e.g. Khwaja et al., 2007). Time-inconsistent hyperbolic discounting also involves difficult conceptual problems. First, it needs to address how individuals deal with their inconsistency problem (in a naive or sophisticated way). Second, the question arises as to whose welfare should be considered by policy interventions. It is thus useful to have an alternative modeling device of present-biased decision making, which models behavior that is time-consistent yet suboptimal from the perspective of a rational long-run planner.

Conceptually, the dual-self model is closer to the notion of multiple simultaneously-operating brain systems in psychology and neuroscience. It takes into account insights from neurology showing that different areas of the brain are occupied with short-run (impulsive) behavior and long-run (planned) behavior (McClure et al., 2004; Bechera, 2005; Hare et al., 2009). Affective states are triggered by the evolutionary older limbic system, which responds to stimuli without accounting for long-term consequences. Abstract thinking and long-term planning are located in the prefrontal cortex, the evolutionary newest area of the brain. The degree by which processes in the prefrontal cortex inhibit and override processes of the limbic system is called self-control or willpower and it is person-specific (i.e. brain-specific).

A series of empirical studies have provided evidence for imperfect self-control as a driver of impulsive consumption and low investment in general (Shiv and Fedorikhin, 1999; Baumeister, 2002; Ameriks et al., 2007) as well as driver of unhealthy eating (Crescioni et al., 2011; Dassen et al., 2015; Stutzer and Meier, 2016) and lack of physical exercise (Bogg and Roberts, 2004; Della Vigna and Malmendier, 2006; Cobb-Clark et al., 2014; Connell-Price and Jamison, 2015). Low self-control in childhood is a strong predictor of health and financial status in adulthood (Moffitt et al., 2011). Adolescents with low self-control develop more health deficits later in life (Miller et al., 2011) and individuals with low self-control tend to die earlier (Kern and Friedman, 2008).

Inferences about causality, however, are difficult to obtain from empirical studies because there exists no counterfactual or treatment group. Since we cannot observe the same person twice, both with and without self-control (aside from rare exceptions, Navqi et al., 2007), there remains

an identification problem. This makes it hard to assess how much low self-control contributes to inferior health behavior and premature death. Here, I suggest addressing this problem with counterfactual computational experiments. I calibrate a dual-self life cycle model of health behavior for a Reference American with limited self-control and then perform a counterfactual exercise by removing the self-control problem.

It should be noted that the method of counterfactual experiments is not meant as a replacement of econometric analysis. It is a complementing, alternative method of inference that is particularly designed to avoid some problems like those originating from backward causality and omitted variables. In the present context, for example, one can easily imagine that self-control also influences variables other than eating, exercising, and health investments. A plausible channel, for example, would be work effort such that self-control exerts an indirect effect on health through income. The perhaps most useful feature of computational experiments is that certain channels of influence can be shut down by design. In the example, by holding income constant, the computational experiment controls for the impact that self-control could have through income or other confounders on health behavior and health outcomes.

The life cycle model of health deficit accumulation (Dalgaard and Strulik, 2014) is a an appropriate tool with which to discuss how current unhealthy behavior affects the deterioration of bodily function and causes premature death. It is particularly useful for quantitative explorations since it employs a straightforward measure of health that has been well-established in gerontology, the health deficit index.<sup>4</sup> The health deficit (or frailty) index measures the number of health deficits that a person has at a given age relative to the number of potential health deficits. It was introduced by Mitnitski et al. (2002) and has instigated a very large body of literature in the medical sciences.

The health deficit model is integrated with the dual-self model in order to discuss the impact of limited self-control on food consumption, exercise, health investments, obesity, health deficit accumulation, and longevity.<sup>5</sup> The model is calibrated to match food expenditure, exercise, and other choices of an average U.S. American and then used for counterfactual computational

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<sup>4</sup>The health capital model (Grossman, 1972), in contrast, investigates a latent variable, unknown to doctors and medical scientists, a fact that confounds a serious calibration of the model. See Case and Deaton, 2005; Wagstaff, 1986; Zweifel and Breyer, 1997; Almond and Currie, 2011 Dalgaard and Strulik, 2015; and Dalgaard et al., 2017 for a detailed critique of the health capital model.

<sup>5</sup>Earlier quantitative studies using the health deficit model were concerned with the Preston curve (Dalgaard and Strulik, 2014), the education gradient (Strulik, 2018a), the long-term evolution of the age at retirement (Dalgaard and Strulik, 2017), and the gender gap in mortality (Schuenemann et al. 2017a).

experiments in order to assess the importance of self-control problems for health behavior and premature death.

A static dual-self model of food consumption has been proposed by Ruhm (2012). Naturally, the impact of current eating behavior on the future development of health deficits and on longevity cannot be discussed in a static framework. A dynamic dual-self model in the context of health deficit accumulation has been developed in Strulik (2018b). The present paper applies this framework to discuss self-control problems in food consumption and extends it by considering self-control problems in physical exercise. Food consumption and exercise interact as determinants of body mass, health deficit accumulation, and the length of life.

The remainder of the paper is organized as follows. The next section presents the model and introduces the key assumptions and mechanisms. In Section 3, the model is calibrated for a 20-year-old American man (called the Reference American). Section 4 presents life cycle behavior and health outcomes for the Reference American and an otherwise identical person without self-control problems. The results suggest that imperfect self-control reduces average lifetime by up to five years. After scrutinizing this finding with extensive robustness checks, we investigate the role of food prices, income, and medical progress for eating behavior and body weight and use the model to develop a test for whether obesity is driven by excessive desire for food or lack of self-control. Section 5 concludes with limitations and potential extensions for future work.

## 2. THE MODEL

Consider an individual who derives utility from consuming non-food goods  $c$  and food  $u$ . For a plausible numerical implementation, we need to assume that not all food consumption provides utility. Let  $\bar{u} \geq 0$  denote unexciting food consumption performed in daily routine to provide the body with energy and nutrients. Utility is then derived from  $u - \bar{u}$ , in which  $\bar{u}$  plays the role of “subsistence consumption” of the conventional Stone-Geary-type utility function. It is thus excess food consumption that is desirable and tempting for the individual. Specifically, let the utility function for consumption be given by

$$U(c, u) = \frac{[(1 - \beta)c^\psi + \beta(u - \bar{u})^\psi]^{\frac{1-\sigma}{\psi}} - 1}{1 - \sigma}, \quad \beta, \sigma > 0, \quad \psi < 1. \quad (1)$$



The elasticity of substitution between excess food consumption  $u - \bar{u}$  and non-food consumption is given by  $1/(1 - \psi)$  and by estimating a high value to  $\psi$  we would assign a high elasticity of substitution. At the same time, the elasticity of substitution between aggregate food consumption  $u$  and non-food consumption is relatively low, capturing the fact that large parts of food consumption are essential to fulfil metabolic needs and cannot be easily substituted by non-food goods. The elasticity of intertemporal substitution is given by  $1/\sigma$  and the utility weight  $\beta$  is a measure for the general desirability (or tastefulness) of food.

As motivated in the Introduction, individuals are conceptualized as dual selves, and modeled as suggested by Fudenberg and Levine (2006). The impulsive short-run self desires to maximize instantaneous gratification from consumption of food and non-food goods and neglects the long-run consequences of consumption on body weight and health. Let  $w$  denote labor income and  $q$  denote the relative price of food. The price of non-food consumption goods is normalized to unity. An individual can spend at most  $\theta w$  instantaneously to buy food and other goods. For  $\theta = 1$ , this formulation could capture the idea that the short-run self wants to spend all liquid funds immediately and that non-labor (i.e. financial) income is not perfectly liquid. Maximizing utility of the short-run self (1) subject to the budget constraint  $\theta w = c + qu$  provides the solution

$$u = u_s \equiv \max \left\{ 0, \frac{\theta w + \chi \bar{u}}{\chi + q} \right\}, \quad \chi \equiv \left[ \frac{\beta}{q(1 - \beta)} \right]^{\frac{1}{\psi - 1}}. \quad (2)$$

Desired food consumption increases in available funds ( $\theta$ ) and in the weight of food in utility ( $\beta$ ) and it declines in the price of food ( $q$ ). The implied non-food consumption is  $c = c_s \equiv \chi(u - \bar{u})$  and the implied indirect utility is  $U(c_s, u_s)$ .

The proximate consequence of excess food consumption is an increase in body mass, measured by the body mass index  $b$  (as weight/height<sup>2</sup>). By exerting effort (physical exercise), denoted by  $x$ , individuals can work off the calories from excess food consumption and control body weight. This effort, however, reduces utility and since it provides no immediate pleasure, the short-run self prefers to exert no effort.<sup>6</sup> In order to arrive at closed-form solutions, we assume that food consumption and exercise have a linear effect on body mass such that

$$b = \nu u - \epsilon x. \quad (3)$$

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<sup>6</sup>The assumption that the short-run self is a “coach potato” is a convenient normalization. It could be generalized by assuming that the short-run self likes some physical exercise but less than needed to achieve ideal body mass.

We could thus imagine that  $\nu$  is an “energy exchange rate” that converts units of food consumption into calories. The parameter  $\epsilon$  measures the efficacy of physical exercise in working off excess calories.

The long-run self faces the same instantaneous utility from consumption (1) and takes the future consequences of current actions into account. The long-run self considers the effect of accumulating excess body mass on health and longevity, spends some income on health care and saves some income for later use, particularly for remedying health deficits in old age. This means that the long-run self faces the budget constraint

$$\dot{k} = w + rk - c - qu - ph, \quad (4)$$

in which  $k$  is financial wealth,  $r$  is the interest rate,  $h$  is health expenditure, and  $p$  is the relative price of health.

Following the gerontological literature, human aging is conceptualized as the accumulation of health deficits. The health deficit index (or frailty index), denoted by  $D$ , measures the relative number of health deficits present in a person (from a long list of potential health deficits). As individuals age, health deficits grow at a natural rate  $\mu$  (Mitnitski et al., 2002). As in Dalgaard and Strulik (2014), we assume that health investments slow down the rate of health deficit accumulation. Moreover, excess body mass increases health deficit accumulation such that the evolution of deficits is given by

$$\dot{D} = \mu [D - Ah^\gamma + B(b - \bar{b}) - a], \quad b \geq \bar{b}. \quad (5)$$

The parameters  $A$  and  $\gamma$  determine the available medical technology, as explained in detail in Dalgaard and Strulik (2014). The parameter  $B$  determines the unhealthiness of excess body mass and  $\bar{b}$  denotes the “ideal” (healthy) BMI. To facilitate closed-form solutions, we assume a linear association between excess body weight and health deficits. This approximation ignores potential non-linear (convex) effects and the possibility that mild overweight could be healthy (Flegal et al., 2013). Here, we focus on individuals that are not underweight, which covers the average citizen as well as great parts of the body mass distribution in most modern societies. Allowing individuals to be underweight would require a functional form where deficits depend non-monotonously on  $b$ .

Without self-control problems, the long-run self experiences utility

$$U(c, u, x) = \frac{[(1 - \beta)c^\psi + \beta(u - \bar{u})^\psi]^{\frac{1-\sigma}{\psi}} - 1}{1 - \sigma} - \delta \left( \frac{D}{D_0} \right)^\eta x^\phi, \quad \eta, \phi > 1. \quad (6)$$

The disutility from exercise  $x$  is increasing in the number of health deficits present in a person and in the intensity of exercise. With self-control problems, individuals also suffer pain from not conceding to the cravings of the short-run self. Following Fudenberg and Levine (2006), let  $\omega$  denote the cost of self-control such that the long-run self maximizes lifetime utility

$$V = \int_0^T e^{-\rho t} \{U(c, u, x) - \omega [U(c_s, u_s) - U(c, u, x)]\} dt. \quad (7)$$

The term in square brackets in (7) reflects the difference between the utility desired by the short-run self  $U(c_s, u_s)$  and the utility derived from the actually realized choices  $U(c, u, x)$ . The parameter  $\omega$  measures the cost of self-control. The special case of  $\omega = 0$  captures the standard assumption of an individual with perfect self-control for which the model is reduced to a conventional life cycle model of health deficit accumulation. The main aim of the calibrated model will be to estimate the model with self-control costs  $\omega > 0$  and then to run counterfactual experiments by setting  $\omega = 0$  and to discover how individuals would behave and how long they would live if there were no self-control problems.

The current value Hamiltonian associated with problem (1)–(7) is given by

$$H = (1 + \omega)U(c, u, x) - \omega U(c_s, u_s) + \lambda_k [w + rk - c - qu - ph] + \lambda_D \mu [D - Ah^\gamma + B(\nu u - \epsilon x - \bar{b}) - a]. \quad (8)$$

The individual maximizes (8) by controlling consumption of food and non-food, health investments, and physical exercise. Since  $T$  is endogenous, this constitutes a free terminal time problem. The long-run self takes into account how health behavior affects the age at death. For simplicity, the model is deterministic and death occurs when  $\bar{D}$  health deficits have been accumulated. As shown in Strulik (2015) and Schuenemann et al. (2017b), death can be modeled as a stochastic event, for which the probability depends on the number of accumulated health deficits. This leads to increased mathematical complexity with, in the present context, little value added with respect to insights.

In the Appendix it is shown that the associated first order conditions and costate equations lead to the following solution.

$$u = c \left\{ \left[ \frac{pB}{\gamma Ah^{\gamma-1}} + q \right] \frac{(1-\beta)}{\beta} \right\}^{\frac{1}{\psi-1}} + \bar{u}, \quad (9)$$

$$x = \left\{ \left( \frac{D_0}{D} \right)^\eta \frac{p\epsilon B}{\gamma \delta \phi Ah^{\gamma-1}} (1-\beta) c^{-\sigma} \left[ (1-\beta) + \beta d^\psi \right]^{\frac{1-\sigma}{\psi}-1} \right\}^{\frac{1}{\phi-1}}, \quad (10)$$

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma} \left( r - \mu - \frac{\mu B \epsilon \eta x}{\phi D} \right), \quad (11)$$

$$\frac{\dot{c}}{c} = \frac{1}{\sigma} \left\{ r - \rho + (1-\sigma-\psi) \frac{\beta d^\psi}{(1-\beta) + \beta d^\psi} \cdot \frac{r - \mu}{(\psi-1) \left( 1 + \frac{q \gamma Ah^{\gamma-1}}{p \nu B} \right)} \right\}, \quad (12)$$

with  $d \equiv (u - \bar{u})/c$ . Since  $\psi < 1$ , the solution (9) shows that, *ceteris paribus*, individuals consume more exciting food if the price of food  $q$  is low, if the taste for food  $\beta$  is high, if obesity causes little health damage (i.e. if  $B$  is low), or if health deficits can easily be repaired (i.e. if the marginal productivity of health investments  $\gamma Ah^{\gamma-1}$  is high or the price of health care  $p$  is low). Individuals also tend to consume more food if non-food consumption  $c$  is high because the two goods are not perfect substitutes. This alone would indicate that richer individuals consume more food. However, there is a counterbalancing effect because richer individuals tend to invest more in health. The aggregate effect of income on food consumption is thus ambiguous and which effect dominates is an interesting question that will be answered with the calibrated model in Section 3.

Since  $\phi > 1$ , the solution (10) shows that, *ceteris paribus*, individuals exert more physical exercise if they are healthy (i.e. if  $D$  is low), if, at any given health status, the utility cost of exercise is low (if  $\delta$  and  $\phi$  are low), if exercise is effective in reducing weight (i.e. if  $\epsilon$  is high), if obesity causes much health damage (i.e. if  $B$  is high) or if health deficits are hard to repair (i.e. if the marginal productivity of health investments  $\gamma Ah^{\gamma-1}$  is low or the price of health care  $p$  is high). Again, we observe counterbalancing effects of non-food consumption  $c$  and health expenditure on exercise such that the income effect is *a priori* ambiguous.

For  $x \rightarrow 0$ , equation (11) collapses to the “Health Euler” for the lifetime trajectory of health expenditure, as derived and explained in Dalgaard and Strulik (2014),  $\dot{h}/h = (r - \mu)/(1 - \gamma)$ . It shows that health expenditure increases with age if the return on financial investments  $r$  exceeds the natural rate of health deficit accumulation  $\mu$ . When individuals exercise (i.e. for  $x > 0$ ),

health investments increase at a lower rate than in the standard model. For  $\beta \rightarrow 0$ , i.e. when there is no joy from food consumption, equation (12) reduces to the standard Ramsey rule for non-food consumption  $\dot{c}/c = (r - \rho)/\sigma$ .

An interesting feature of the solution (9)–(12) is that it is independent from the self-control cost  $\omega$ . This perhaps unexpected result becomes obvious once we consider that instantaneous desires should not affect the intertemporal allocation, i.e. the *time derivative* of life cycle choices. This does, of course, not mean that the optimal life time paths are independent from  $\omega$ . Self-control affects the *level* of life cycle choices through the terminal condition. The terminal condition requires that the Hamiltonian at the time of death is zero, which can be equivalently written as in (13):

$$0 = \bar{H} \equiv \frac{H(T)}{1 + \omega} = U(c(T), u(T)) - \frac{\omega}{1 + \omega} U(c_s, u_s) \quad (13)$$

$$+ \frac{\lambda_k(T)}{1 + \omega} [w(T) + rk(T) - c(T) - qu(T) - ph(T)] + \frac{\lambda_D(T)}{1 + \omega} \mu [D - Ah(T)^\gamma + Bu(T) - a].$$

Notice that less self-control, i.e. a higher value of  $\omega$ , amplifies the negative impact of short-run desires  $U(c_s, u_s)$  on  $\bar{H}$  and that it reduces the positive impact of wealth accumulation ( $\lambda_k \dot{k}$ ) and health deficit accumulation ( $\lambda_D \dot{D}$ ). How exactly life cycle plans are affected by self-control can only be determined by numerical analysis. The other boundary conditions are that the optimal life cycle trajectory has to fulfil  $k(0) = k_0$ ,  $D(0) = D_0$ ,  $k(T) = \bar{k}$ , and  $D(T) = \bar{D}$ .

### 3. CALIBRATION

The model is calibrated to match initial deficits  $D_0$ , final deficits  $\bar{D}$ , health investments, food consumption, exercise, BMI, and the expected age at death of a single, white, 20-year-old U.S. American male in the year 2010. From Mitnitski et al. (2002), I take the estimate for the rate of aging,  $\mu = 0.043$ .<sup>7</sup> I set  $r$  to 0.07 according to the long-run real interest rate (Jorda et al., 2017) and  $\rho = r$  as in Dalgaard and Strulik (2014). In the year 2010, the average life expectancy of a 20-year-old American male was 57.1 years, i.e. the expected age at death was 77.1 (NVSS, 2014). From Mitnitski et al. (2002), I infer terminal health deficits  $\bar{D} = D(77.1) = 0.106$  and initial health deficits  $D(0) = D(20) = 0.0273$ . In order to get an estimate of  $a$ , I assume that before the 20th century, the impact of medical technology on adult mortality was virtually zero and

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<sup>7</sup>Mitnitski et al. (2002) estimate health deficit accumulation for Canadian men. Deficit accumulation within the USA and Canada appears to be similar enough to justify it as a good approximation for the U.S. (Rockwood and Mitnitski, 2007).

overweight was not an issue for society at large. According to Haines (1979), the life expectancy of a white 20-year-old American was 40.2 years in the mid-19th century. I thus set  $a$  such that a person who is not overweight and has no access to life prolonging medical technology expects to die at age 60.2.

When the individual is between 20 and 65 years old, I set  $w = 27,928$ , which is the average labor income for single men in the year 2010 (BLS, 2012). For older individuals, I set  $w = 0.45 \cdot 27,928$  using an average replacement rate of 0.45 from the OECD (2016). In order to assure that the savings motive is confined to that of health and consumption expenditure, I assume that the initial and final capital stock are zero. I assume for the benchmark case that all labor income and pension payments are liquid but that capital income is illiquid, i.e.  $\theta = 1$ . Furthermore, I set the healthy BMI  $\bar{b}$  to 22, a value at the center of the BMI range regarded as healthy (WHO, 2018), and normalize the price of health and food to unity,  $p = q = 1$ . This is an interesting benchmark case because it eliminates any price channel through which individuals may have an incentive to consume more food or spend less on health. We later investigate the sensitivity of results with respect to alternative values for prices and the other prespecified parameters.

The remaining parameters are estimated jointly for specific values of self-control  $\omega$  such that the predicted life cycle trajectories match observed age-dependent food expenditure, exercise, health expenditure, and BMI. In order to quantify  $\omega$  and to meet the involved parameter uncertainty, I tried a series of values and found that a value of about 0.75 constitutes a natural upper bound in the sense that then all excess body weight of the calibrated average American is explained by lack of self-control (see below). I thus run most experiments for  $\omega \in \{0.25, 0.5, 0.75\}$ , conceptualized as high, medium, and low self-control and define the benchmark case as medium self-control ( $\omega = 0.5$ ).

As a stylized fact, as individuals age, they spend more on health, exercise less, and spend a smaller fraction on food while there seems to be little variation in BMI across the ages. For health care expenditure, the calibration matches average expenditure of male Americans in the year 2010 at the age of 35 and 70 (MEPS, 2010).<sup>8</sup> Physical exercise is measured in metabolic equivalents (METs), defined as the energy cost of a given physical activity divided by energy

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<sup>8</sup>The health data from MEPS (2010) represent total health services including inpatient hospital and physician services, ambulatory physician and nonphysician services, prescribed medicines, home health services, dental services, and various other medical equipment and services that were purchased or rented during the year.

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 across ages. The average U.S. American 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., 2003). Studies from the UK (Townsend et al., 2015) and Canada (Statistics Canada, 2007) suggest that physical 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, the calibration matches this age-gradient together with the average level of physical activity.

As individuals age, they also tend to consume less food. I use data from BLS (2015) and try to match the food expenditure share at age 20, 40, and 60. The calories consumed from fast food decline faster with age than aggregate food expenditure, for men, by about factor 2.3 from age 30 to age 60 and above (CDC, 2013). This feature is approximated (but not calibrated) by the prediction that the expenditure share of exciting food  $u - \bar{u}$  declines much faster than the expenditure share of all food  $u$ . The effects of declining food consumption and declining exercise seem to roughly neutralize each other since there is no pronounced age gradient of obesity (CDC, 2016). While the CDC reports period data, a study by Cook and Daponte (2008) suggests that the secular increase of body weight is largely driven by period effects rather than cohort or age effects. The calibration matches the average BMI of American men in 2011 of 28.7 (CDC, 2016). Finally, I require that the age of death increases by 2.7 years when  $B$  is reduced from its calibrated value to zero. This matches the estimated loss of 2.7 years of longevity that results from overweight ( $\text{BMI} \in [25, 30]$ ) for 20-39 year old American men according to Grover et al. (2015).<sup>9</sup>

Summarizing, the parameters,  $A$ ,  $B$ ,  $\beta$ ,  $\gamma$ ,  $\delta$ ,  $\epsilon$ ,  $\eta$ ,  $\phi$ ,  $\nu$ ,  $\sigma$ ,  $\psi$ , and  $\bar{u}$ , are calibrated jointly with  $\omega$  such that: the model predicts the actual accumulation of health deficits over a lifetime (as estimated by Mitnitski et al., 2002); death occurs at the moment when  $\bar{D}$  health deficits have been accumulated at age 77.1; health expenditure matches health care expenditure of American men in 2010 at the age of 35 and 70 (MEPS, 2010); the model approximates the food expenditure share at age 20, 40, and 60 (BLS, 2015); physical exercise equals a daily expenditure of 1.14 MET (Moore et al., 2003) and it declines by factor 2 from age 35 to age 70; the average BMI is

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<sup>9</sup>Other studies provide similar result for years of life lost due to obesity, see Fontaine et al. (2003); Peeters et al. (2003).

28.7 (CDC, 2016); there is no BMI trend over the life cycle, and overweight causes a loss of life of 2.7 years of life compared to lean BMI  $\bar{b}$  (Grover et al., 2015). This leads to the estimates:  $A = 0.00154$ ,  $B = 3.5 \cdot 10^{-5}$ ,  $\beta = 0.345$ ,  $\gamma = 0.225$ ,  $\delta = 4.6 \cdot 10^{-4}$ ,  $\epsilon = 7.55$ ,  $\eta = 2.75$ ,  $\phi = 2.95$ ,  $\nu = 0.0155$ ,  $\sigma = 1.13$ ,  $\psi = 0.70$ , and  $\bar{u} = 3,600$ .

The estimated value of  $\sigma$  is in line with empirical studies suggesting that the intertemporal elasticity of substitution is close to unity (Chetty, 2006). The implied price elasticity of food is -0.12 which is at the lower end of the empirical estimates of the demand elasticity for food compiled in Andreyeva (2010). The implied elasticity for exciting food  $u - \bar{u}$ , however, is -1.30 in the upper range of the empirical estimates of the price elasticities for beef, soft-drinks, and food away from home (Andreyeva, 2010). As another plausibility check of the calibration, I calculate 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, period utility is converted by the unit value of a “util”,  $u'(c)$ . The VOL at the initial age is obtained by applying the formula  $VOL = \int_0^T e^{-\rho(\tau-t)} U(c(\tau), u(\tau), x(\tau)) d\tau / [\partial U(c(0), u(0), x(0)) / \partial c(0)]$ . The benchmark calibration predicts a VOL of \$ 5.05 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.

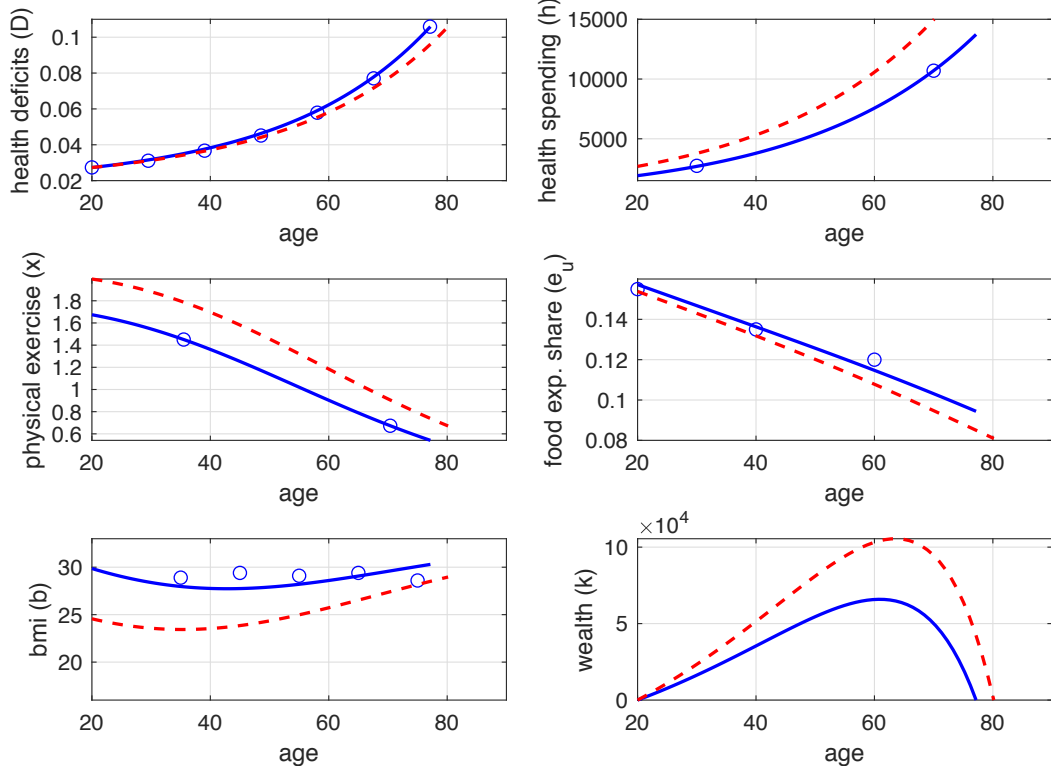
## 4. RESULTS

**4.1. Limited Self-Control, Food Consumption, Exercise, and Health Outcomes.** The life cycle health behavior of the Reference American and the implied accumulation of health deficits is shown in Figure 1 by blue (solid) lines. Dots indicate the targeted data points. With increasing age, the Reference American accumulates more health deficits, spends more on health, consumes less food, and exercises less, while BMI stays approximately constant throughout life. Through young and middle adulthood, he saves and accumulates wealth, which is depleted in old age in order to finance increasing health expenditure.

Dashed lines in Figure 1 show the outcome when  $\omega$  is set to zero and all other calibrated values are kept, i.e. when the self-control problem of the Reference American is removed. The elicited aggregate changes are summarized as case 1 in Table 1. Endowed with unlimited self-control, the individual saves more and spends more on health care, particularly in old age. Discounted



FIGURE 1: THE IMPACT OF SELF-CONTROL ON WEALTH AND HEALTH OVER THE LIFE CYCLE: BENCHMARK CASE



Blue (solid) lines: limited self-control ( $\omega = 0.5$ ;  $T + 20 = 77.1$ ). Red (dashed lines): perfect self-control ( $\omega = 0$ ;  $T + 20 = 81.1$ ). Dots indicate calibrated data points, see text for details.

lifetime health expenditure increases by 35 percent. With unlimited self-control, the individual spends less on food at all ages. The center-right panel in Figure 1 shows the effect of self-control on the expenditure share of food and Table 1 provides two measures for the change in the level of lifetime food expenditure. The first column shows that total food consumption declines by 7.8 percent. The second column shows that exciting food consumption, i.e. food consumption in excess of “subsistence” consumption  $\bar{u}$ , defined as  $\tilde{u} \equiv u - \bar{u}$ , declines by 39 percent. The center-left panel in Figure 1 shows that the individual with perfect self-control exercises more at any age. Lifetime exercise increases by 22.7 percent.

As a result of reduced food consumption and elevated exercise, the perfectly self-controlled individual is leaner at any age, and in particular, in young adulthood. As shown in the bottom-left panel, the perfectly self-controlled individual becomes mildly overweight only in old age. On average, lifetime BMI declines by -3.4 units. As a result of healthier behavior, the age at death

increases by 3.0 years, which constitutes, for the benchmark case, the estimated loss of longevity from having limited self-control.

Case 2 in Table 1 shows the results for when the model is recalibrated for a higher self-control cost of  $\omega = 0.75$ . This means that a larger part of observed excess BMI is explained by self-control problems and less is explained by standard preferences as, for example, by the taste for food ( $\beta$ ) and the dislike of physical exercise ( $\delta$ ). Obviously, in this case, the removal of the self-control problem, by setting  $\omega$  to zero, has a larger impact on the reduction of food intake and on the increase of exercise, savings, and health expenditure. As a result, BMI declines by 5 units and the age of death declines by 5 years. A reduction of BMI by 5 units (from 28.7) means that the individual achieves about his ideal weight once the self-control problem is removed. This is the reason why I argued above that  $\omega = 0.75$  operates as an upper bound for the self-control cost of the Reference American. This feature does of course not imply that  $\omega = 0.75$  should be considered as a general upper bound. In fact, other individuals in society could face much higher costs of self-control, which explains why they eat more, exercise less, and are heavier than the Reference American (see Section 4.2 below).

Table 1: The Impact of Self-Control on Health Behavior and Longevity

case	change	remark	$\Delta u/u$	$\Delta \tilde{u}/\tilde{u}$	$\Delta x/x$	$\Delta h/h$	$\Delta b$	$\Delta T$
1	–	benchmark ( $\omega = 0.5$ )	-7.8	-39.0	22.7	35.3	-3.4	3.0
2	$\omega = 0.75$	lower self-control	-11.9	-46.8	32.8	47.2	-5.0	5.0
3	$\omega = 0.25$	higher self-control	-4.3	-25.6	12.0	19.2	-2.0	1.4
4	$q = 0.5$	$u$ less expensive	-12.5	-47.2	23.0	35.0	-5.1	4.1
5	$p = 0.5$	$h$ less expensive	-7.8	-38.9	22.7	35.2	-3.4	3.0
6	$\theta = 0.5$	less liquid funds	-7.4	-37.2	21.3	32.8	-3.2	2.8
7	$\bar{b} = 20$	lower ideal weight	-7.7	-39.7	22.7	35.3	-3.2	2.8
8	$r = 0.06$	lower interest rate	-10.0	-41.1	24.0	32.0	-4.4	4.3
9	$\psi = 0.35$	low elast. of substitution	-7.4	-23.9	22.4	34.8	-3.3	3.4
10	$\bar{u} = 3000$	more exciting $u$	-11.8	-42.2	23.0	35.7	-5.4	2.8
11	$B = 5 \cdot 10^{-5}$	obesity more unhealthy	-6.8	-44.1	28.8	34.8	-3.1	3.1
12	$\eta = 2$	slow decline of $x$	-7.8	-38.9	22.5	35.3	-3.5	3.0
13	$\eta = 1.5$	slower decline of $x$	-7.8	-38.8	22.3	35.3	-3.6	3.0
14	$\bar{x} = 0.4$	joy of exercise	-7.8	-39.0	15.0	35.6	-2.8	2.9
15	$\bar{x} = 0.7$	more joy of exercise	-7.8	-39.0	10.0	35.7	-2.4	2.9

The table shows the impact of self-control by reducing  $\omega$  to zero;  $\Delta T$  is measured in years;  $\Delta b$  is measured in units of BMI;  $\Delta u/u$ ,  $\Delta \tilde{u}/\tilde{u}$ ,  $\Delta h/h$ , and  $\Delta x/x$  are measured in percent;  $\Delta \tilde{u}/\tilde{u}$  is the relative increase in exciting food consumption  $\tilde{u} \equiv u - \bar{u}$ .

Case 3 in Table 1 shows the results for when the model is recalibrated for  $\omega = 0.25$ . Naturally, the potential gain from a transition to perfect self-control is small when self-control is high to

begin with since overweight is to a large extent determined by preferences for food consumption ( $\beta$ ) and exercise ( $\delta$ ). Altogether, the elicited behavioral changes from eliminating self-control problems explain a reduction of BMI by 2 units and an increase of longevity by 1.4 years.

We next investigate the sensitivity of results with respect to other model parameters. In order to be brief, these robustness checks refer to the benchmark case of  $\omega = 0.5$ . Case 4 shows results when the price of food is 0.5 (instead of 1). The main effect of a lower price of food is a lower calibrated value of  $\beta$  in order to elicit the same food consumption as in the benchmark case. This means that relatively less of the eating behavior is explained by the preference for food and more by self-control. As a consequence, removing the self-control problem elicits a greater reduction of food consumption and BMI and leads to the prediction of a greater gain in terms of longevity (4 years instead of 3).

Case 5 shows the results for when the price of health care is 0.5 (instead of 1). Here, the lower price is almost entirely balanced by a lower estimated value of the level of medical technology  $A$  and causes only insignificant changes in health behavior and outcomes. Case 6 sets  $\theta = 0.5$  (instead of 1), thereby assuming that less liquid funds are available to satisfy short-term desires. This means that self-control plays a relatively smaller role in explaining health behavior and consequently, the estimated changes in behavior from eliminating self-control problems are somewhat smaller than for the benchmark case. Case 7 assumes a lower value for healthy BMI by setting  $\bar{b} = 20$  (instead of 22). The recalibration requires a lower marginal impact of food consumption on weight gain ( $\nu$ ) and a lower marginal impact of exercise on weight loss ( $\epsilon$ ). Consequently, the predicted effects of removing self-control problems are somewhat smaller. It is, however, reassuring that the results are not very sensitive to the choice of  $\bar{b}$  since healthy BMI needs to be represented by a unique value in the model whereas its clinical definition covers an interval. The value we select has apparently only a small influence on the results. Case 8 considers a lower return on capital of 6 percent (instead of 7 percent). In this case, the prediction of an almost constant BMI throughout life is lost. Instead, BMI increases from 27 to 32 as individuals age. Apparently this feature leads to the estimate of a greater effect of removing self-control problems on the life cycle trajectory of BMI such that the predicted age of death increases by 4.3 (instead of 3) years.

Finally, we check the sensitivity of results with respect to some parameters that were estimated rather than pre-specified. Naturally this leads to a second best approximation of the targeted

outcomes. Case 9 and 10 show that results are not greatly affected if we assume a lower elasticity of substitution between exciting food  $u - \bar{u}$  and non-food  $c$  (by setting  $\psi = 0.35$  instead of 0.70) or if we assume that a greater part of all food consumption consists of exciting food ( $\bar{u} = 3000$  instead of 3600). In case 11, we assume a higher health damage due to overweight. By setting  $B = 5 \cdot 10^{-5}$  we assume that overweight costs the Reference American 3.8 years of life, a value at the upper end of Grover et al. (2015)'s confidence interval around the point estimate of 2.7 years lost for overweight men. The predicted years of life that could be gained by perfect self-control, however, remain relatively unaffected by this change (3.1 years instead of 3.0).

A potential concern could be that the age-pattern of exercise has been calibrated according to British/Canadian data and U.S. Americans could be different in this regard. We thus consider with case 12 and 13 different age-patterns of exercise by imposing different  $\eta$ 's. This parameter controls the impact of health deficits on exercise and thus the decline of exercise with aging (accumulation of health deficits). The size of  $\delta$  is adjusted such that individuals perform on average over the lifetime the same activity level as in the benchmark case. Case 12 sets  $\eta = 2$  implying that the exercise level declines by only 1/3 (instead of 1/2) between age 35 and age 70. Case 13 assumes  $\eta = 1.5$  and thus a 20% decline of the exercise level. We see that a slower decline of exercise with age mildly reduces the impact of self-control on lifetime exercise and average body weight and has insignificant impact on longevity.

Another concern could be that results are sensitive to the assumption that physical exercise is a joyless activity. We thus refine the utility function (6) such that

$$U(c, u, x) = \frac{[(1 - \beta)c^\psi + \beta(u - \bar{u})^\psi]^{\frac{1-\sigma}{\psi}} - 1}{1 - \sigma} - \delta \left( \frac{D}{D_0} \right)^\eta (x - \bar{x})^\phi, \quad \eta, \phi > 1, \quad (14)$$

in which the new parameter  $\bar{x}$  controls for pleasure from exercise. For activity levels  $x < \bar{x}$ , the individual experiences (on net) pleasure from exercise, which is declining in the intensity of exercise and health deficits;  $\bar{x}$  is the threshold at which exercise becomes (on net) painful, i.e. costly in terms of utility. These amendments lead to mild modifications of the optimal solution for exercise and the health-Euler equation (see Appendix) and the prediction that, ceteris paribus, individuals exercise more because they like it. When  $\bar{x}$  rises we thus need to adjust  $\delta$  and  $\eta$  in order to match mean lifetime exercise and the decline of exercise with age as calibrated for the Reference American. Case 14 in Table 1 shows results for  $\bar{x} = 0.4$ , implying that on average about 1/3 of exercise activities are motivated by the sheer joy of it and 2/3

are solely motivated by positive health effects. The recalibration provides the new estimates  $\delta = 7.6$  and  $\eta = 3.5$ . Case 15 shows results for  $\bar{x} = 0.7$ , implying that on average about 60 percent of exercise are motivated by the joy of exercising ( $\delta = 8.1$ ,  $\eta = 5.3$ ). Exercising for fun reduces markedly the extra exercise that is elicited from abolishing self-control problems;  $\Delta x/x$  increases by only 15 or 10 percent, respectively. As a result body size declines by less than without joy from exercising (by 2.8 or 2.4 units of bmi, respectively). Joy of exercising has also a small impact on health expenditure, which increases a bit more than in the benchmark experiment. The impact on health and longevity, however, is small. The estimated lifetime gain is now 2.9 years instead of 3.0 years.

Since the health effects of limited self-control run through several channels, it would be interesting to identify how much can be directly attributed to eating and exercising. In order to address this question, I run the model with perfect self-control and estimate the resulting lifetime trajectories for food demand and exercise as polynomial functions of age. It turns out that very good approximations ( $R^2 > 0.99$ ) are already obtained for cubic polynomials. I then feed the estimated  $u(age)$  and  $x(age)$  functions into the model and solve it again with and without self-control. As a result, I obtain the difference in behavior and health outcomes that is caused exclusively by self-control issues regarding savings and (old-age) health expenditure. The residual with respect to the original difference then identifies the health consequences that run directly through obesity (i.e. eating and exercising).

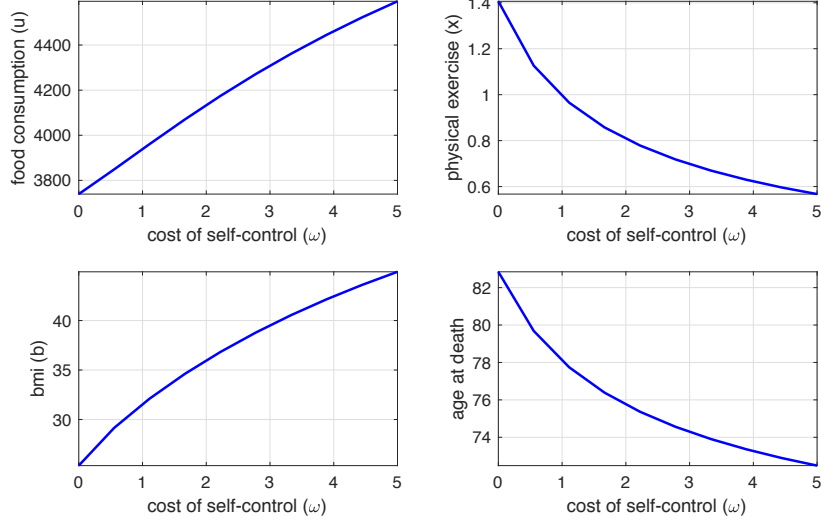
For the benchmark model (case 1), this experiment suggests that of the 3.0 life years lost by low self-control, 2.4 are explained by lower savings for old age and less health care expenditure while 0.6 (i.e. 20 percent) are explained by eating and exercising behavior. The obesity channel gains more power when the desire for food is more important for self-control problems. For example, in the calibration where the food price is 0.5 instead of 1 (case 4 in Table 1), the same experiment provides the result that 1.8 of 4.1 life years lost (i.e. 44 percent) are attributed to eating and exercising behavior.

To summarize, the model suggests that the Reference American could gain up to 5 years of life through perfect self-control, with an intermediate estimate of about 3 years, a result that appears to be relatively robust against parameter variation.

**4.2. Lack of Self-Control, Obesity, and Age at Death.** As argued above, the narrow upper bound of  $\omega$  estimated for the Reference American does not preclude that other individuals suffer

(much) greater self-control problems. In order to estimate how much self-control could contribute to explain the variance in obesity and health outcomes, we perform out-of-sample predictions by endowing the Reference American with different  $\omega$ 's (and, this time, by not re-calibrating the other parameters). This means that we consider individuals that are in every respect identical with the Reference American except for self-control.

FIGURE 2: SELF-CONTROL, OBESITY, AND AGE AT DEATH



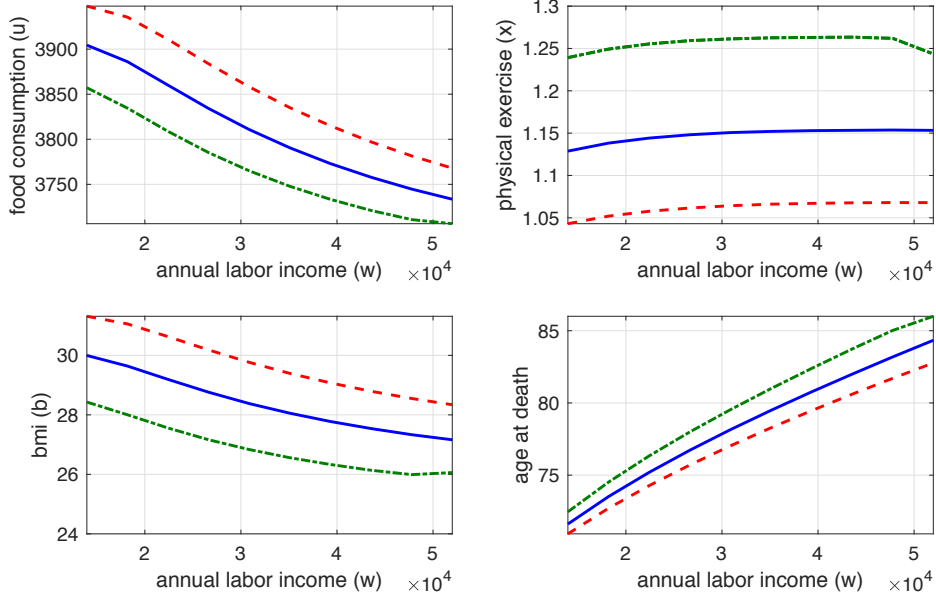
The figure shows predictions for the Reference American endowed with different costs of self-control.

Results are shown in Figure 2. With increasing self-control costs, individuals eat more and exercises less. Both effects contribute to increasing BMI, which increases from mild overweight to severe obesity as  $\omega$  goes from 0 to 5. The bottom right panel shows the associated age at death. Grover et al. (2015) estimate that for men aged 20 to 39, overweight (BMI between 25 and 30) reduces life expectancy by 2.7 years (CI 1.6 to 3.8), that obesity (BMI 30 to 35) reduces life expectancy by 5.9 years (CI 4.4 to 7.4), and that severe obesity (BMI above 35) reduces life expectancy by 8.4 years (CI 7.0 to 9.8). Gauged by these facts, the model gets the obesity gradient of longevity about right by varying the degree of self-control. It should be noted, however, that low self-control affects longevity not only through increased BMI. The generally increased desire for immediate gratification leads also to less savings and health expenditure.

**4.3. Socioeconomic Status, Self-Control, and Health Behavior.** We next feed different levels of annual labor income into the calibrated model. In Figure 3, blue (solid) lines show results for medium self-control ( $\omega = 0.5$ ). The model predicts, in line with the empirical evidence

(e.g. Cawley and Ruhm, 2012), that higher income is associated with healthier behavior. This outcome is perhaps most intuitive with respect to health care expenditure. As discussed in detail in Dalgaard and Strulik (2014), the income gradient predicted by the standard health deficit model follows from the feature that marginal utility from instantaneous consumption is strongly declining in expenditure while lifetime utility is roughly linear in lifetime years of consumption. The incentive to invest in health care in order to prolong life thus increases with increasing levels of income and consumption. This health care effect operates at all income levels and all degrees of self-control. Results for lower self-control (cost of self-control  $\omega = 0.75$ ) and higher self-control ( $\omega = 0.25$ ) are shown by red (dashed) and green (dash-dotted) lines in Figure 3.<sup>10</sup>

FIGURE 3: INCOME, SELF-CONTROL, BMI, AND AGE AT DEATH ( $\psi = 0.7$ )



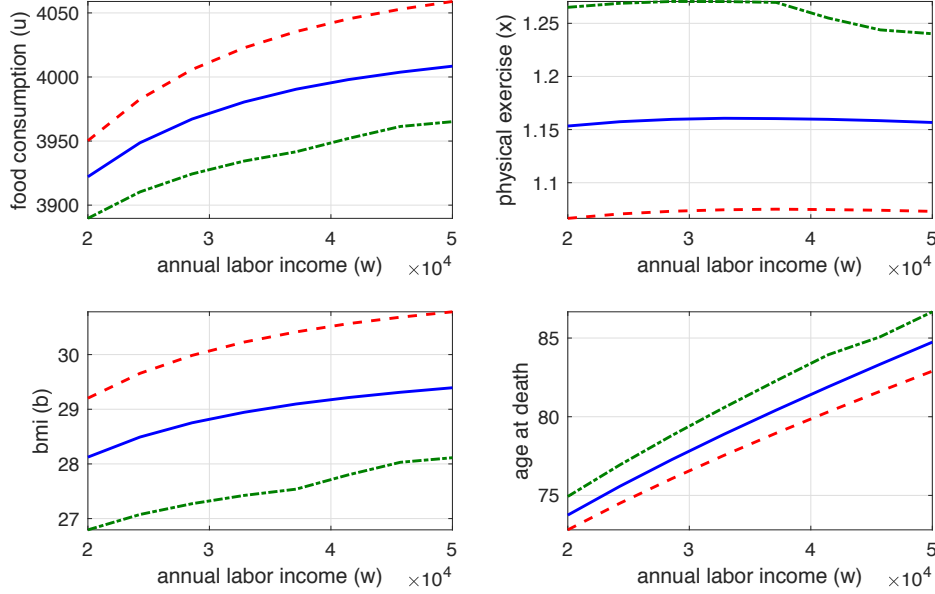
Blue (solid) lines: medium self-control ( $\omega = 0.5$ ); red (dashed) lines: lower self-control ( $\omega = 0.75$ ); green (dash-dotted) lines: higher self-control ( $\omega = 0.25$ ).

For eating behavior and physical exercise, the income mechanism is less obvious and indeed it is not entirely robust. Figure 3 shows that, for the benchmark calibration, food consumption declines with income while physical exercise increases mildly such that richer individuals are predicted to be leaner, *ceteris paribus*. As discussed in conjunction with equation (9), this outcome requires that the positive income effect on food consumption through its complementarity to non-food consumption is trumped by the income effect that runs through increasing health

<sup>10</sup>The income gradient of health and longevity would become steeper if we additionally assume that richer individuals do not only spend more on health but also have access to better health technology.

expenditure of the rich, which decreases food consumption. The benchmark calibration assumes a relatively high elasticity of substitution between exciting food and non-food. If the elasticity declines sufficiently such that, for example, an ice-cream and a new T-shirt are considered poor substitutes by the shopper, the result can be overturned.

FIGURE 4: INCOME, SELF-CONTROL, BMI, AND AGE AT DEATH ( $\psi = 0.35$ )



Blue (solid) lines: medium self-control ( $\omega = 0.5$ ); red (dashed) lines: lower self-control ( $\omega = 0.75$ ); green (dash-dotted) lines: higher self-control ( $\omega = 0.25$ ).

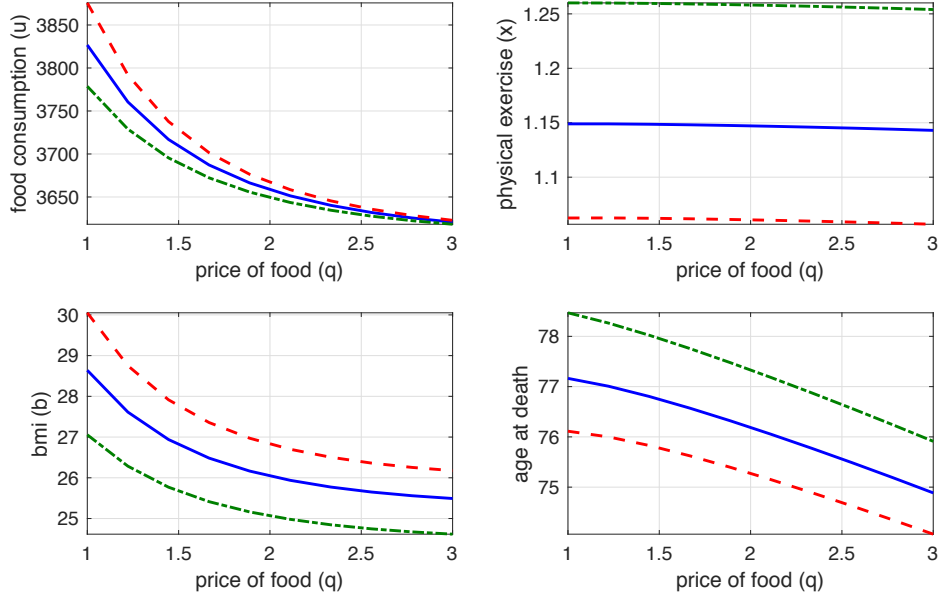
Figure 4 shows results for the same experiment when  $\psi$  is 0.35 (instead 0.7). Higher income is now associated with more food consumption and higher BMI. The income gradient of life expectancy, however, remains positive because of the dominating effect of income on health investments. Taken together, these results could motivate the frequently insignificant and inconclusive empirical findings on the income gradient of BMI (see the discussion in Cawley, 2015). In the model, the ambiguity would be explained by unobserved preference heterogeneity with respect to  $\psi$ .<sup>11</sup> The predictions for the impact of self-control on BMI and age at death, however, are hardly affected by the reversing income gradient. The robustness of the self-control effect on health behavior and health outcomes is visible in Figures 3 and 4 and by comparing case 1 and 9 in Table 1.

<sup>11</sup>Likewise, if  $\phi$  gets sufficiently low, richer individuals start to exercise less. A glimpse of this feature can be seen in the income-exercise curve for high self-control individuals (dashed-dotted line), which declines when income becomes sufficiently high.



**4.4. Secular Trends: Food Prices, Energy Density, and Medical Technology.** Average male BMI in the U.S. increased from 25.2 in 1975 to 28.9 in 2014 (NCD Risk Factor Collaboration, 2016). In this section, we investigate to what extent the present model can explain this trend based on food prices, energy density, and technological progress as well as the impact of these trends for individuals of varying degree of self-control.

FIGURE 5: FOOD PRICES, SELF-CONTROL, BMI, AND AGE AT DEATH



Blue (solid) lines: medium self-control ( $\omega = 0.5$ ); red (dashed) lines: lower self-control ( $\omega = 0.75$ ); green (dash-dotted) lines: higher self-control ( $\omega = 0.25$ ).

In Figure 5, we look at the impact of food prices on food consumption, BMI, and age at death for different levels of self-control. A trend of falling food prices is represented by a move from the right to the left in the four panels. When food prices are relatively high, there is little difference in eating behavior of individuals with low, medium, and high costs of self-control and differences in health outcomes are explained by differences in health expenditure and physical exercise. As food prices decline, all individuals eat more but individuals of low self-control (represented by red, dash-dotted lines) respond with particularly strong increases in food consumption.

Since declining food prices elicit only marginally more effort in physical exercise, they are associated with increasing BMI and declining longevity. BMI rises from 25.6 to 28.7, i.e. by about the actually observed increase from 1975 to 2014. Declining food prices also widen the self-control gradient of health outcomes. The difference in BMI between individuals of low-

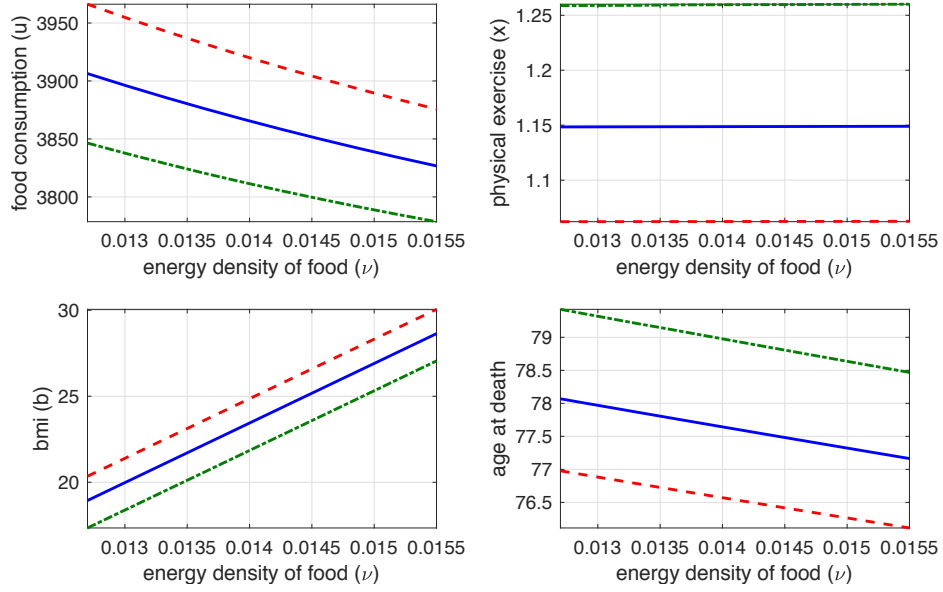
and high self-control increases from 1.5 to 3.0 units when prices decline by a factor of 3. The difference in age at death increases from 2 to 2.5 years.

A price decline from 3 to 1 would reflect, for example, an annual price decline of 2.75 percent from the year 1975 to the year 2014. A similarly high reduction of prices was observed for Coca Cola, which declined by an annual average rate of 2.5 percent between 1990 and 2007 (Christian and Rashad, 2009). However, the price of other food items declined by less or even increased. The model, by design, cannot capture substitution effects that are likely when the relative price of only some food items increases. Moreover, there are important timing issues in the trends of food prices and obesity, which are not addressed here (see the discussion in Ruhm, 2012, and Strulik, 2014). The idea that the *entire* BMI trend could be explained by food prices is thus hardly supported by the model.

Higher food prices reduce food consumption and weight and improve health and longevity but they are not necessarily conducive to higher welfare. In this respect they are inferior to policies that restrict continuous access to food. Restricted access, in the model conceptualized as a lower income share available for spontaneous consumption (lower  $\theta$ ), unambiguously improves health *and* welfare. Moreover, food price policy has detrimental distributional effects since it hits the poor (who spend relatively more on food) particularly hard, in particular if the resulting loss in real income is not compensated. According to model, an uncompensated doubling of  $q$  would reduce welfare, computed as the present value of lifetime utility, by 1.0 percent for the calibrated Reference American. It would reduce welfare of an individual with half of benchmark income (who is otherwise identical) by 3.2 percent. A fully compensated doubling of  $q$  could be imagined as a 100 percent tax of  $q$  with perfect individual-specific rebate of the collected tax revenue. In this case, the benchmark American loses 0.03 percent of welfare while the poor individual loses 0.3 percent of welfare.

Another potential candidate for secularly increasing body mass is an increasing energy density of food. In the model, this is captured by an increase of  $\nu$  such that the same food expenditure feeds more calories. Figure 5 shows the predicted behavior when  $\nu$  increases from 0.0115 to 0.0154, which could reflect, for example, an annual increase of  $\nu$  by 0.7 percent from 1975 to 2014. Individuals respond to increasing energy density by reducing their food expenditure. Yet this reduction is not enough to counterbalance the energy effect such that BMI increases from about 19 to 28.7 for an intermediate degree of self-control ( $\omega = 0.5$ ; solid lines). The increase

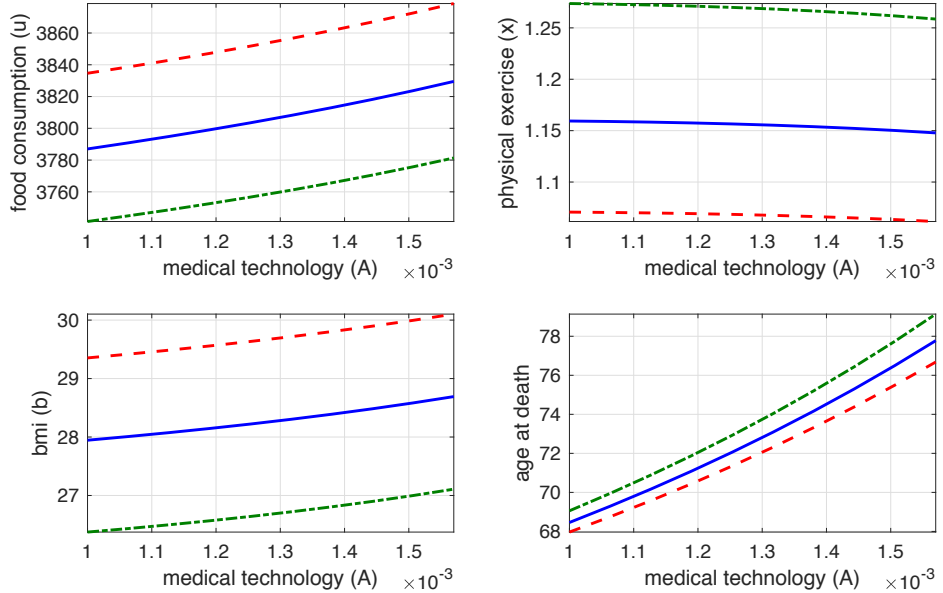
FIGURE 6: ENERGY DENSITY, SELF-CONTROL, BMI, AND AGE AT DEATH



Blue (solid) lines: medium self-control ( $\omega = 0.5$ ); red (dashed) lines: lower self-control ( $\omega = 0.75$ ); green (dash-dotted) lines: higher self-control ( $\omega = 0.25$ ).

in BMI is predicted to be about equally large for all degrees of self-control. This result would potentially change in a more detailed model that distinguishes energy density of exciting and unexciting food.

FIGURE 7: MEDICAL TECHNOLOGY, SELF-CONTROL, BMI, AND AGE AT DEATH



Blue (solid) lines: medium self-control ( $\omega = 0.5$ ); red (dashed) lines: lower self-control ( $\omega = 0.75$ ); green (dash-dotted) lines: higher self-control ( $\omega = 0.25$ ).

Finally, increasing body size could be motivated by improving medical technology: later-born people get fatter because they can. Specifically, innovations of the cardiovascular revolution that started in the 1970s reduced health damages from obesity (Hansen and Strulik, 2017). In the model, these innovations are captured by increasing  $A$  and it is evident from solution (9) and (10) that optimizing individuals respond to improving medical technology by eating more and exercising less. Figure 7 shows the predictions from the calibrated model when  $A$  increases from 0.0010 to 0.00157. Such an increase would reflect, for example, medical technical progress at an annual rate of 1.1 percent from 1975 to 2014. We see that increasing BMI is now associated with a strongly increasing life span. The behavioral changes for exercise and food consumption elicited by medical progress, however, are relatively small. According to the calibrated model, improving medical technology explains an increase of BMI of less than 1 unit, irrespective of the individual costs of self-control.

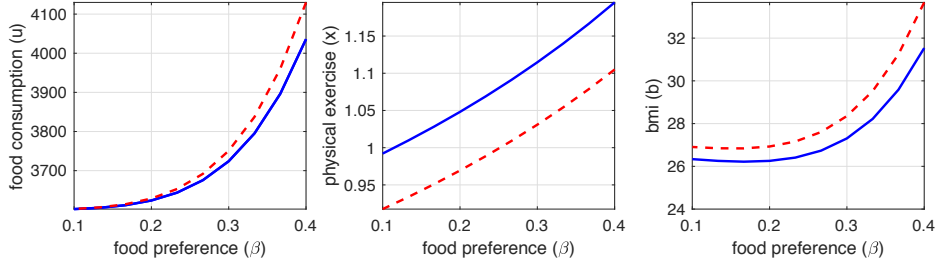
**4.5. Food Preferences vs. Self-Control.** Is a person obese because he highly enjoys eating or because he has a (severe) self-control problem? While this question is generally hard to address, our model can be used to develop a test based on revealed behavior with respect to physical exercise. Blue (solid) lines in Figure 8 show predicted food consumption and exercise levels when the Reference American is endowed with different  $\beta$ 's, i.e. when we consider persons that are in every respect identical to the Reference American but in their taste for (exciting) food. The model predicts that, *ceteris paribus*, persons who like eating more do not only eat more and are bigger, but they also exercise more. Working off excess calories is the rational response to overeating. As long as excess calories are not worked off completely, more food consumption is associated with higher BMI (right panel in Figure 8). This means that, if differences in excess food consumption are driven by differences in the taste for food, individuals who eat a lot and are thus bigger are predicted to exercise more than lean individuals.<sup>12</sup>

If, however, excessive food consumption is explained by low self-control, then individuals who eat more exercise less as shown in Figures 1 and 2. This is because low self-control operates in both domains of health behavior, it leads to more excessive food consumption *and* less exercise. These observations suggest that (after controlling for confounders like income and age) the cause of obesity can be identified by observing the association between food consumption or BMI and

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<sup>12</sup>An analogous argument can be made based on the utility cost of exercise ( $\delta$ ). The model predicts that persons who find exercising particularly hard and are thus bigger would consume less food than lean persons.

FIGURE 8: FOOD PREFERENCES AND SELF-CONTROL



Blue (solid) lines: benchmark calibration; red (dashed) lines: lower self-control ( $\omega = 0.75$ ).

exercise. The available literature lends support to the self-control channel: individuals who consume more calories and are heavier tend to exercise less (e.g. Schulz and Schoeller, 1994; Miller et al., 1990).

## 5. CONCLUSION

This paper has proposed a dual-self life cycle model of endogenous health behavior and endogenous health outcomes in order to better understand the drivers of excess body mass and its implied health outcomes. The dual-self model explains why overweight individuals think that they eat too much and exercise too little and why overeating and inactivity are frequently observed simultaneously. The suggested reason is lack of self-control that operates in both domains of human behavior. After calibrating the model for a Reference American with limited self-control, we considered the counterfactual experiment and removed the self-control problem to solve the life cycle problem again. These computational experiments suggest a gain in lifetime of up to 5 years, achieved by reduced food consumption, greater health investments, and more physical exercise.

The model captures the basic features of limited self-control on overweight and obesity and its implied health outcomes. Naturally, it could be refined and extended in several directions. The most obvious extension is perhaps to consider that food differs in price, energy content, and desirability. For evolutionary reasons it seems reasonable that energy-dense food like icecream or burgers are particular desirable from the perspective of the affective short-run self whereas healthy and perhaps more expensive food is preferred by the reflective long-run self. These extensions could potentially modify the estimated income gradient of unhealthy food consumption. Other extensions might consider that individuals adapt to increasing body weight and its repercussions on health (as in Schuenemann et al., 2017b) or that they become addicted to some

(particularly fatty or sugary) food or to the eating process as such (Olsen, 2011; Hedebrand et al., 2014).

The present study focuses on lifetime outcomes for adults. In this context, self-control is perhaps best treated as an exogenous character trait. At shorter time intervals of hours or days, however, it has been found that self-control is more like an endogenous resource which can be depleted and re-filled (Baumeister et al., 2007). The integration of short-run behavior capturing effort and fatigue in decision making (Dragone, 2009) could be another useful generalization of the theory.

In an intergenerational context, one could study the inheritance of low self-control through low socioeconomic status (Moffitt et al., 2011). The character trait of self-control is likely to be malleable in (early) childhood like other non-cognitive skills (Diamond et al., 2007; Heckman, 2007) and childhood self-control is a good predictor for adult self-control (Mishel et al, 1989). By treating self-control as exogenous by adulthood, the present paper has also highlighted the potentially large gains in health behavior, longevity, and welfare that can be achieved from policies that support the learning of self-control early in life.

## APPENDIX

**5.1. Derivation of (9)–(12).** The first order conditions with respect to consumption  $c$ , unhealthy consumption  $u$ , and health investments  $h$  are:

$$\frac{\partial U}{\partial c} = \frac{\lambda_k}{1 + \omega} \quad (\text{A.1})$$

$$\frac{\partial U}{\partial u} = \frac{\lambda_k q - \lambda_D \mu \nu B}{1 + \omega} \quad (\text{A.2})$$

$$\lambda_D \mu \gamma A h^{\gamma-1} = \lambda_k p \quad (\text{A.3})$$

$$\lambda_D \mu \epsilon B = (1 + \omega) \delta \left( \frac{D}{D_0} \right)^\eta \phi x^{\phi-1}. \quad (\text{A.4})$$

The costate equations for problem (8) are:

$$r \lambda_k = \rho \lambda_k - \dot{\lambda}_k \quad \Rightarrow \quad \frac{\dot{\lambda}_k}{\lambda_k} = \rho - r \quad (\text{A.5})$$

$$-(1 + \omega) \delta \left( \frac{D}{D_0} \right)^\eta x^\phi \frac{\eta}{D} \lambda_D \mu = \rho \lambda_D - \dot{\lambda}_D. \quad (\text{A.6})$$

Inserting  $\partial U / \partial c$ , computed from (6), and using the notation  $d \equiv (u - \bar{u}) / c$  condition (A.1) can be written as

$$(1 + \omega) \left[ (1 - \beta) + \beta d^\psi \right]^{(1-\sigma)/\psi-1} (1 - \beta) c^{-\sigma} = \lambda_k \quad (\text{A.7})$$

and log-differentiation provides

$$\frac{\dot{\lambda}_k}{\lambda_k} = \left( \frac{1 - \sigma}{\psi} - 1 \right) \left[ (1 - \beta) + \beta d^\psi \right]^{-1} \beta \psi d^{\psi-1} \frac{\dot{d}}{d} - \sigma \frac{\dot{c}}{c}. \quad (\text{A.8})$$

Inserting  $\partial U / \partial u$ , computed from (6), in (A.2) and substituting  $\lambda_D$  from (A.3) and  $\lambda_k$  from (A.7) provides

$$d = \left\{ \left[ q + \frac{\beta p \nu}{\gamma A h^{\gamma-1}} \right] \frac{1 - \beta}{\beta} \right\}^{\frac{1}{\psi-1}}. \quad (\text{A.9})$$

Inserting the definition of  $d$  and solving for  $u$  provides (9) in the text. Differentiation of (A.9) with respect to age provides:

$$\frac{\dot{d}}{d} = \frac{1 - \gamma}{\psi - 1} \left[ 1 + \frac{q \gamma A h^{\gamma-1}}{p B \nu} \right]^{-1} \frac{\dot{h}}{h}. \quad (\text{A.10})$$

And differentiation of (A.3) with respect to age provides:

$$\frac{\dot{h}}{h} = \frac{1}{1 - \gamma} \left( \frac{\dot{\lambda}_D}{\lambda_D} - \frac{\dot{\lambda}_k}{\lambda_k} \right). \quad (\text{A.11})$$

Inserting  $\lambda_D$  from (A.4) into (A.6) provides:

$$\frac{\dot{\lambda}_D}{\lambda_D} = \rho - \mu - \frac{\mu \epsilon \eta B x}{\phi D}. \quad (\text{A.12})$$

Inserting (A.5) and (A.12) into (A.11) provides (11) in the text. Inserting (A.10) and (11) into (A.8) provides (12) in the text. Inserting (A.3) into (A.5) provides:

$$(1 + \omega)\delta \left( \frac{D}{D_0} \right)^\eta \phi x^{\phi-1} = \frac{\lambda_k p \epsilon B}{\gamma A h^{\gamma-1}}. \quad (\text{A.13})$$

Substituting  $\lambda_k$  from (A.7) and solving for  $x$  provides (10) in the text.

**5.2. Joy from Exercise.** Recalculating these computation when individuals experience positive utility from exercise, as in (14), we obtain the solution for exercise (A.14) and the health-Euler equation (A.15). The rest of the model is maintained as in the main text.

$$x = \left\{ \left( \frac{D_0}{D} \right)^\eta \frac{p \epsilon B}{\gamma \delta \phi A h^{\gamma-1}} (1 - \beta) c^{-\sigma} \left[ (1 - \beta) + \beta d^\psi \right]^{\frac{1-\sigma}{\psi}-1} \right\}^{\frac{1}{\phi-1}} + \bar{x}, \quad (\text{A.14})$$

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma} \left( r - \mu - \frac{\mu B \epsilon \eta (x - \bar{x})}{\phi D} \right). \quad (\text{A.15})$$



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