Hooked on Weight Control: An Economic Theory of Anorexia Nervosa, and Its Impact on Health and Longevity

Holger Strulik

GEORG-AUGUST-UNIVERSITÄT
GÖTTINGEN

Hooked on Weight Control: An Economic Theory of Anorexia Nervosa, and its Impact on Health and Longevity[*]

Holger Strulik[†]

September 2021

Abstract. In this paper, I combine economic theories of health behavior and addiction in order to explain the phenomenon of anorexia nervosa and its impact on health and longevity. Individuals consume normal goods and foods and can work off excess calories with physical exercise. There exists a healthy body mass index and deviations from it increasingly cause health deficits due to obesity or underweight. There exists also a subjective target weight and being heavier than target weight causes a loss of utility from body image. Individuals for whom the utility loss from missing target weight is large exert more weight control, i.e. they eat less and exercise more. Anorexia is initiated in individuals who are particularly successful in weight control and prone to addiction. Addiction to weight control motivates anorexic individuals to perpetually adjust their target weight downwards and to eat less and exercise more. With declining weight, health deficits accumulate faster and mortality risk rises. I calibrate the model to a reference American with bmi 28. Due to weight loss addiction, the bmi gradually declines to a level of 15 and causes a loss of 21 years of life expectancy at the age of 20.

Keywords: weight control, addiction, eating disorder, physical exercise, health deficits, mortality.

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

"It is the way it starts as a need, a compulsion to lose weight or gain control, then the habits you develop to fulfill that become ingrained and the compulsion is no longer owned by you, you become addicted and sticking to your rules is the most important thing. It also makes recovery hard because trying to ignore the compulsions you feel is like trying to get a heroin addict to turn down heroin- near on impossible."

(Anonymous anorexia patient, quoted from Godier and Park, 2015)

1. Introduction

In this paper, I propose an economic theory of anorexia nervosa (henceforth anorexia) and its impact on health and longevity. Anorexia is a severe and potentially life-threatening eating disorder, which affects about 0.1 percent of the U.S. population, with similar prevalence rates in other Western countries (GBD, 2016). In order to be diagnosed with anorexia, a person must meet the following DSM-5 criteria: (i) restriction of energy intake leading to significantly low body weight (ii) intense fear of gaining weight or becoming fat, (iii) distorted body image perception, i.e. perceiving oneself as too fat when being underweight (APA, 2013). A significantly low body weight is commonly conceptualized as 15% or more below the expected weight for one's age, gender, and height and expressed in a body mass index (bmi) below 17.5 kg/m^2. In the brain, anorexia is associated with increased activity of the nucleus accumbens, a region of the ventral striatum that controls reward and motivation. The cause of the disease is basically unknown and treatment remains ineffective for most patients, with only 30 percent fully recovering over the course of their life with various behavioral therapies (Nestler, 2003). It has been estimated that more than half the risk of developing anorexia is due to genetic factors (Bulik et al., 2000). Anorexia is about ten times more prevalent among women than men. Other risk factors are being an adolescent, having an obsessional style, and the exposure to thin peers and role models.

Anorexia exhibits the highest mortality rate of any psychiatric illness. Up to 10 percent of anorexia patients are estimated to die from suicide (Pompili et al., 2006). Here, we focus on the long-run consequences that are caused by the inadequate supply of energy and nutrients and lead to faster health deficit accumulation, frail bodies, and premature death by 'natural causes' such as cardiac diseases and organ failure. Among the long-term effects of anorexia are osteoporosis, cardiovascular disturbances, diabetes mellitus, thyroid disorders, and gastrointestinal disorders. About 80 percent of anorexic patients have cardiac complications, and most sudden cardiac death in anorexic patients is due to abnormal heartbeat (Meczekalski et al., 2013; Casiero and

Frishman, 2006). The mortality rate for all causes of death is estimated to be up to six times higher than that of the general population and Canadian women diagnosed with anorexia by the age of 20 had a predicted life expectancy of 36 years compared to 60 years in the general female population (Harbottle et al., 2008; Meczekalski et al., 2013).

For the general population, it has been shown that the trait of high self-control is predictive for restrained eating, exercise and weight loss (Crescioni et al., 2011; Cobb-Clark et al., 2014; Stutzer and Meier, 2016). Since anorexia patients are particularly "successful" in weight loss, it has been argued that individuals with anorexia are characterized by a high levels of high self-control (Fairburn et al., 1999; Steinglass et al., 2012; Butler and Montgomery, 2005) and that high self-control partly explains the persistence of the disease and the resistance to treatment (Pauligk et al., 2021). Walsh (2013) argues that anorexia is initiated in young women by initial successes in weight control, which then 'takes on a life of its own and evolves into an unrelenting pursuit that becomes the individual's primary focus.' Reinforcement mechanisms make restrained eating and exercise a habit and anchored in neural mechanisms that underlie the formation and persistence of habits. This way, Walsh argues, anorexia resembles the neural mechanisms underlying addiction to substance abuse.

The similarity of anorexia to addiction and substance dependence has been highlighted in a series of studies (e.g. Barbarich-Marsteller et al., 2011; Kaye et al., 2013; Godier and Park, 2014, 2015; Compan et al., 2015). The onset of the anorexia, characterized by an initial success in weight loss is experienced as rewarding and pleasurable (resembling the initial phase of an addiction). The positive experiences motivate anorexic individuals to continue setting increasingly more ambitious weight goals (resembling the tolerance effect in addiction). As the disease continues, individuals find it increasingly more difficult to give up restrained eating and to return to healthy behavior and healthy body weight (resembling the withdrawal effect in addictions). The initially positive feelings associated with weight loss, however, cannot be maintained and are replaced by negative feelings associated with not being able to reach the increasingly ambitious target weight. The target weight adjustment implies that, despite increasing emaciation, individuals perceive themselves as to fat (distorted body image) such that the relentless pursue of weight loss turns into a joyless and painful activity. It is thus understandable that anorexia patients perceive their behavior as an illness and want to be cured from the disease (resembling substance addicts). Aside from searching professional help they form self help groups such as

anorexic anonymous that resemble similar self help groups of substance addicts. A high level of self-control, however,is uncommon in substance addicts. It is a distinctive feature of anorexia. In fact, it has been argued that high-self control protects individuals with anorexia from substance abuse (Kaye et al., 2013).

In the health-economic theory of anorexia proposed below, I take these features into account. I set up a life cycle model of endogenous health deficit accumulation (based on Dalgaard and Strulik, 2014), in which individuals consume food and non-food goods and invest in their health and exercise. Body size increases with food consumption and declines with exercise. Deviations from a healthy bmi accelerate the speed of health deficits accumulation and, at any age, survival depends on the level of health deficits. Additionally, individuals derive positive or negative utility from body image, depending on the relative deviation of actual bmi from target bmi. Target bmi may deviate from the healthy bmi because it is socially constructed. In non-anorexic individuals target bmi is constant or changes only occasionally and exogenously. Anorexia is characterized by a habituation process (addiction) that leads to a continuous reduction of target bmi. It is initiated by weight loss successes in individuals who are (genetically) susceptible to target weight updating and for whom body imagine is an important factor of experienced utility, a feature which implies a high level of self-control in terms of body weight. Individuals are assumed to be perfectly rational except for the fact that they cannot plan their target weight updating. I model this as an imperfectly controlled addiction as proposed in Strulik (2018, 2021). Anorexia is conceptualized as a disease because addiction is unplanned and involuntary. This feature deviates from the standard economic theory of rational addiction built on Becker and Murphy (1988) where addiction is an optimally planned process and therefore cannot be understood as a disease.

While there exists a rich economic literature on overweight and obesity, relatively little attention has been paid to the phenomenon of underweight. Ham et al. (2013) show that bulimia nervosa is to a large extent driven by path dependence and argue that the eating disorder shares many characteristics with common addictions (substance abuse). Costa-Font and Jofre-Bonet (2013) show that European women are more likely to be anorexic when they are exposed to low peer-group bmi (whereby anorexia is defined as bmi below 17.5 and seeing oneself as fine or too fat). Arduini et al. (2019) show that eating disorders (purging) of female adolescents are influenced by peers' body size through interpersonal comparisons. Goldfarb et al. (2009) propose a

static model of rational thinness and underweight. Dragone and Savorelli (2011) developed a theory of body size evolution where food consumption causes utility, deviations from healthy body weight as well as deviations from socially desirable weight cause utility losses, and eating increases body weight. They show that one of several steady states to which rational individual converge is characterized by underweight and underconsumption of food. In a society in which individuals are heterogenous with respect to healthy weight and face the same exogenously determined desirable weight, a higher desirable weight reduces the prevalence of unhealthy thinness but may exacerbate the prevalence of obesity. Strulik (2014) proposed a theory in which social approval of physical appearance is endogenous and shows how this feature explains the obesity epidemic. The theory focusses on obesity but also considers the whole distribution of body weights in society.

The paper is organized as follows. In the next section, I set up the theoretical model, derive the implied dynamics for life cycle behavior, and obtain analytical solutions for optimal food consumption and optimal exercise. In Section 4, I calibrate a benchmark version of the model to an average 20-year-old American male such that predicted health behavior supports a bmi of 28.7 and a life expectancy at 20 of 57 years. An otherwise identical individual endowed with a greater importance of body image (greater self-control) is predicted to achieve a bmi of 19 and a 2.5 years higher life expectancy. In Section 4, I show that if this individual is additionally susceptible to anorexia, a process sets in such that target bmi is continuously reduced to approach a value around 14 and eating is restricted and exercise increased such that actual bmi reaches a trough at 15. The entailed acceleration of health deficits reduces life expectancy at 20 to 36 years.

This paper was partly inspired by the fate of Bahne Rabe, a 2 meter tall German rower who won the Olympic gold medal with the eights in 1988 when he had a bmi of 23.7 and who died of a lung infection in 2001 shortly before his 38th birthday with a bmi of 14.6 (Kurbjuweit, 2001). In fact, anorexia appears to be particularly prevalent among professional athletes (Sudi et al., 2004) and using the model I offer an explanation for the phenomenon of anorexia athletica. I then turn to a calibration for an adolescent girl and try to give an explanation of why the disease is much more common among women than men. I use this model version to discuss the failure and success of two stylized therapies (target weight reset and body image reset). Section 5 concludes the paper.

2. The Model

Individuals are assumed to consume non-food goods c and food u. For non-food consumption we maintain the conventional assumption of an iso-elastic utility function, $(c^{1-\sigma} - 1)/(1 - \sigma)$, in which σ is the inverse of the elasticity of intertemporal substitution. For food consumption we assume a quadratic utility function, $\alpha u - \beta/2u^2$. This form allows us to derive some results analytically. It also captures the natural feature that for food consumption (in contrast to non-food consumption) there exists satiation. After a certain threshold is crossed (here, $u = \alpha/\beta$), utility from eating would decline if individuals were forced to eat even more. Individuals have the possibility to work off body weight by physical exercise x. Physical exercise may provide utility to some individuals and disutility to others. In order to avoid unnecessary case differentiation, we assume that exercise competes with other more pleasurable ways to spend leisure time. Normalizing the utility from these other (sedentary) forms of leisure to zero, we assume a quadratic disutility function for exercise. Additionally, we assume that the disutility from exercise increases as the individual ages and becomes more frail. With D denoting health deficits, the utility function is given by $-\delta/2(D/\underline{D})^\eta x^2$, in which $\underline{D}$ is a normalizing constant, specifying the state of best health. Summarizing, instantaneous utility is given by

$$\tilde{U}(c, u, x) = \frac{c^{1-\sigma} - 1}{1 - \sigma} + \alpha u - \frac{\beta}{2}u^2 - \frac{\delta}{2}\left(\frac{D}{\underline{D}}\right)^\eta x^2. \tag{1}$$

In order to avoid unnecessary complications, I assume that all individuals share the same given height but may differ in weight. In order to arrive at closed-form solutions, I assume that food consumption and exercise exert linear effects on body mass b, measured by the body mass index (bmi), such that

$$b = \nu u - \epsilon x. \tag{2}$$

We can imagine ν is an "energy exchange rate" that converts units of food consumption into calories. The parameter ϵ measures the efficacy of physical exercise in working off excess calories. Since height is a given constant, (changes of) weight and bmi can be considered to be equal up to a constant and they are treated interchangeably.

Individuals face the budget constraint

$$\dot{k} = w + rk - c - qu - ph, \tag{3}$$

in which k is financial wealth, r is the interest rate, w is labor income, h is health expenditure, and q and p are the relative prices of unhealthy goods and health care.

Following the gerontological literature, physiological aging is conceptualized as the accumulation of health deficits. The 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 μ (Mitnitski et al., 2002). As in Dalgaard and Strulik (2014), we assume that health investments slow down the rate of health deficit accumulation. Moreover, deviation from healthy body mass increases health deficit accumulation such that the change in health deficits is given by

$$\dot{D} = \mu \left[D - Ah^\gamma + \frac{B}{2}(b - b_H)^2 - a \right]. \tag{4}$$

The parameters A and γ determine the available medical technology, as explained in detail in Dalgaard and Strulik (2014). The parameter B determines the unhealthiness of deviating from (ideal) healthy BMI b_H. In contrast to a related model on obesity (Strulik, 2019), we assume that health damage increases quadratically in the deviation from ideal BMI, in order to capture in a convenient way that the marginal damage from gaining or losing body weight is greater when the individual is already severely overweight or underweight.

The health deficit model is particularly useful to discuss anorexia since its physiological foundation is based on the depletion of redundancy in human bodies (Gavrilov and Gavrilova, 1991; Dalgaard et al., 2021). Insufficient nutrition leads to faster depletion of redundancy in organ and tissue reserve such that health deficits accumulate faster in emaciated bodies, expressed as an increasing frailty index D. The accumulation of health deficits reduces survival probability and ultimately leads to death. Death is a stochastic event such that, at any age, the survival probability is given by $S(D)$ with $S'(D) < 0$. This modeling captures the biological approach to aging, which replaces chronological age as a proximate determinant of death by the loss of bodily function as a deep determinant of death (Arking, 2006). We exclude the possibility of an infinite life. This means that there exists an upper limit of health deficits $\bar{D}$ beyond which life ends with certainty, $S(\bar{D}) = 0$. Formally, the human life cycle is thus characterized as a free terminal time problem.

Individual utility is also affected by physical appearance. Specifically, we assume that utility declines in body mass and that individuals have a target bmi b_T defined as the threshold at

which satisfaction with appearance turns into dissatisfaction with appearance. We conceptualize *perceived* overweight as relative deviation of actual bmi from individual-specific target bmi b_T. Total instantaneous utility is thus given by

$$U = \tilde{U}(c, u, x) - \omega \left(\frac{b - b_T}{b_T} \right). \tag{5}$$

The parameter $\omega \geq 0$ measures how much an individual cares about physical appearance. The individual assignment of great importance of physical appearance is thus another motive of restrained food consumption and physical exercise, aside from the consideration of health effects. Target bmi b_T may or may not coincide with healthy bmi b_H such that $b_T = b_H/z$. For $z > 1$, target bmi is lower than healthy bmi, perhaps because celebrities or other peers convey the image of being thin as desirable. While healthy bmi is conceptualized as a given population-specific constant, target bmi is conceptualized as an individual-specific and malleable expression of preferences.

The subutility $I \equiv -\omega(b - b_T)/b_T$ is the model's conceptualization of *body image*, defined as the thoughts, feelings and perception of the aesthetics or attractiveness of an individual's own body (Grogan, 1999). Most of the research on body image has focussed, as the present study, on (dis-) satisfaction with weight and the desire to be thin (Grogan, 1999). A distorted body image is defined as an bmi below the healthy level but above the individual target weight b_T, i.e. by the feature that an undernourished individual considers himself or herself as "too fat".

Since the size of ω determines, ceteris paribus, the individual effort in weight reduction through restrained eating and physical exercise, it may be interpreted as will power or self-control. This conceptualization of self-control, however, deviates from the one in Strulik (2019). There, self-control was conceptualized as impulse control of a dual-self personality, based on Fudenberg and Levine (2006). Here, because of the different focus of analysis, we ignore aspects of affective behavior and non-anorexic individuals are considered to be fully rational. They take the consequences of health behavior on health and longevity fully into account and, if they are overweight or obese, their body weight is simply explained by a relatively high preference for food consumption and/or a relatively strong dislike of exercise in combination with a relatively low regard of body image.

For non-anorexic individuals, target bmi is treated as a given parameter. Anorexic individuals, in contrast, continuously adjust their target bmi downwards. As in the economics of addiction

this behavior is modeled as an adaptation to past behavior. Formally, it is captured by a gradual increase of the factor z by which target bmi falls short of healthy bmi. The level of z is determined by past experiences of weight control. A stock variable summarizing past consumption behavior has been conceptualized as "habit stock" in the economics of habit formation (e.g. Dynan, 2000; Caroll et al., 2000) and as "addictive capital" in the economic of addiction (e.g. Becker et al., 1991; Strulik, 2018). In the present context, z could be labeled as "anorexic capital". Specifically, we assume:

$$\dot{z} = \max\left\{0, \ \theta\left(z^{\kappa}\frac{b_H}{z} - b\right)\right\}, \tag{6}$$

with $\theta \geq 0$ and $0 \leq \kappa < 1$. Anorexia is characterized by $\dot{z} > 0$. We define $\theta > 0$ as (genetic) susceptibility to anorexia. For most individuals $\theta = 0$, and anorexia is no issue. For individuals susceptible to anorexia, $\theta > 0$, and anorexia can be triggered by sufficiently large successes in weight control. For $\kappa = 0$, anorexia is triggered in susceptible individuals when actual weight b is reduced below target weight, i.e. for $b < b_T = b_H/z$. As a response, $\dot{z} > 0$, implying that target weight b_H/z declines. For $\kappa > 0$, it requires a smaller weight loss to trigger addiction but then the disease takes a more severe turn. To see this, note that $\partial \dot{z}/\partial z = (\kappa - 1)b_H z^{\kappa-2} < 0$. The feature that $\partial \dot{z}/\partial z < 0$ is conducive to stability, i.e. for the convergence of z towards an upper limit. The speed of convergence is slower and the upper limit is higher for larger κ. For given κ, the speed at which addiction develops is determined by θ. The interaction between parameters θ and κ thus determines the severity and speed at which the disease develops.

A characteristic feature of anorexia is that individuals derive no pleasure from being thin. The initially experienced utility gain from weight loss that triggered the disease vanishes and turns into a utility loss. To see this feature in the model, notice from (6) that target weight stops declining when $b \geq z^{\kappa-1}b_H \geq b_H/z = b_T$ such that at best the individual achieves target weight. A sufficient (not necessary) condition for $b > b_T$ at the steady state is $\kappa > 0$. It implies that in the progression of the disease a point is reached where the individual does no longer manage to achieve target weight and thus experiences disutility from body image. In simple words, individuals who are unhealthily thin perceive themselves as being "too fat". They have developed a distorted body image.[1]

---

[1] The reason why $\kappa > 0$ is not necessary to generate disutility from body image is that the model allows overshooting of z such that $b > b_H/z$ at the steady state even for $\kappa = 0$ (see Section 4.2.).

Whereas an "ordinary addiction" is promoted by consumption of addictive goods, in the present context, *not* consuming food goods and thus being thin promotes the development of anorexia. Formally, this feature is expressed by $\partial \dot{z}/\partial b < 0$. As in ordinary addictions, there are tolerance, withdrawal, and reinforcement effects. Tolerance is expressed by the feature that utility (5) declines in the level of addictive (anorexic) capital z. The withdrawal effect is reflected by the feature that the utility loss experienced from returning to healthy weight b_H is the larger the lower the individual target weight $b_T = b_H/z$, i.e. the further an addiction has advanced. Reenforcement is reflected the negative cross derivative $\partial I^2/\partial z \partial b = -\omega$, implying that the utility gain from weight loss (lower b) is larger when z is high (at an advanced stage of the addiction). Notice that the strength of the reenforcement effect is increasing ω, i.e. in the importance of body image.

Individuals maximize expected lifetime utility $V = \int_0^T e^{-\rho t} S(D) \left\{ \tilde{U}(c, u, x) - \omega \left(\frac{b - b_T}{b_T} \right) \right\} \mathrm{d}t$ subject to (1)–(4). Individuals fail to take into account the law of motion (6). This behavior has been introduced in Strulik (2018) as imperfectly controlled addiction and contrasted with Becker and Murphy's (1988) theory of rational addiction. The theory of imperfectly controlled addiction is a minimal-invasive deviation from rationality that allows us to conceptualize addiction as a disease, i.e. as welfare reducing behavior that is not under individual control. According to Becker and Murphy (1988) and the economic literature of rational addiction, individuals develop addictive behavior only if addiction enhances lifetime utility, which makes it impossible to discuss addiction as a disease. The theory of imperfectly controlled addiction is minimal-invasive because it allows individuals to behave fully rational with respect to all other decisions aside from the one on body weight. More severe deviations from rationality, such as time-inconsistency (e.g. due to hyperbolic discounting) could also explain welfare reducing addiction but they would imply suboptimal decision making in all other domains of human life as well. Individuals subject to imperfectly controlled addiction correctly perceive the health consequences of their behavior and correctly take them into account in their fully rational decisions in all other domains of their life. The only imperfection is that they fail to control their weight loss addiction. In contrast to the theory of rational addiction, imperfectly controlled addiction fulfils the DSM-5 diagnostic criterion of addiction requiring that addicted individuals experience impaired control over the use of a substance (APA, 2013) with the distinction that, in the case of weight loss, the addiction concerns *not* using goods, namely not consuming food in sufficient amounts.

The current value Hamiltonian associated with problem (1)–(4) is given by

$$\mathcal{H} = S(D) \left[\tilde{U}(c, u, x) - \omega \left(\frac{zb}{b_H} - 1 \right) \right]$$

$$+ \lambda_k \left[w + rk - c - qu - ph \right] + \lambda_D \mu \left[D - Ah^\gamma + \frac{B}{2} (\nu u - \epsilon x - b_H)^2 - a \right], \quad (7)$$

with $\tilde{U}(c, u, x)$ given by (1). Individuals maximizes (7) by controlling consumption of food and non-food, health investments, and physical exercise. The optimal solution fulfils the first order conditions (8)–(11) and the costate equations (12)–(13):

$$S(D)c^{-\sigma} = \lambda_k, \quad (8)$$

$$S(D)(\alpha - \beta u) = \frac{\nu \omega z}{b_T} + \lambda_k q - \lambda_D \mu B \nu (\nu u - \epsilon x - b_H) \quad (9)$$

$$-S(D)\lambda_D \mu A \gamma h^{\gamma - 1} = \lambda_k p, \quad (10)$$

$$S(D)\frac{\epsilon \omega z}{b_T} - \lambda_D \mu \epsilon B(\nu u - \epsilon x - b_H) = S(D)\delta \left(\frac{D}{D_0} \right)^\eta x \quad (11)$$

$$\lambda_k r = \lambda_\rho - \dot{\lambda}_k \quad (12)$$

$$S'(D)\tilde{U}(\cdot) - S(D)\frac{\delta}{2} \left(\frac{D}{D_0} \right)^\eta \frac{\eta}{D} x^2 + \lambda_D \mu = \lambda_D \rho - \dot{\lambda}_D. \quad (13)$$

Condition (8) requires that the marginal utility from non-food consumption is equal to the marginal cost, which is given by a unit less of savings evaluated by the shadow price of capital λ_k. Condition (9) requires that the marginal utility of food consumption is equal to the marginal cost. Additional to the marginal cost from saving q units of income less, the marginal cost consists of the marginal disutility from weight gain plus the health cost of the induced change in body weight. To see the latter, notice that the shadow price of health deficits λ_D is negative since health deficits increase mortality and reduce lifetime utility. If the individual is overweight, actual bmi exceeds the healthy bmi, $b = \nu u - \epsilon x > b_H$, and an additional unit of food consumption leads to health deficits $\mu B \nu (\nu u - \epsilon x - b_H)$ which the individual evaluates at shadow price λ_D. Condition (10) requires that marginal utility of health expenditure equals marginal costs. The marginal utility of health expenditure consists of the reduction in health deficits $-\mu A \gamma h^{\gamma - 1}$, evaluated at the (negative) shadow price of health deficits, the marginal cost consists of p units less savings evaluated at λ_k. Condition (11) requires that the marginal utility from a unit of exercise equals the marginal cost. The marginal utility consists of the direct utility gain from

weight loss and the impact that the reduced weight has on health deficits, evaluated at the shadow price for health deficits. The marginal cost consists of the disutility of exercise.

A part of the model is accessible via an explicit analytical solution. From (8)–(11) we obtain the interior solution for food consumption and exercise as:

$$u = \frac{a_1 a_2 + a_3 \left[a_1 \epsilon^2 + a_2 \nu b_H\right] - a_2 \nu \omega z / b_T}{\beta a_2 + a_3 \left[\beta \epsilon^2 + a_2 \nu^2\right]}, \tag{14}$$

$$x = \frac{\epsilon \left\{a_3 \left[a_1 \nu - \beta b_H\right] + \beta \omega z / b_T\right\}}{\beta a_2 + a_3 \left[\beta \epsilon^2 + a_2 \nu^2\right]} \tag{15}$$

$$\text{with } a_1 \equiv \alpha - qc^{-\sigma}, \quad a_2 \equiv \delta \left(\frac{D}{D_0}\right)^{\eta} > 0, \quad a_3 \equiv \frac{pc^{-\sigma} B}{A \gamma h^{\gamma-1}} > 0.$$

The compound variable a_2 measures the marginal disutility of a unit of exercise. The compound variable a_3 is the health effect of a unit of weight gain on lifetime utility measured in terms of utility from non-food consumption. In principle, the optimal solution could be at a corner (no food consumption and/or no exercise). In order to avoid uninteresting case differentiation, we assume that parameters and initial conditions are such that the solution is interior.

PROPOSITION 1. *For given state of health and given expenditure on health care and non-food consumption, individuals for whom body image is important (large ω) or who aspire a low target weight (large z) eat less and exercise more.*

The proof follows from inspection of (14) and (15). The interesting observation here is that an increase in ω and an increase in z are observationally equivalent in their impact on food consumption and exercise. Anorexic behavior can be thus perceived as continuous decline in target bmi, or as continuous increase in the importance of body image for utility, or both. The result also implies that the observation of low food consumption and a high levels of exercise (and thus low body weight) is insufficient to diagnose anorexia for the model-individual. We may equivalently observe a healthy individual with very low ideal weight and/or great importance of body image for utility (for example, an ascetic monk). Distinctive features of anorexia are (i) a continuous update of target weight and, in the advanced state of the disease (ii), a dissatisfaction with weight loss achievements, i.e. the experiences of being too fat while actually being emaciated (distorted body image).

Inspection of (14) and (15) lets us also conclude that individuals with a high preference for food consumption or individuals facing a low relative price of food (large a_1) eat more and

exercise more while individuals in bad health (large a_2) eat less and exercise less. Aside from these intuitive results of comparative statics, the model cannot be assessed analytically. In order to facilitate a numerical solution we first eliminate the costate variables and obtain from (8) to (15) the dynamic solutions for non-food consumption and health expenditure:

$$\frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \tag{16}$$

$$\frac{\dot{h}}{h} = \frac{1}{1 - \gamma} \left[r + m - \mu - S'(D)\tilde{U}(\cdot) + S(D)\frac{\delta}{2} \left(\frac{D}{D_0} \right)^\eta \frac{\eta}{D} x^2 \right] \tag{17}$$

with mortality rate $m \equiv -\dot{S}/S$. Equation (16) is the familiar Ramsey rule or Euler equation for consumption. Equation (17) is the dynamic condition for optimal health expenditure. Without uncertainty (for $\dot{S} = S' = 0$) and without consideration of physical exercise (for $x = 0$) it collapses to the Health-Euler-equation in Dalgaard and Strulik (2014).

The life cycle problem is fully described by the solution of (1)–(6) and (14)–(17) that fulfils the initial conditions $k(0) = 0$, $D(0) = D_0$, $z(0) = z_0$ the terminal time conditions $k(T) = 0$, $D(T) = \bar{D}$, and the transversality condition $\mathcal{H}(T) = 0$.

3. Calibration

As a benchmark, the model is calibrated to approximate a single, white, 20-year-old U.S. American male in the year 2010, such that results will be comparable to those of earlier applications of the health deficit model (Dalgaard and Strulik, 2014; Strulik, 2018, 2019). Since anorexia is more prevalent in women and frequently starts already in adolescence, we will later also consider a calibration approximating a 16-year-old U.S. American women. The benchmark American does not suffer from anorexia but from overweight. His bmi is 28.7, according to the average male bmi in 2010 (Abarca-Gomez et al., 2017). The benchmark individual is assumed to be immune to anorexia by setting $\theta = 0$.

A parsimonious representation of the survival function is given by the concave function:

$$S(D) = \Psi - \frac{\phi}{1 - \chi D}. \tag{18}$$

In order to determine the parameters, I feed in the estimates from Mitnitski et al. (2002) on the association between age τ and health deficits D and adjust the parameters of (18) such that the predicted association of age and survival $S(\tau) = S(\tau^{-1}(D))$ approximates the actual $S(\tau)$ estimated from U.S. life tables (taken from Strulik and Vollmer, 2013). This leads to the

estimates $\psi = 1.93$, $\phi = 0.85$, and $\xi = 3.18$. Figure A.1 in the Appendix shows the implied $S(D)$ function and the match of the predicted $S(t)$ curve and the data. From Mitnitski et al. (2002), I set the force of aging μ to 0.043 and infer initial health deficits at age 20 as $D(0) = 0.0273$.

When the individual is below age 65, 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 set r to 0.07 according to the long-run real interest rate on equity and real estate (Jorda et al., 2019). I normalize $p = q = 1$ and $\underline{D} = 0.0273$. I set $\bar{D} = 0.18$, which will imply a maximum age at death of 95 for the benchmark individual. It should be noted, however, that individuals who live a healthier life can face a much higher maximum age due to their slower accumulation of health deficits. Finally, I set the healthy bmi b_H to 24 and the target bmi b_T to 19, which provides $z(0) = 1.26$. The assumed value of b_H is needed to be from the upper range of the values considered as healthy (CDC, 2021) because the quadratic damage function requires the distance between healthy weight and severe underweight to be large in order to calibrate plausible effects of low body weight on mortality. The value of b_T is somewhat arbitrary but its value is also not decisive for any of the results. It seems to be reasonable (but not essential) to assume that the aspired weight lies below the healthy weight due to the influence of role models of thin actors and celebrities.

The remaining parameters are calibrated jointly to match the following stylized facts. 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). 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).[2] Physical exercise is measured in metabolic equivalents (MET), defined as the energy cost of a given physical activity divided by energy expenditure at rest. This metric allows for the aggregation of different kinds of physical exercise 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)

---

[2]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.

on physical exercise, an equivalent of about 23 minutes of brisk walking per day (Moore et al., 2003). We thus calibrate an average life cycle level of x of 1.14. 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. The weight loss in middle aged overweight men due to low-level exercise has been estimated to be on average 1.3 kg (Slentz et al., 2004). Given an average height of men born 1990 of 1.77 meters, low level exercise reduces bmi by 0.74, which is the value targeted by the calibration. 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 effects of declining food consumption and declining exercise 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 leads to the following parameter estimates: $a = 0.0127$, $A = 0.0016$, $B = 5 \cdot 10^{-4}$, $\alpha = 0.0016$, $\beta = 3.7 \cdot 10^{-7}$, $\gamma = 0.26$, $\delta = 0.005$, $\epsilon = 0.39$, $\eta = 0.75$, $\nu = 0.008$, $\rho = 0.05$, $\sigma = 1.20$, $\omega = 0.20$. The predicted life cycle trajectories for health deficits, survival probability, exercise, food expenditure share, bmi, and health spending are shown by blue (solid) lines in Figure 1. Dots indicate calibrated data points. As a first exercise and plausibility check, we consider an individual who puts more importance on body image and set $\omega = 1.7$. This value is chosen such that the individual achieves about target weight (bmi 19). All other parameters and initial values are kept from the benchmark American. Predicted life cycle trajectories are shown by green (dash-dotted) lines in Figure 1. The greater importance of body image leads to more healthy behavior, the mean exercise level increases to about 3.6, a more than threefold increase of benchmark exercise, and food consumption declines pronouncedly. The income saved from reduced food expenditure is partly spent on health care such that the health expenditure trajectory lies slightly above the one of the benchmark individual. With increasing age, exercising becomes harder and body size increases somewhat but remains close to target weight throughout life. As a result, survival prospects improve, and life expectancy at age 20 rises to 79.6. The predicted difference in life expectancy compared to benchmark is 2.5 years, a value that is close to the empirical estimates of years lost of life expectancy at age 40 due to

FIGURE 1: BENCHMARK CALIBRATION AND BENCHMARK ANOREXIA
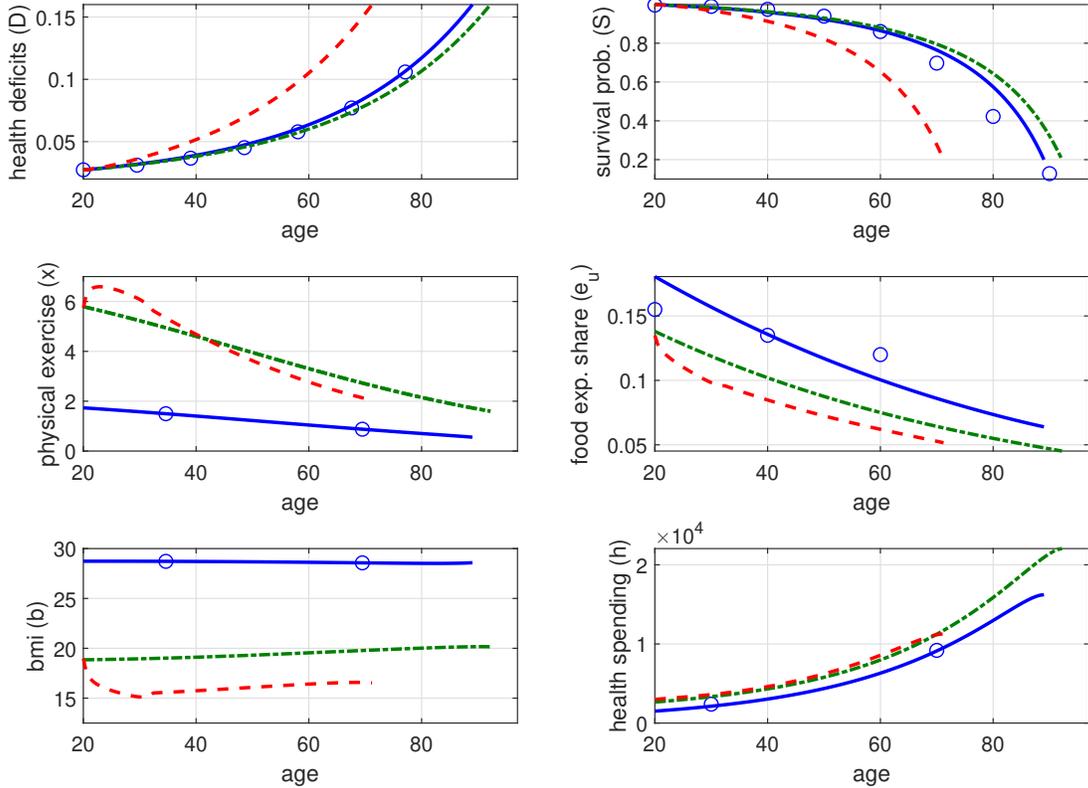
Blue (solid) lines: calibrated benchmark American. Dots indicate calibrated data points, see text for details. Green (dash-dotted lines): individual with higher self-control ($\omega = 1.7$). Red (dashed) lines: anorexic individual: high self-control and addiction to weight control ($\theta = 0.6$, $\kappa = 0.18$).

overweight (defined as $25 <$ bmi < 30), see Peters et al. (2003) and Grover et al. (2005). As another plausibility check, I compute the value of life of the benchmark individual as $ 10.6 million.[3] This prediction agrees reasonably well with empirical estimates of the value of life, ranging from $ 7 million (Murphy and Topel, 2006, Fig. 3) to $ 11.6 million (U.S. Department of Transportation 2021).

4. RESULTS

4.1. **A benchmark Case of Anorexia.** In order to set up a case of severe anorexia, I consider the benchmark individual with strong importance of body image ($\omega = 1.7$) and adjust θ and κ to match a life expectancy at 20 of 36 years (Harbottle et al., 2008; Meczekalski et al., 2013) and a lifetime minimum bmi of 15. The latter value is chosen because the starvation threshold

---

[3]The value of life converts expected lifetime utility measured in "utils" into monetary equivalents and is given by $VoL = \int_0^T e^{-\rho\tau} S[D(\tau)] U[c(\tau), u(\tau), x(\tau), b(\tau)] d\tau / U_c[c(0)]$ where U_c denotes the marginal utility of consumption.

is estimated to be around a bmi of 14 for men (Henry, 2001). We thus assume that death from starvation is avoided and that the low life expectancy is caused by the fast accumulation of health deficits in undernourished bodies. This provides the estimate $\theta = 0.6$ and $\kappa = 0.18$. All other parameters are taken from the benchmark calibration. The anorexic individual thus differs from the individual with healthy weight (green lines in Figure 1) only by being susceptible to weight loss addiction.

The predicted lifetime trajectories are shown by red (dashed) lines in Figure 1. The positive utility experienced from the initial success in weight loss ($b < b_H/z^{1-\kappa}$) triggers anorexia ($\dot{z} > 0$) and the individual further reduces his target bmi. In order to achieve the more ambitious weight goals, he eats less and exercises more than needed to sustain a healthy body weight. In a vicious cycle of adaptation, downward adjustment of ideal body weight, and less food consumption, bmi reaches as trough at a level of about 15. The level of z reaches a steady state when the body is too weak to push weight loss ambitions further downwards, i.e. formally when $b \geq b_H z^{\kappa-1}$. After target bmi stabilizes at a bmi level of about 14, the individual slowly regains weight because working off calories with exercise becomes harder due to the accumulated health deficits. Eventually, exercise falls below the level maintained by the healthy individual. Due to the low level of food consumption, however, bmi stays below 17 throughout life. As a result of the undernourished and weak body, health deficits accumulate quickly and survival probability decreases drastically (upper left and right panel in Figure 1), an outcome that cannot be prevented by health expenditure, which is above the calibrated benchmark level (lower right panel).

FIGURE 2: ASPIRED BMI AND UTILITY FROM BODY IMAGE
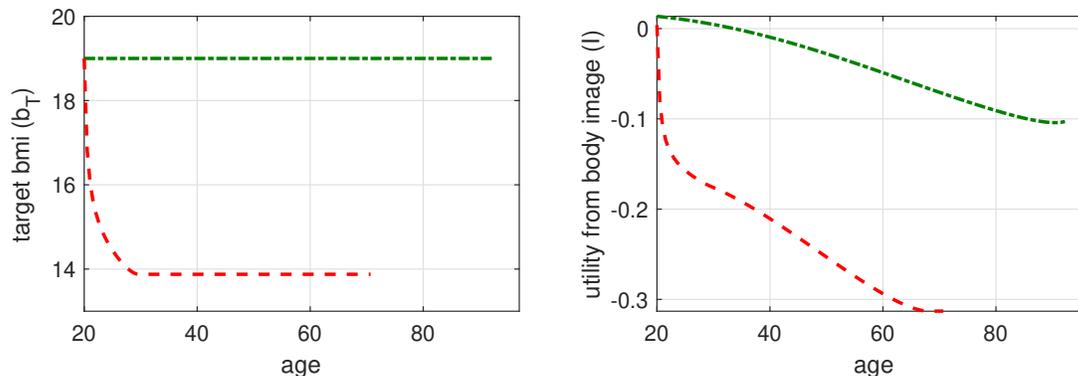
Green (dash-dotted lines): individual with higher self-control ($\omega = 1.7$). Red (dashed) lines: anorexic individual: high self-control and addiction to weight control ($\theta = 0.6$, $\kappa = 0.18$). Parameters as for Figure 1.

As discussed above, a key feature of the disease is that the anorexic individual fails to enjoy his low body weight. Even without the detrimental side effects on health and longevity and ignoring the forgone utility from food consumption, the individual would be better off without the disease. This feature is illustrated in Figure 2, which shows additional life cycle outcomes for the individual with healthy body weight (green dash-dotted lines) and the anorexic individual (red dashed lines). All parameters are as for Figure 1. The healthy individual maintains a target bmi of 19 throughout life (panel on the left hand side). Until his mid-thirties, he experiences positive utility from body image by (mildly) surpassing target weight (panel on the right-hand side). Later in life, utility from body image turns negative due to the (mild) weight gain and the failure to maintain the target weight. The anorexic individual, in contrast, enjoys utility from body image only in the initial period. The success in weight loss triggers a continuous downward adjustment of target bmi, which is, however, never reached again such that the utility from body image declines. Due to the distorted body image, the individual perceives himself as "too fat" notwithstanding the continuously deteriorating bmi. However, regaining weight and returning to the initial situation, would cause an even greater disutility from body image and is thus not considered as a solution by the anorexic individual. This lock-in feature resembles the withdrawal effect in ordinary addictions.

4.2. **Sensitivity Analysis: Different Turns of the Disease.** In this and the next subsection we consider by way of example the sensitivity of behavior and outcomes to alternative parameter values. The parameter κ controls susceptibility to disease and the self-productive nature of weight loss addiction. A lower value of κ implies that a larger initial success in weight loss is needed to trigger the disease and that the weight loss goals are less ambitious. This claim is verified for the case of $\kappa = 0.16$ (instead of 0.18). The predicted life cycle trajectories are shown by green (dashed-dotted) lines in Figure 3. Similarly, the disease takes a weaker course for lower levels of θ (not shown).

These results of comparative statics hold, of course, only ceteris paribus and the disease can take a more severe turn even for lower levels of κ when body image becomes more important. This feature is verified for the case $\omega = 1.9$ (instead of 1.7) and $\kappa = 0$ (instead of 0.16) The predicted life cycle trajectories are shown by red (dashed) lines in Figure 3. The high importance of body image (i.e. the high level of self-control in weight management) enables the individual to relentlessly pursue his weight loss goals and to adjust the target bmi further downward to a level

around 13. The predicted life cycle behavior and health outcomes resemble those of benchmark anorexia in Figure 1 and life expectancy at 20 is obtained as 33 years. These predictions are in line with studies finding that a high level of self-control conducive to the development of anorexia (see Introduction).

FIGURE 3: SENSITIVITY ANALYSIS: DIFFERENT TURNS OF THE DISEASE
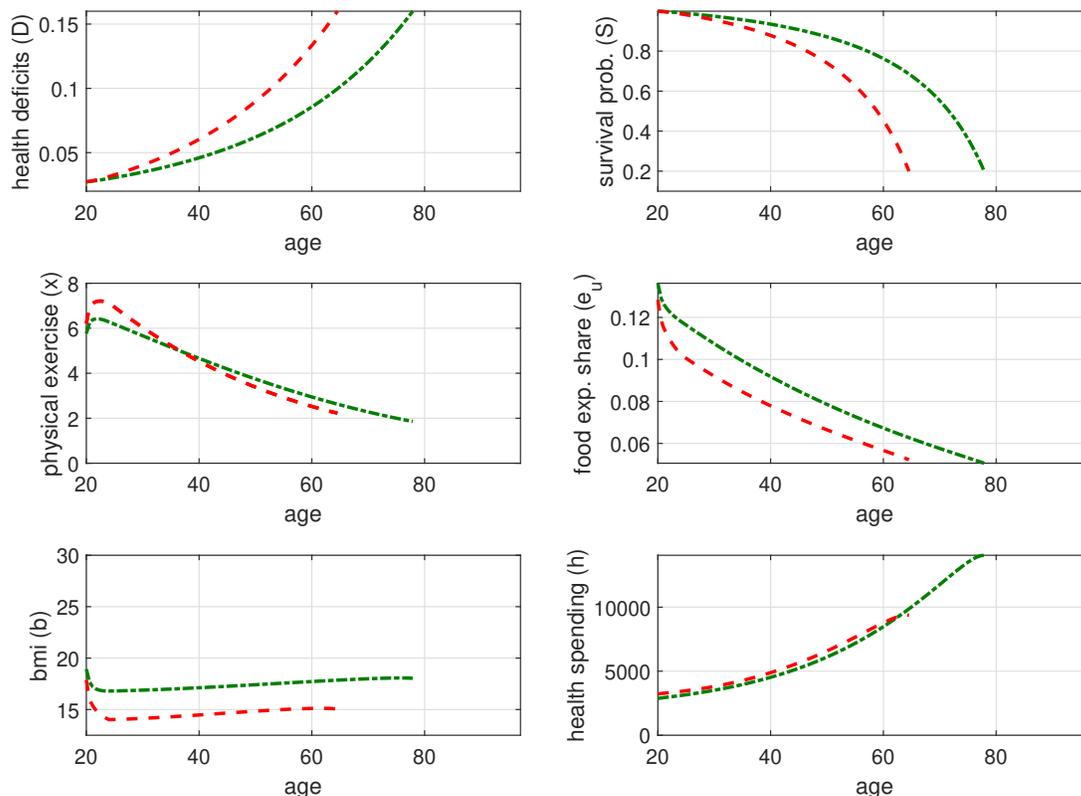
Green (dash-dotted lines): $\kappa = 0.16$: Red (dashed) lines: $\kappa = 0$, $\omega = 1.9$. All other parameters as for benchmark.

4.3. **Anorexia Athletica.** Anorexia athletica is a variant of anorexia, which is particularly prevalent in athletes and characterized by excessive exercise in addition to restrained eating. Its implementation requires a re-interpretation of utility from body imagine, which is now to a lesser degree driven by social or cultural norms of thinness but by the requirements and prospective gains from being thin in terms of success in sports. Naturally, the phenomenon is prevalent mostly in sports where low weight provides an advantage such as ski jumping, road cycling, figure skating, ballet, or horse riding (Sudi et al., 2004; Sundgot-Borgen and Torstveit, 2004).

In order to capture the phenomenon by the model, we set $\omega = 1.5$ and $z(0) = 1.3$ (implying an initial target bmi of 18.5) and consider two values for the disutility from exercise: 0.0048

FIGURE 4: SENSITIVITY ANALYSIS: ANOREXIA ATHLETICA
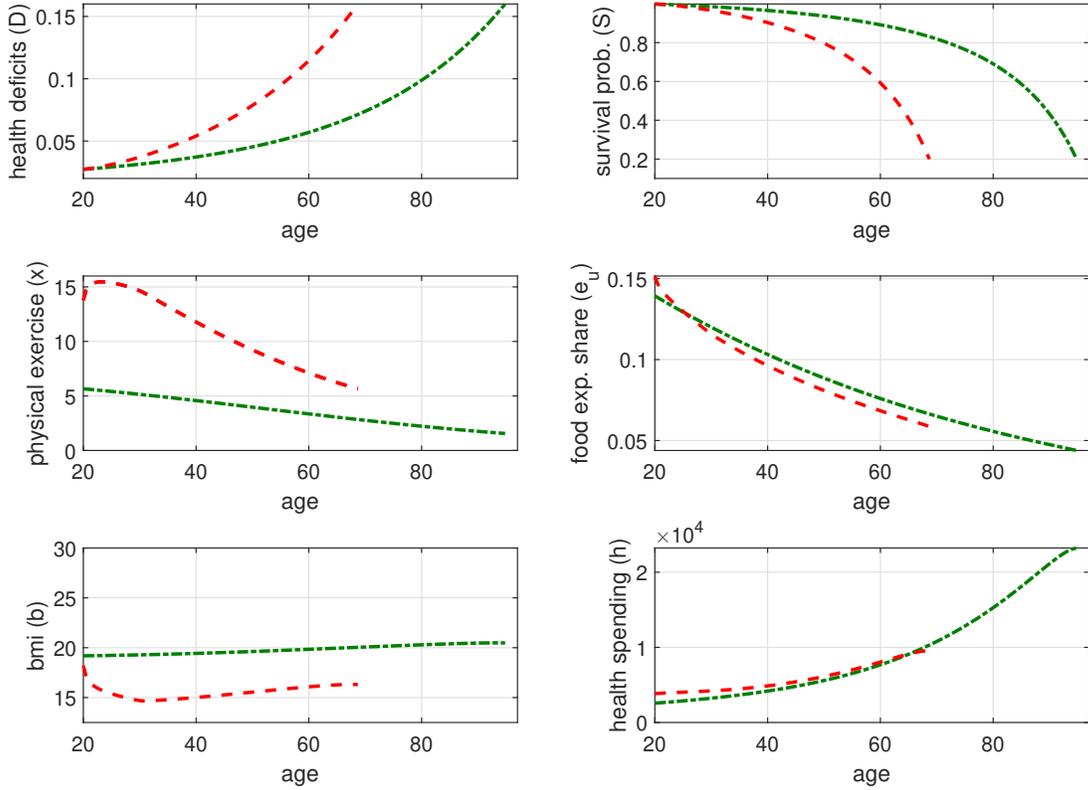
Green (dash-dotted lines): $\delta = 0.0048$ (as in benchmark): Red (dashed) lines: $\delta = 0.0016$. Both lines: $\omega = 1.5$, $z(0) = 1.3$. All other parameters as for benchmark.

(benchmark) and 0.0016 (a threefold lower value). All other parameters values are as for the benchmark calibration, except that both individuals face the benchmark addiction parameters ($\theta = 0.6$, $\kappa = 0.18$). The calibration is specified such that preferences for exercise are conducive to the development of the disease. Although both individuals are susceptible to anorexia, the disease does not develop in the individual with the larger disutility from exercise. As shown by green (dashed-dotted) lines in Figure 4, this individual is predicted to maintain a healthy bmi around 19 and life cycle behavior and outcomes resembles those of the healthy individual of the benchmark run (green lines in Figure 1). Although the individual faces an ambitious weight goal, he is protected from the disease by his distaste for exercise. The individual with the low disutility from exercise is represented by red (dashed) lines in Figure 4. The high level of physical exercise (center-left panel) leads to greater initial success in weight loss and triggers the disease: the individual responds with downward adjustment of target bmi, even more restraint in eating behavior, and, at least in the early phase, with even more exercise. In the course of the disease, bmi reaches a trough at about 15 and the life cycle trajectories resemble those of

19

conventional anorexia in Figure 1 with difference that now the disease is driven to a greater extent by excessive exercise. The predicted life expectancy at 20 is 38 years.

Exercise attains a maximum of 16 MET hours per day, i.e. 116 MET hours per week, a level that can be achieved by about 20 hours of vigourous exercise per week. This is a very high but not implausibly high exercise effort. It is commonly seen in people training for competition in marathons or triathlons. There is a debate in the literature whether excessive exercise causes cardiac problems and faster aging of the heart (see e.g. O'Keefe et al., 2016; Abdullah et al., 2016), a phenomenon, which would not be captured by the model. Excessive exercise, however, is certainly health-damaging when it occurs in conjunction with insufficient food intake. The anorexic individual is predicted to eat about as much as the healthy individual (center right panel in Figure 4) although he spends up to three times more MET hours on exercise (center left panel). As the individual ages and accumulates health deficits, MET hours declines due to the increasing pain from exercise. However, even close to death, the exercise level exceeds the level exerted by the healthy individual.

The model can be applied to the example of Bahne Rabe from the Introduction. All of the members of the Olympic eight rowing team likely shared a trait of high self-control and a penchant for exercise (low δ), features that enabled them to train very hard and win the gold medal. But only Bahne developed anorexia. The explanation offered by the model is that only Bahne had a genetic disposition for the disease ($\theta > 0$). Bahne's brother and sister also suffered from anorexia in adolescence (Kurbjuweit, 2001). But only Bahne developed a severe, persistent, and eventually fatal form of the disease. The explanation offered by the model is that the Rabe family shared a genetic disposition for the disease, which, however, only occurred in Bahne in combination with high level of self-control and a penchant for exercise, which enabled the severe development of the disease.

Another interesting observation is that one cannot be a top athlete without being healthy. In the model, a low level of initial health deficits reduces the disutility from exercise. Ceteris paribus, being healthy induces not only a high level of exercise, it is also conducive to develop the anorexia athletica (given genetic susceptibility). In other words, for given preferences, being unhealthy is protective against the diseases. We would thus expect anorexia to be more prevalent among young and healthy people than among the unhealthy.

4.4. **Anorexia in Women.** We next consider a recalibration for women. This exercise adds more relevance since anorexia is more widespread among women. Moreover, a comparison with the calibration for men will provide an explanation for the greater prevalence of the disease among women. In order to capture an onset of the disease in adolescence, I set the initial age to 16. As for men, I take the data for women and feed into the survival function (18) the estimates from Mitnitski et al. (2002) on the association between age τ and health deficits D and adjust the parameter values such that the predicted association of age and survival $S(\tau) = S(\tau^{-1}(D))$ approximates the actual $S(\tau)$ estimated from U.S. life tables. This leads to the estimates $\psi = 1.40$, $\phi = 0.35$, and $\xi = 3.60$. Red lines in Figure A.1 in the Appendix show the implied $S(D)$ function and the match of the predicted $S(\tau)$ curve and the data. From Mitnitski et al. (2002), I set the force of aging μ to 0.031 and infer initial health deficits at age 16 as $D(0) = 0.0360$. We thus observe the compensation effect of morbidity, i.e. the feature that women are initially less healthy than men but age slower than men (see Gavrilov and Gavrilova, 1991; Mitnitski et al., 2002, Abeliansky and Strulik, 2018, for a discussion) and the morbidity mortality paradox, i.e. the feature that women are for given age less healthy than men but live longer (see Case and Paxson, 2005; Kulminsky et al., 2008; Schuenemann et al., 2017, for a discussion).

When the individual is below age 65, I set $w = 17,303$, which is the average labor income for single women in the year 2010 (BLS, 2012). I keep the interest rate, the time preference rate, and all prices from the benchmark calibration. I set the bmi to 28.6, according to the average female bmi in 2010 (Abarca-Gomez et al., 2017), a value that basically coincides with the benchmark value for men. I set the same healthy bmi (24) and target bmi (19) and thus the same $z(0)$, as for the benchmark model. I also keep the parameters on metabolism, exercise, and health from the benchmark model ($\delta = 0.048$; $\epsilon = 0.39$; $\eta = 0.75$; $\nu = 0.0078$; $a = 0.0127$, $\gamma = 0.26$, $A = 0.00159$, $B = 5 \cdot 10^{-4}$). Given that women display the same bmi as men but earn substantially less, the food expenditure share will be higher for women. According to the BLS (2021), total food expenditure of single women is 94 percent of that of single men while food at home expenditure of single women is 111 percent that of single men. Instead of targeting the food expenditure share, I thus require that the benchmark woman spends in terms of lifetime averages about as much on food as the benchmark man. I estimate the remaining parameters in order to match food expenditure, health expenditure, and a life expectancy at 20 of 61.7

(expected death at age 81.7), in accordance with actual female life expectancy at 20 in 2010 (NVSS, 2014).

This leads to the estimates $\sigma = 1.42$, $\alpha = 0.0015$, and $\omega = 0.30$. The result for σ indicates that the utility function for non-food consumption exhibits more curvature for women (as in Schuenemann et al., 2017). It implies that women face a higher degree of relative risk aversion, as found in experimental and non-experimental studies (Croson and Gneezy, 2009; Cohen and Einav, 2007; Mazzocco, 2008). The result for α implies that women experience slightly lower utility from eating than men and the result for ω implies that women put more emphasis on body image than men. The implied life cycle trajectories are shown by blue lines in Figure 5, data points are indicated by circles. The higher value of σ for women is needed to match the higher health expenditure for women compared to men of the same age. More curvature of the utility function, i.e. more steeply declining marginal consumption, implies that women put less emphasis on instantaneous consumption and more emphasis on a long life (a feature that is in detail explained in Dalgaard and Strulik, 2014). They thus spend more on health and live longer than men.

As for men, we next consider a women with healthy bmi. An average bmi of about 19 is elicited by setting $\omega = 1.35$ and the associated life cycle trajectories are shown by green (dash-dotted) lines in Figure 5. Remarkably, the healthy behavior is elicited for a much lower value of body image utility (self-control) for woman than for men (for men, $\omega = 1.70$ was needed to produce about the same behavior). The reason is that women experience less utility from consumption because (i) they earn less and spend less on consumption and (ii) utility from consumption exhibits more curvature (marginal utility is more steeply declining). This means that, for given size of ω, utility from body image plays a relatively larger role for total utility of women such that a smaller value of ω is needed to elicit strong responses of eating behavior and exercising.

We next consider women susceptible to anorexia ($\theta = 0.6$, as for men). In order to create a case of fast onset of the disease in adolescence and severe progression with a minimum bmi level of about 14, I set $\omega = 1.46$ and $\kappa = 0$. The resulting health behavior and health outcomes are shown by red (dashed) lines in Figure 5. The high importance of body image leads to large initial successes in weight reduction and triggers the disease. The predicted life cycle trajectories resemble those obtained for anorexic men (cf. Figure 1 and 3). The implied life expectancy at 16 is 46.
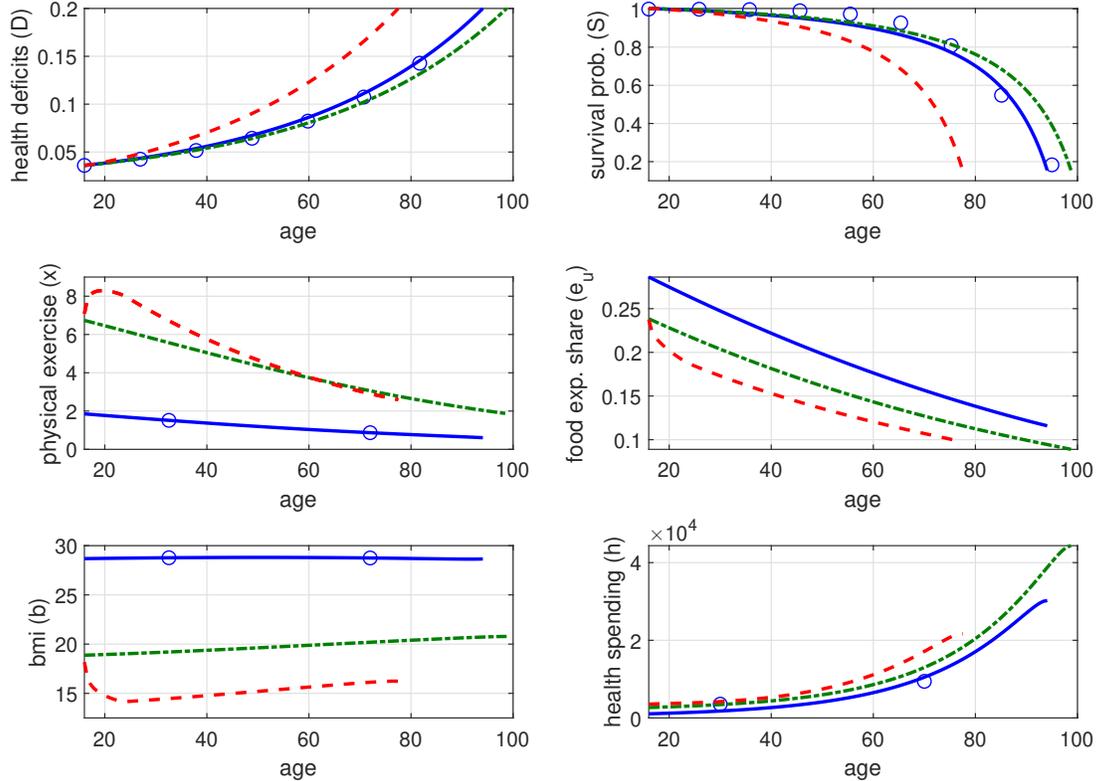
Blue (solid) lines and green (dash-dotted lines): as for Figure 1: Red (dashed) lines: anorexic individual: high self-control and addiction to weight control ($\theta = 0.6$, $\kappa = 0.18$).

By comparing men and women, we can derive an explanation for why anorexia is more widespread among women. To this end, we note that a level of $\omega = 1.46$ would not induce anorexia in the calibrated average American man. Similar behavior as for women required a value of $\omega = 1.7$ for men. Thus, if we assume that ω as well as (genetic) susceptibility to anorexia (captured by θ and κ) are equally distributed among men and women, there will be more women suffering from the disease. The reason is that women experience lower utility from consumption such that body image becomes relatively more important and a lower level of ω is sufficient to elicit the initial weight reduction needed to trigger the disease. This outcome would be further amplified if women put actually more emphasis on body image, expressed by, on average, higher values of ω than for men.[4]

---

[4]It can be shown that the lower income of women is not driving these results. If the calibrated woman is endowed with the male reference income, she will spend more health and therefore develop health deficits more slowly. The predicted eating and exercise behavior and thus predicted bmi, however, almost match the trajectories shown by red lines in Figure 5. The minimum bmi is 14.5 instead of 14.0, as in the benchmark run.

4.5. **Anorexia Therapies.** While there is still no established treatment or cure for anorexia in practise, it is interesting to use the model to investigate how stylized therapies work in theory. We first consider an addiction reset. In addictions to the consumption of goods (e.g. drugs, collecting, or gambling), the easiest albeit potentially painful reset of addiction can be generated by abstention from consumption. Here, the addiction is concerned about *not* consuming food and an analogous withdrawal effect is conceptually more difficult. Perhaps, a reset can be realized by removing the individual from an anorexic environment or by persuading the individual to abandon the very low target weight. Anyway, the point is, that the model-therapy does not work. To see this, we consider the calibrated anorexic women from above and assume a reset of z, i.e. a reset of target bmi from 14 to 19 at age 20. The resulting behavior and outcome is shown by blue (solid lines) in the panels on the left hand side of Figure 6. Red (dashed) lines re-iterate the anorexia trajectories from Figure 5. To save space, the figure focusses on exercise, nutrition, and bmi. The other trajectories look similar as in Figure 5. As response to the target weight reset, the individual eats more, exercises less, and gains weight. However, the fact that the actual bmi is still sufficiently below the target bmi, rekindles the disease and the therapy remains unsuccessful in the long-run.

Next, we consider a successful model-therapy, namely a reset of the importance of body image to benchmark value (from 1.46 to 0.3) at age 20. Here, too, it remains unclear how such a therapy could be realized in practice. Perhaps a new normal- or overweight friend at school could cause such a reset. Or taking up of a new hobby increases utility from consumption and/or the disutility from exercise, which would mean that body image becomes relatively less important. As shown by blue (solid) lines in the panels on the right-hand side of Figure 6, the therapy is successful in terms of long-run weight gains. The individual eats more and exercises less because being thin is less important. The individual, however, still suffers from failing to meet the low target bmi. This problem can be solved by a combined model therapy that resets ω and z at age 20. The resulting trajectories are shown by green (dash-dotted) lines in Figure 6. Withdrawal and resetting the target weight are now also successful in the long-term. Withdrawal amplifies the positive effects of reduced body image on food consumption and exercise and the individual regains a healthy bmi. In summary, it can be said that a therapy with the aim of reducing the importance of body image is successful in isolation, a therapy with the aim of restoring the target weight is only successful in conjunction with the body image therapy.

FIGURE 6: ANOREXIA THERAPIES
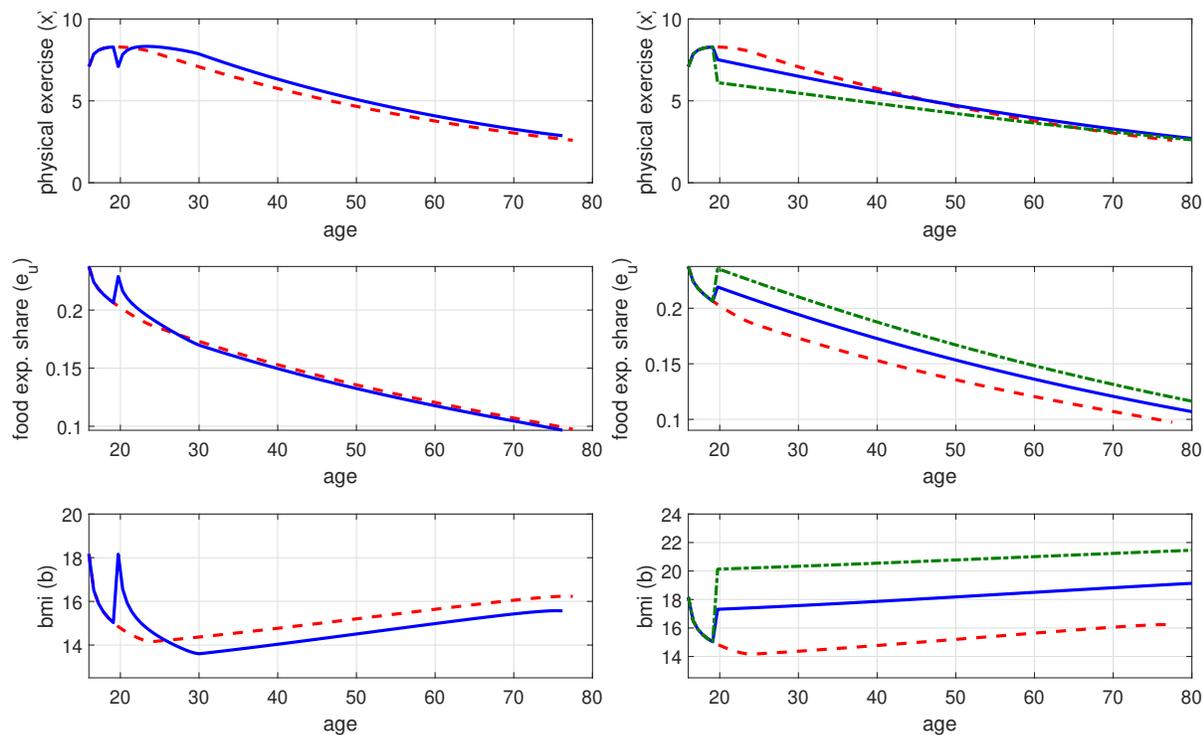
Red (dashed) lines: benchmark anorexia, as red (dashed) lines in Figure 5. Panels on the left-hand side: Blue (solid) lines: reset $z = 1.26$ ($b_T = 19$) at age 20. Panels on the right-hand side: Blue (solid) lines: $\omega = 0.2$ at age 20. Green (dash-dotted lines): $\omega = 0.3$ and reset $z = 1.26$ ($b_T = 19$) at age 20.

5. CONCLUDING REMARKS

In this paper, I proposed a health-economic theory of anorexia that combines eating and exercising behavior in a model of endogenous health and longevity with an economic theory of addiction. The theory emphasizes the importance of body image and self-control for the development of the disease, which is conceptualized as a habituation process that leads to a continuous downward adjustment of target weight. Although the model is stylized, it is sufficiently detailed to allow a calibration of life cycle choices and health outcomes of an average American man at age 20 and an average American adolescent woman. As any economic theory, the model is explorative in nature. It emphasizes particular mechanisms by suppressing other details. It has not the ambition to take into account all aspect of the complex disease of anorexia, which to this day is not fully understood. Recent advances in cognitive psychology and neurobiology suggest that habituation processes and self-control issues in combination with a distorted body image are at the core of the disease and are of first order importance for behavioral therapies while self-esteem, emotional problems, and family difficulties, are perhaps

less important than conventionally thought (Fairburn et al., 1998; Walsh, 2003). The proposed model supports this view, formalizes it in an economic life cycle theory, and discusses the long-term implications for health behavior and health outcomes. In contrast to other economic models of underweight and in contrast to the economic theory of rational addiction, anorexia, conceptualized as addiction to weight control is not the outcome of optimal life cycle planning. It is a disease with severe health implications and those suffering from it would benefit greatly from treatment in terms of health, longevity, and the value of life.

Figure A.1: Health-Dependent Survival and Survival by Age
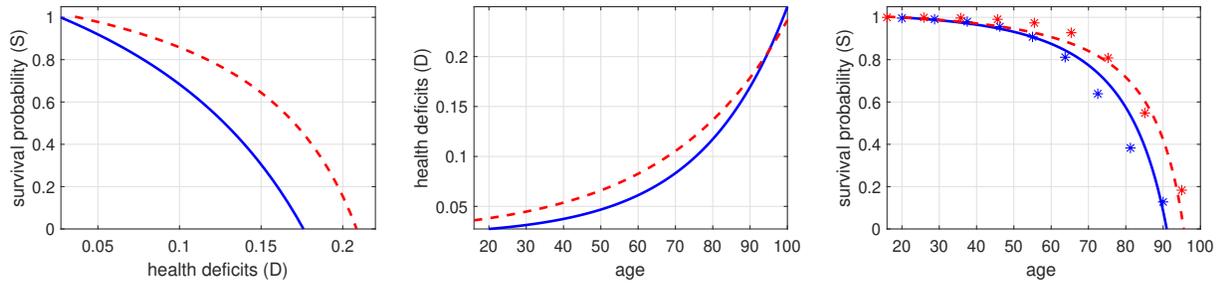
$S(t)$ is the unconditional probability of surviving until age t. Left: Assumed function $S(D)$. Middle: Estimated association $D(t)$ (Mitnitski et al., 2002). Right: Predicted (line) and estimated (stars) association between age and survival probability (estimates from Strulik and Vollmer, 2013). Blue (solid) lines: men; red (dashed) lines: women.

<center>REFERENCES</center>

Abarca-Gomez, L., Abdeen, Z. A., Hamid, Z. A., Abu-Rmeileh, N. M., Acosta-Cazares, B., Acuin, C., ... and Cho, Y. (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *The Lancet* 390(10113), 2627-2642.

Abdullah, S. M., Barkley, K. W., Bhella, P. S., Hastings, J. L., Matulevicius, S., Fujimoto, N., ... and Levine, B. D. (2016). Lifelong physical activity regardless of dose is not associated with myocardial fibrosis. *Circulation: Cardiovascular Imaging* 9(11), e005511.

Abeliansky, A. and Strulik, H. (2018). How we fall apart: Similarities of human aging in 10 European countries, *Demography* 55(1), 341-359.

APA (2013). Diagnostic and Statistical Manual of Mental Disorders, 5th edition. American Psychiatric Association Publishing, Washington D.C.

Arduini, T., Iorio, D., and Patacchini, E. (2019). Weight, reference points, and the onset of eating disorders. *Journal of Health Economics* 65, 170-188.

Arking, R. (2006). *The Biology of Aging: Observations and Principles*, Oxford University Press, Oxford.

Barbarich-Marsteller, N., W Foltin, R., and Timothy Walsh, B. (2011). Does anorexia nervosa resemble an addiction?. *Current Drug Abuse Reviews* 4(3), 197-200.

Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: a neurocognitive perspective. *Nature Neuroscience* 8(11), 1458-1463.

Becker, G. S., and Murphy, K. M. (1988). A theory of rational addiction. *Journal of Political Economy* 96(4), 675-700.

Becker, G. S., Grossman, M., and Murphy, K. M. (1991). Economics of Drugs: Rational Addiction and the Effect of Price on Consumption. *American Economic Review* 81(2), 237-241.

BLS (2012). Consumer Expenditure Survey. U.S. Bureau of Labor Statistics, September 2012 (`http://www.bls.gov/cex/csxcross.htm#y1011`).

BLS (2015). Consumer expenditures vary by age. Beyond the Numbers Vol. 4, 14. Bureau of Labor Statistics. `https://www.bls.gov/opub/btn/volume-4/consumer-expenditures-vary-by-age.htm?view_full`.

BLS (2021). Consumer units of single males (females) by income before taxes. Bureau of Labor Statistics. `https://www.bls.gov/cex/2019/crosstabs/singlesbyinc/malesinc.pdf`.

Bogg, T., and Roberts, B.W. (2004). Conscientiousness and health-related behaviors: a meta-analysis of the leading behavioral contributors to mortality. *Psychological Bulletin* 130(6), 887-919.

<center>28</center>

Bulik, C. M., Sullivan, P. F., Wade, T. D., and Kendler, K. S. (2000). Twin studies of eating disorders: a review. *International Journal of Eating Disorders* 27(1), 1-20.

Butler, G.K.L., and Montgomery, A.M.J. (2005). Subjective self-control and behavioural impulsivity coexist in anorexia nervosa. *Eating Behaviors* 6(3), 221-227.

Carroll, C. D., Overland, J., and Weil, D. N. (2000). Saving and growth with habit formation. *American Economic Review* 90(3), 341-355.

Case, A., and Paxson, C. (2005). Sex differences in morbidity and mortality. *Demography* 42(2), 189-214.

Casiero, D., and Frishman, W. H. (2006). Cardiovascular complications of eating disorders. *Cardiology in Review* 14(5), 227-231.

CDC (2013). Caloric Intake form Fast Food Among Adults: United States, 2007–20010. NCHS Data Brief 111. Centers for Disease Control and Prevention, February 2013.

CDC (2016). Anthropometric Reference Data for Children and Adults: United States, 2011–2014. National Center for Health Statistics. Vital Health Statistics 3(39).

CDC (2021). Healthy Weight–Assessing your Weight. `https://www.cdc.gov/healthyweight/assessing/index.html`

Ciarcia, C., Falsaperla, P., Giacobbe, A., and Mulone, G. (2015). A mathematical model of anorexia and bulimia. Mathematical Methods in the Applied Sciences, 38(14), 2937-2952.

Cobb-Clark, D.A., Kassenboehmer, S.C., and Schurer, S. (2014). Healthy habits: The connection between diet, exercise, and locus of control. *Journal of Economic Behavior & Organization* 98, 1-28.

Cohen, A., and Einav, L. (2007). Estimating risk preferences from deductible choice. *American Economic Review* 97(3), 745-788.

Compan, V., Walsh, B. T., Kaye, W., and Geliebter, A. (2015). How does the brain implement adaptive decision making to eat?. *Journal of Neuroscience* 35(41), 13868-13878.

Costa-Font, J., and Jofre-Bonet, M. (2013). Anorexia, body image and peer effects: evidence from a sample of European women. Economica, 80(317), 44-64.

Crescioni, A.W., Ehrlinger, J., Alquist, J.L., Conlon, K.E., Baumeister, R.F., Schatschneider, C., and Dutton, G. R. (2011). High trait self-control predicts positive health behaviors and success in weight loss. *Journal of Health Psychology* 16(5), 750-759.

Croson, R., and Gneezy, U. (2009). Gender differences in preferences. *Journal of Economic Literature* 47(2), 448-74.

Dalgaard, C-J., and Strulik, H. (2014). Optimal aging and death: Understanding the Preston Curve, *Journal of the European Economic Association* 12, 672-701.

Dalgaard, C. J., Hansen, C. W., and Strulik, H. (2021). Fetal origins – A life cycle model of health and aging from conception to death. *Health Economics* 30(6), 1276-1290.

Dragone, D., and Savorelli, L. (2011). Thinness and obesity: A model of food consumption, health concerns, and social pressure. *Journal of Health Economics* 31, 243-256.

Dynan, K. E. (2000). Habit formation in consumer preferences: Evidence from panel data. *American Economic Review* 90(3), 391-406.

Fairburn, C. G., Shafran, R., and Cooper, Z. (1999). A cognitive behavioural theory of anorexia nervosa. *Behaviour Research and Therapy* 37(1), 1-13.

Flegal, K.M., Kit, B. K., Orpana, H., and Graubard, B.I. (2013). Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *Jama*, 309(1), 71-82.

Fudenberg, D., and Levine, D. K. (2006). A dual-self model of impulse control, *American Economic Review* 96, 1449-1476.

Gavrilov, L.A. and Gavrilova, N.S. (1991). *The Biology of Human Life Span: A Quantitative Approach*, Harwood Academic Publishers, London.

GBD (2016). Global Burden of Disease Study 2016 (GBD 2016) Results. Global Burden of Disease Collaborative Network. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2017.

Godier, L. R., and Park, R. J. (2014). Compulsivity in anorexia nervosa: a transdiagnostic concept. *Frontiers in Psychology* 5, 778.

Godier, L. R., and Park, R. J. (2015). Does compulsive behavior in anorexia nervosa resemble an addiction? A qualitative investigation. *Frontiers in Psychology* 6, 1608.

Goldfarb, R. S., Leonard, T. C., Markowitz, S., and Suranovic, S. (2009). Can a rational choice framework make sense of anorexia nervosa?. National Bureau of Economic Research, Working Paper No. w14838.

Grogan, S. (1999). Body image: Understanding body dissatisfaction in men, women and children. Routledge, London.

Grover, S.A., Kaouache, M., Rempel, P., Joseph, L., Dawes, M., Lau, D. C., and Lowensteyn, I. (2015). Years of life lost and healthy life-years lost from diabetes and cardiovascular disease in overweight and obese people: A modelling study. *The Lancet Diabetes & Endocrinology* 3(2), 114-122.

Ham, J. C., Iorio, D., and Sovinsky, M. (2013). Caught in the bulimic trap? Persistence and state dependence of bulimia among young women. *Journal of Human Resources* 48(3), 736-767.

Harbottle, E. J., Birmingham, C. L., and Sayani, F. (2008). Anorexia nervosa: a survival analysis. *Eating and Weight Disorders* 13(2), e32-4.

Henry, C. J. K. (2001). The biology of human starvation: some new insights. *Nutrition Bulletin* 26(3), 205-211.

Jorda, O., Knoll, K., Kuvshinov, D., Schularick, M., and Taylor, A.M. (2019). The rate of return on everything, 1870–2015. *Quarterly Journal of Economics* 134(3), 1225-1298.

Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N., Wagner, A., and Bischoff-Grethe, A. (2013). Does a shared neurobiology for foods and drugs of abuse contribute to extremes of food ingestion in anorexia and bulimia nervosa?. *Biological Psychiatry* 73(9), 836-842.

Keeney, R. L. (2008). Personal decisions are the leading cause of death. *Operations Research* 56(6), 1335-1347.

Kulminski, A. M., Culminskaya, I. V., Ukraintseva, S. V., Arbeev, K. G., Land, K. C., and Yashin, A. I. (2008). Sex-specific health deterioration and mortality: The morbidity-mortality paradox over age and time. *Experimental gerontology* 43(12), 1052-1057.

Kurbjuweit, D. (2001). Locker, Bahne, locker. *Der Spiegel* 45. `https://www.spiegel.de/pan orama/locker-bahne-locker-a-6c573e8f-0002-0001-0000-000020521514`

Mazzocco, M. (2008). Individual rather than household Euler equations: Identification and estimation of individual preferences using household data. Discussion Paper, available at SSRN 695285 .

MEPS (2010). U.S. Department of Health & Human Services. Center for Financing, Access and Cost Trends, Agency for Healthcare Research and Quality: Medical Expenditure Panel Survey, 2010 (`https://meps.ahrq.gov/mepsweb/data_stats/quick_tables_results.j sp?component=1&subcomponent=0&year=-1&tableSeries=1&searchText=&searchMethod= 1&Action=Search`)

Meczekalski, B., Podfigurna-Stopa, A., and Katulski, K. (2013). Long-term consequences of anorexia nervosa. *Maturitas* 75(3), 215-220.

Miller, H.V., Barnes, J.C., and Beaver, K.M. (2011). Self-control and health outcomes in a nationally representative sample. *American Journal of Health Behavior* 35(1), 15-27.

Miller, W.C., Lindeman, A.K., Wallace, J., and Niederpruem, M. (1990). Diet composition, energy intake, and exercise in relation to body fat in men and women. *American Journal of Clinical Nutrition* 52(3), 426-430.

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002). The accumulation of deficits with age and possible invariants of aging. *Scientific World* 2, 1816-1822.

Moore, S.C., Patel, A.V., Matthews, C.E., de Gonzalez, A.B., Park, Y., Katki, H.A., ... and Thun, M. (2012). Leisure time physical activity of moderate to vigorous intensity and mortality: a large pooled cohort analysis. *PLoS Medicine* 9(11), e1001335.

Murphy, K.M. and Topel, R.H., 2006, The value of health and longevity, *Journal of Political Economy* 114, 871–904.

NCD Risk Factor Collaboration. (2016). Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. *The Lancet*, 387, 1377-1396.

Nestler, E. J. (2013). Treating the brain deep down: brain surgery for anorexia nervosa?. *Nature Medicine* 19(6), 678-679.

NVSS (2014). US department of health and human services, national vital statistics system. National Vital Statistics Report 63(7).

OECD (2016), Gross pension replacement rates. OECD Data (`https://data.oecd.org/pension/gross-pension-replacement-rates.htm`

O'Keefe, E. L., Torres-Acosta, N., O'Keefe, J. H., and Lavie, C. J. (2020). Training for longevity: the reverse J-curve for exercise. *Missouri Medicine* 117(4), 355.

Pauligk, S., Seidel, M., Fuertjes, S., King, J. A., Geisler, D., Hellerhoff, I., ... and Ehrlich, S. (2021). The costs of over-control in anorexia nervosa: evidence from fMRI and ecological momentary assessment. *Translational Psychiatry* 11(1), 1-12.

Peeters, A., Barendregt, J.J., Willekens, F., Mackenbach, J.P., Al Mamun, A., and Bonneux, L. (2003). Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Annals of Internal Medicine* 138(1), 24-32.

Pompili, M., Girardi, P., Tatarelli, G., Ruberto, A., and Tatarelli, R. (2006). Suicide and attempted suicide in eating disorders, obesity and weight–image concern. *Eating Behaviors* 7(4), 384-394.

Rockwood, K. and Mitnitski, A.B. (2007). Frailty in Relation to the Accumulation of Deficits. *Journals of Gerontology Series A: Biological and Medical Sciences* 62, 722-727.

Schünemann, J., Strulik, H., Trimborn, T. (2017). The Gender Gap in Mortality: How Much Is Explained by Behavior?. *Journal of Health Economics* 54, 79-90.

Schulz, L.O., and Schoeller, D.A. (1994). A compilation of total daily energy expenditures and body weights in healthy adults. *American Journal of Clinical Nutrition* 60(5), 676-681.

Slentz, C. A., Duscha, B. D., Johnson, J. L., Ketchum, K., Aiken, L. B., Samsa, G. P., ... and Kraus, W. E. (2004). Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE – a randomized controlled study. *Archives of Internal Medicine* 164(1), 31-39.

Statistics Canada (2011), Physical activity of Canadian adults: Accelerometer results from the 2007 to 2009 Canadian Health Measures Survey, `http://www5.statcan.gc.ca/access_acces/alternative_alternatif.action?l=eng&loc=11396-eng.pdf`.

Steinglass, J. E., Figner, B., Berkowitz, S., Simpson, H. B., Weber, E. U., and Walsh, B. T. (2012). Increased capacity to delay reward in anorexia nervosa. *Journal of the International Neuropsychological Society* 18(4), 773-780.

Strulik, H. (2014). A mass phenomenon: The social evolution of obesity. *Journal of Health Economics* 33, 113-125.

Strulik, H. (2018). Smoking kills: An economic theory of addiction, health deficit accumulation, and longevity. *Journal of health Economics* 62, 1-12.

Strulik, H. (2019). I shouldn't eat this donut: Self-control, body weight, and health in a life cycle model. *Journal of the Economics of Ageing* 14, 100175.

Strulik, H. (2021). From pain patient to junkie: An economic theory of painkiller consumption and its impact on wellbeing and longevity. *Journal of Health Economics* 76, 102432.

Strulik, H., and Trimborn, T. (2018). Hyperbolic discounting can be good for your health. *Journal of Economic Psychology* 69, 44-57.

Strulik, H., and Vollmer, S., (2013). Long-run trends of human aging and longevity. *Journal of Population Economics* 26(4), 1303-1323.

Stutzer, A., and Meier, A.N. (2016). Limited Self-control, Obesity, and the Loss of Happiness. *Health Economics* 25, 1409-1424.

Sundgot-Borgen, J., and Torstveit, M. K. (2004). Prevalence of eating disorders in elite athletes is higher than in the general population. *Clinical Journal of Sport Medicine* 14(1), 25-32.

Sudi, K., Oettl, K., Payerl, D., Baumgartl, P., Tauschmann, K., and Mueller, W. (2004). Anorexia athletica. *Nutrition* 20(7-8), 657-661.

Townsend, N., Wickramasinghe, K., Williams, J., Bhatnagar, P., and Rayner, M. (2015). *Physical Activity Statistics 2015*. British Heart Foundation: London.

U.S. Department of Transportation (2021). Departmental Guidance on Valuation of a Statistical Life in Economic Analysis. `https://www.transportation.gov/office-policy/transportation-policy/revised-departmental-guidance-on-valuation-of-a-statistical-life-in-economic-analysis`

Walsh, B. T. (2013). The enigmatic persistence of anorexia nervosa. *American Journal of Psychiatry* 170(5), 477-484.

WHO (2018): Obesity: Situation and trends `http://www.who.int/gho/ncd/risk_factors/overweight/en/`