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The Gender Gap in Mortality: How Much Is Explained by Behavior?*

Johannes Schünemann†, Holger Strulik‡ and Timo Trimborn§

July 2016.

Abstract. In developed countries, women are expected to live about 4-5 years longer than men. In this paper we develop a novel approach in order to gauge to what extent gender differences in longevity can be attributed to gender-specific preferences and health behavior. For that purpose we set up a physiologically founded model of health deficit accumulation and calibrate it using recent insights from gerontology. From fitting life cycle health expenditure and life expectancy we obtain estimates of the gender-specific preference parameters. We then perform the counterfactual experiment of endowing women with the preferences of men. In our benchmark scenario this reduces the gender gap in life expectancy from 4.6 to 1.4 years. When we add gender-specific preferences for unhealthy consumption, the model can motivate up to 88 percent of the gender gap. Our theory offers also an economic explanation for why the gender gap declines with rising income.

Keywords: health, aging, longevity, gender-specific preferences, unhealthy behavior.
JEL: D91, J17, J26, I12.

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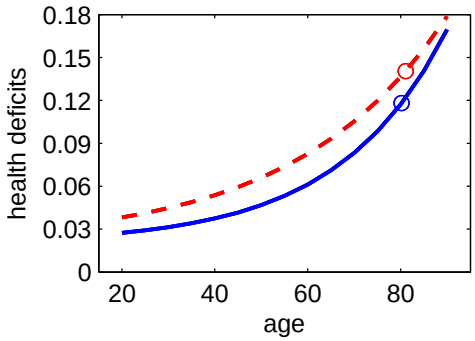
1. Introduction

In this paper we propose a new approach, based on counterfactual computational experiments, in order to identify the behavioral (or economic) contribution to observed sex differences in morbidity and mortality. Since the first life tables were constructed in the mid 18th century it is a well established fact that women, on average, live longer than men (Luy, 2003). While the gender-gap, defined as female excess life expectancy, was first observed in the now developed countries, in the 21st century it is basically a universal phenomenon. Woman are now on top everywhere (Barford et al., 2006). The size of the gender gap is not a natural constant. In the OECD countries it was increasing in the period 1950-1970 and declining afterwards. Moreover, across contemporaneous countries, the gender gap appears to be strongly negatively associated with GDP per capita (Cullen et al., 2015). In the richest countries women are expected to live about 4 to 5 years longer than men (Oksuzyan et al., 2008). The variance of the gender gap across time and countries is a first indication that it cannot be explained as a purely biological phenomenon.

Any attempt to explain the gender gap in mortality has to address the phenomenon that women, at any age, appear to be less healthy than men. Figure 1, replicated from Mitnitski et al. (2002a), shows the estimated association between age and health deficits for Canadian men and women, measured by the frailty index. The frailty index is suggested by gerontologists as one particularly straightforward metric to measure health deficits. As humans age they develop an increasing number of aging-related disorders, ranging from mild nuisances (e.g., reduced vision, incontinence) to serious conditions (e.g., strokes, cancer). The frailty index provides the proportion of deficits that an individual has, from a long list of potential deficits. The power law association shown in Figure 1 is estimated with great precision ($R^2 > 0.95$) for men and women and suggests a "natural rate" of aging, which is estimated to be around 4 percent for men and 3 percent for women. Similar results have been obtained for similar populations (e.g. U.S. Americans, Australians, and Swedes; Rockwood and Mitnitski, 2007). The stylized fact that women start out unhealthier and age slower than men has a microfoundation in biology, based on reliability and redundancy of body cells (Gavrilov and Gavrilova, 1991; Fries, 1980).

Combined with the gender-gap in longevity, the law of health deficit accumulation implies that, on average, women at the time of death have accumulated more health deficits than men.

FIGURE 1: HEALTH DEFICITS AND LIFE EXPECTANCY FOR MEN AND WOMEN



Estimated frailty index for Canadian men (solid line) and women (dashed line). Data from Mitnitski et al. (2002a). Dots: life expectancy at 20, Data from World Life Expectancy (2015).

This notion is confirmed by another study of Mitnitski et al. (2002b) showing that the sex-specific mortality rates are estimated with great precision as a power law (log-log association) of the frailty index ($R^2 > 0.95$) and that men are more likely to die than women for any given number of health deficits. The inverse association between health and longevity across sexes is known as the morbidity-mortality paradox (Verbrugge, 1988; Case and Paxson, 2005; Kulminsky et al., 2008).

The gender gap and the morbidity-mortality paradox has attracted researchers from the natural and social sciences. Across fields there seems to be consent that any sufficient explanation should be based on biological as well as on behavioral factors (Oksuzyan et al., 2008). However, it seems to be hard to assess how much biology and behavior contribute to the explanation of these phenomena. Answering these questions is important in order to assess how much of the gender-gap could potentially be closed by policy and in order to predict future trends of the gender gap.

At the biological side it has been observed that women are more likely to suffer from acute illnesses and nonfatal chronic conditions, including arthritis, constipation, thyroid conditions, gall bladder conditions, headaches, and migraine. These conditions cause poorer self-rated health but contribute little to the risk of death. For given age, men are more likely to suffer from life-threatening chronic diseases, including coronary heart disease, some cancers, cerebrovascular disease, emphysema, liver cirrhosis, and kidney disease (Verbrugge, 1985; Bird and Rieker, 1999; Case and Paxson, 2005). Biology offers explanations based on hormonal, autoimmune, and genetic factors. For example, women have lower risk of cardiovascular disease due to the

protective effects of estrogen which lowers the levels of low density lipoproteins. Women are also equipped with a better immune system, which makes them less likely to die from parasitic and infectious diseases but more likely to suffer from autoimmune diseases. Regarding genetics, women are less likely to suffer from diseases linked to the X-chromosome, of which men have only one. Since X-linked defects are usually encoded in recessive genes, men are more likely to develop these conditions (Holden, 1987; Austad, 2006; Oksuzyan et al. 2008).

At the behavioral side it is observed that women are on average less willing to engage in risk-taking health behaviors, such as smoking, drinking, drug use, and hazardous driving. Women are also more likely to prefer fruits and low fat foods and to consume less meat and salt. Women utilize health care services more frequently than men, even when controlled for sex specific-conditions; women use more prescription- and over-the-counter-medicine, they demand more vitamin supplements, and they utilize routine screening exams more frequently (Waldron, 1985; Wardle et al., 2004). One study concludes that women demand not only more total medical care but also more of each type of care, an observation which seems to hold across developed countries and over time (Sindelar, 1982).

Most of the studies on gender specific health behaviors and outcomes (including those on the frailty index) are based on self-assessments. This raises the concern of gender-specific reporting attitudes. On average more health-conscious women could over-emphasize their health deficits while on average more status-concerned men could under-report them. The seminal study of Case and Paxson (2005) resolved these concerns by showing that differences in self-rated health can be fully explained by differences in health conditions, leaving no room for a gender-bias in reporting. However, while men and women with the same conditions had similar self-reported health, men were observed to be more likely to die from these conditions, indicating that men experience them with greater severity.

Most important for our study is perhaps the observation that men and women essentially suffer from the same life-threatening diseases but that women tend to experience them later in life. For example, the incidence of coronary heart disease starts rising about 10 years earlier for men than women (Verbrugge, 1985). This observation is useful to explain the morbidity-mortality paradox. It is also quite intuitive. Because the chronic and fatal conditions that lead to an earlier expiry of men are aging related and because both men and women age as they get older, we expect women to develop these diseases as well, yet later in life. Simplifying and summarizing

these insights in terms of Figure 1, we observe a gender gap because women suffer more from non-fatal health deficits and develop fatal health deficits later in life. The female delay in fatal health deficit accumulation can be explained by female biology (e.g. estrogen production) as well as by female behavior (e.g. less smoking).

One approach suggested in order to assess how much of the gender gap can be attributed to biology and behavior is to consider subpopulations, in which men's and women's life style is more similar. Preston and Wang (2006), for example, investigate sex differences in the mortality of smokers and non-smokers and estimate that changes in smoking patterns contributed about 20 % to the declining gender-gap. Luy (2003) compares life expectancy of the cloistered subpopulation in Bavaria with that of the German population at large. For the period 1965 to 1990 he observes that the life expectancy of nuns exceeds that of monks by 2.3 years. At the same time, German women on average lived between 5 and 6 years longer than men. While studies of nuns and monks, smokers, and other subpopulations suggest that a large part of the gender gap can be motivated by gender-specific preferences and behavior, there remains the question of out-of-sample validity.

In this paper we propose an alternative, theory-based approach that utilizes counterfactual computational experiments. We set up a standard economic model of health deficit accumulation (Dalgaard and Strulik, 2014) and calibrate it separately for men and women such that it replicates life expectancy and the life cycle trajectories for health expenditure and health deficit accumulation for average (white) male and female U.S. Americans in the year 2010. This leads to gender-specific estimates of the coefficient of risk-aversion and the weight of health in the utility function. We then re-run the female life cycle with the preference parameters of men. In our benchmark case this experiment reduces the gender gap from 4.6 to 1.4 years, suggesting that about 70 percent of the gender-gap can be motivated by health behavior. We then extend the model by allowing individuals to derive utility from unhealthy consumption and calibrate the model with data on smoking. In contrast to the basic model, for which the foundation in gerontology allows for a calibration with zero degrees of freedom, there is some parameter uncertainty entailed by the augmented model. We thus provide a sensitivity analysis. Depending on specification we estimated with the augmented model that between 85 and 88 percent of the gender gap can be motivated by gender-specific preferences and behavior.

The related health economics literature provides relatively little theory-based discussion of the gender gap of mortality. One reason is certainly that the literature was dominated for several decades by the health capital model (Grossman, 1972, 2000). Health capital is problematic because it is a latent variable, which is exclusively used by economists and which is alien to the medical and biological sciences. The fact that health capital cannot be observed makes it hard if not impossible to calibrate any theory of health behavior with data (for difficulties estimating the health capital model see e.g. Wagstaff, 1986). Health deficits, in contrast, can be reported not only by doctors and scientists but actually by everyone in society. The frailty index provides a straightforward metric for health deficits and its relation to age and mortality can be estimated with high precision. Moreover, since the health capital model counterfactually assumes that healthy people age faster than unhealthy people of the same age, it involves some undesired predictions, which makes it hard to fit actual life cycle trajectories (for a critique, see e.g. Zweifel and Breyer, 1997; Case and Deaton, 2005; Almond and Currie, 2011; Dalgaard and Strulik, 2015; Strulik, 2015a).[1] Finally, the Grossman model, by construction, cannot directly capture the morbidity-mortality paradox as defined in natural science, which would be important for any model focusing on gender specific health behavior and longevity.

An exception is the study by Felder (2006), which avoids the problematic health capital assumption and shows that a gender gap in longevity emerges in a simple life cycle model under the assumptions that, at any age, it is more costly to survive for men than for women and that men and women share the same preferences and are endowed with the same life time wealth. Here, in contrast, we emphasize the role of gender-specific preferences for health behavior and identify their quantitative importance for observable health outcomes and longevity.

Conceptually, our paper is also related to a strand of recent studies utilizing the health deficit approach to (re-)investigate the Preston curve (Dalgaard and Strulik, 2014), the historical evolution of retirement (Dalgaard and Strulik, 2013), the education gradient (Strulik, 2012), age-consumption profiles (Strulik, 2015c), the role of adaptation for health behavior and health outcomes (Schünemann et al., 2015), and the optimal design of social welfare systems (Grossmann and Strulik, 2015).

[1] Ehrlich and Chuma (1990) briefly discuss gender specific mortality trends in the context of the Grossman model, though without developing the model for men and women. Forster (2001) simulates different versions of the Grossman model with unhealthy consumption, though without reference to actual data. Case and Deaton (2005) discuss gender-specific health outcomes against the background of the Grossman model.

The remainder of the paper is organized as follows. The next section sets up the basic model, calibrates it for a male and female Reference American and estimates the behavioral contribution, stemming from health care demand, to the observed gender-gap. Section 3 extends the model to include unhealthy consumption and estimates the contribution of behavior, separated by health care demand and unhealthy consumption, to the observed gender gap. Section 4 concludes.

2. The Basic Model

2.1. **Setup.** We consider the basic health deficit model by Dalgaard and Strulik (2014) augmented with utility derived from a good state of health. This allows us to consider two motives for health investments: the desire for longevity and the experience of high instantaneous utility in a good state of health. All variables and parameters are potentially gender-specific. However, for convenience we omit a gender-specific index for now and introduce it later when it becomes relevant for the calibration of the model. As for the original health capital model (Grossman, 1972) and as for the basic health deficit model, we consider the optimal life cycle decisions of individuals but here we differentiate between men and women.[2] Men and women are subject to biological aging. Following Mitnitski et al. (2002a) and Dalgaard and Strulik (2014) we assume that health deficits evolve according to

$$\dot{D}(t) = \mu(D(t) - Ah(t)^{\gamma} - a), \tag{1}$$

in which t is age. Initial deficits at age 0 are given by $D(0) = D_0$ and terminal health deficits at death are given by $D(T) = \bar{D}$. Since we consider adults, the initial age 0 will later be set to 20 years. Individuals have the possibility to slow down deficit accumulation by deliberate health investments $h(t)$. As explained in detail in Dalgaard and Strulik (2014), μ is considered to be a physiological parameter that captures the inherent force of aging; A (scale) and γ (curvature) represent the parameters of the health technology; and a measures environmental influence beyond individual control. In order to disentangle mechanisms we focus on health care demand as the sole gateway for health behavior. Later, we augment the model by gender-specific demand for unhealthy consumption.

Longevity T is finite and endogenous. For simplicity we consider a deterministic model. Death happens when $\bar{D}$ health deficits have been accumulated. As shown in Strulik (2015b) and

[2] The insights gained in this study could be used to investigate the joint life cycle decisions of aging couples. We leave this interesting application for future research.

Schünemann et al. (2015), allowing death to be a stochastic event adds more realism and complexity but contributes very little to the understanding of mechanisms and leaves quantitative results virtually unchanged. As shown later in greater detail, with gender-specific terminal value $\bar{D}$ we take into account that the health deficits accumulated by men and women of the same age have different impact on survival. Men develop potentially deadly deficits (e.g. cardiovascular diseases) earlier in life and are thus expected to die at a lower level of $\bar{D}$. Women develop more but less severe diseases (e.g. migraine, arthritis) early in life and die at a larger level of $\bar{D}$.

Men and women maximize life time utility,

$$\int_0^T e^{-\rho t} U(c(t), D(t)) \mathrm{d}t, \qquad U(c(t), D(t)) = \left(\frac{D_0}{D(t)}\right)^{\alpha} \tilde{u}(c(t)), \tag{2}$$

$$\tilde{u}(c(t)) \equiv \begin{cases} \frac{(c(t))^{1-\sigma}-1}{1-\sigma} & \text{for } \sigma \neq 1 \\ \log\left(c(t)\right) & \text{for } \sigma = 1 \end{cases}, \tag{3}$$

in which $\rho \geq 0$ is the rate of time preference. Instantaneous utility is a function of consumption and health. In particular, utility is negatively affected by increasing frailty, measured by accumulated health deficits relative to the state of "best health", given by initial deficits D_0. The parameter α controls by how much an additional health deficit affects the marginal utility from consumption. The parameter σ is the inverse of the intertemporal elasticity of substitution. We impose this specific utility function because its calibrated parameters can be confronted with recent estimates of the impact of health deficits on marginal utility by Finkelstein et al. (2013).

Individuals receive a constant gender-specific wage income w which they either spend on buying consumption goods c and health services h or save for later purpose. Individuals have access to financial markets and save or borrow at a net interest rate r such that individual wealth k evolves according to

$$\dot{k}(t) = w + rk(t) - c(t) - ph(t). \tag{4}$$

with $k(0) = k_0$ and $k(T) = k_T$ and p is the exogenous relative price of health goods. Notice that, since individuals base their optimization calculus on permanent income, the solution would not change by the introduction of an exogenous age of retirement or by an interim phase during which men or women are absent from the labor market.

Individuals maximize (2) subject to (1), (3), and (4) and the initial and terminal conditions. The associated Hamiltonian reads

$$\mathcal{H}(t) = U(c(t), D(t)) + \lambda_D(t)\mu(D(t) - Ah(t)^\gamma - a) + \lambda_k(t)(w + rk(t) - c(t) - ph(t)).$$

Since longevity T is endogenous, individuals face a free terminal time problem. The usual transversality conditions are replaced by the requirement that the Hamiltonian assumes the value of zero at the time of death.

From the first-order conditions for the maximization of $\mathcal{H}$, we obtain the Euler equation for consumption growth,

$$\frac{\dot{c}(t)}{c(t)} = \frac{1}{\sigma}\left(r - \rho - \alpha\frac{\dot{D}(t)}{D(t)}\right). \tag{5}$$

Notice that, if the state of health were not affecting instantaneous utility ($\alpha = 0$), equation 5 would reduce to the standard Ramsey rule. For $\alpha > 0$, health deficit accumulation slows down consumption growth over time because the utility function implies a negative cross-derivative, $U_{cD} \equiv [\partial^2 U(c(t), D(t))]/[\partial c \partial D] < 0$. Hence, with deteriorating health, the marginal utility from consumption decreases. This is in line with a recent empirical study by Finkelstein et al. (2013), which estimates that a one-standard deviation increase in chronic diseases is associated with a 10-25% decline in the marginal utility from consumption.

Secondly we derive from the first-order conditions the growth rate of health investments as

$$\frac{\dot{h}(t)}{h(t)} = \frac{1}{1-\gamma}\left(r - \mu - \frac{\alpha\mu A\gamma h(t)^{\gamma-1}}{p}\left(\frac{D_0}{D(t)}\right)^{-\alpha} U(c(t), D(t))\right). \tag{6}$$

Notice that for $\alpha = 0$, equation (6) reduces to the Health Euler equation derived in Dalgaard and Strulik (2014). For $\alpha > 0$, the health component of the utility function reduces growth of health investments over the life cycle. The (Edgeworth-) complementarity between health and consumption induces individuals to spend relatively more on health and consumption early in life, when the state of health is good.

2.2. **Model Calibration.** We calibrate the model to match initial deficits D_0, final deficits $\bar{D}$, longevity T and life cycle health investments $h(t)$ in the year 2010 for 20 years old U.S. American men and women. We mostly follow the calibration strategy of Dalgaard and Strulik (2014). Accordingly, the biological parameters can be easily inferred from Mitnitski et al. (2002a) who estimate a force of aging of $\mu_F = 0.031$ for women and $\mu_M = 0.043$ for men. Since Mitnitski

et al.'s regression does not involve children (individuals under 20 are not well described by the law of frailty), we assume that individuals are born at the age of 20. As the initial value of deficits of a 20-year old individual we can back out $D_F(0) = 0.0381$ and $D_M(0) = 0.0274$ from the regression equation for women and men, respectively. Final deficits are calculated likewise according to the life-expectancy at 20 of women (81,7 years) and men (77,1 years), yielding estimates of $\bar{D}_F = 0.1429$ and $\bar{D}_M = 0.1059$; data on life expectancy is from NVSS (2014). The notion that the health deficits accumulated by women are less fatal than those of men is captured in reduced form by the fact that $\bar{D}_F$ exceeds $\bar{D}_M$. In other words, women develop severe health deficits later in life than men but are afflicted more by mild health deficits at any age.

In order to determine gender-specific income, we take the average of gender-specific wages and salaries for U.S. American single men and women in the year 2010 from BLS (2012), which was $w_F =$ \$ 17303 for females and $w_M =$ \$ 27928 for males. In order to assure that the saving motive is confined to that of health and consumption expenditure, we assume the initial and final capital stock to be zero. Further, we set $r = 0.07$, according to Jones and Williams' (2000) estimate based on Mehra and Prescott (1985).

For the benchmark run we calibrate the model such that men and women share all parameters aside from the biological parameters μ, D_0, and $\bar{D}$, the preference parameters σ and α and the scale parameter of health technology A. We take the estimate of the environmental constant directly from Dalgaard and Strulik (2014), $a = 0.013$. With regard to the health technology, we have reliable estimates by Dalgaard and Strulik (2014) and Hall and Jones (2007) for the curvature parameter of $\gamma = 0.2$. The preference parameters σ and α and the health technology parameter A are calibrated in order to match the trajectory of gender-specific health investments and life expectancy for men and women. We solve the model numerically using the relaxation algorithm approach of Trimborn et al. (2008)[3].

While health technology A positively correlates with life expectancy for given health expenditure, the preference parameters mainly affect the health investment path of individuals. Therefore we next explain the impact of these parameters on the shape and position of the gender-specific health-investment-for-age curve. The predominant effect of increasing σ is an

[3]More information on the solution procedure is provided in the appendix to Schünemann et al. (2015).

upward shift of the health investment path. Intuitively, life time utility consists of instantaneous utility ('utility per year') summed up over the life time ('years per life'). In other words, life-time utility is linear in life time and concave in period utility. A higher σ means more curvature of period utility, i.e. lower marginal utility from consumption at any age. Consequently individuals put less emphasis on period utility and more emphasis on longevity, which allows for an extended experience of period utility. The induced preference for longevity triggers more health expenditure at any age.

The weight of health in utility, α, in contrast, affects predominantly the slope of the health-investment-for-age curve. A higher α flattens the age trajectory of health expenditure by raising $h(0)$ and reducing $h(T)$. Without health in the utility function, individuals prefer an increasing health-investment profile, as long as the return on capital markets r exceeds the rate of aging μ (see also Dalgaard and Strulik, 2014). A direct impact of health on utility increases the cost of investing little in health at young ages, a cost that increases in α. Consequently, individuals prefer a flatter health expenditure profile for higher α's.

We estimate gender-specific α's, σ's and A's such that the model provides the best fit of observed life expectancy and the data on total personal health care spending by gender and age group according to MEPS (2010).[4] We obtain the estimates $\sigma_F = 1.39$, $\sigma_M = 1.15$, $\alpha_F = 0.125$, $\alpha_M = 0.01$ and $A_F = 0.0013$, $A_M = 0.0014$. In order of magnitude our estimates of σ are consistent with recent estimates of the intertemporal elasticity of substitution, suggesting that the "true" value is probably close to unity (e.g. Chetty et al., 2006) or slightly above unity, around 1.2 (Layard et al., 2008). Recalling that σ coincides with the degree of relative risk aversion for time-separable utility, our estimates are also consistent with the result of several studies finding that women tend to be more risk averse than men in a vast majority of economic tasks. Croson and Gneezy (2009) review the experimental economics literature on gender specific preferences and conclude that men take riskier decisions in both hypothetical and real gambles. These results can be also observed when looking at direct evidence from risky decisions made by actual market participants (see, for example, Cohen and Einav (2007) on auto insurance contracts and Sunden (1998) on pension contribution plans). Mazzocco (2008) estimates the gender-specific intertemporal elasticity of substitution using data from the Consumer Expenditure Survey and finds it to be significantly lower for women than for men.

[4]We estimate the paramerter using an extended method of simulated moment; see the Appendix for technical information on the estimation method.

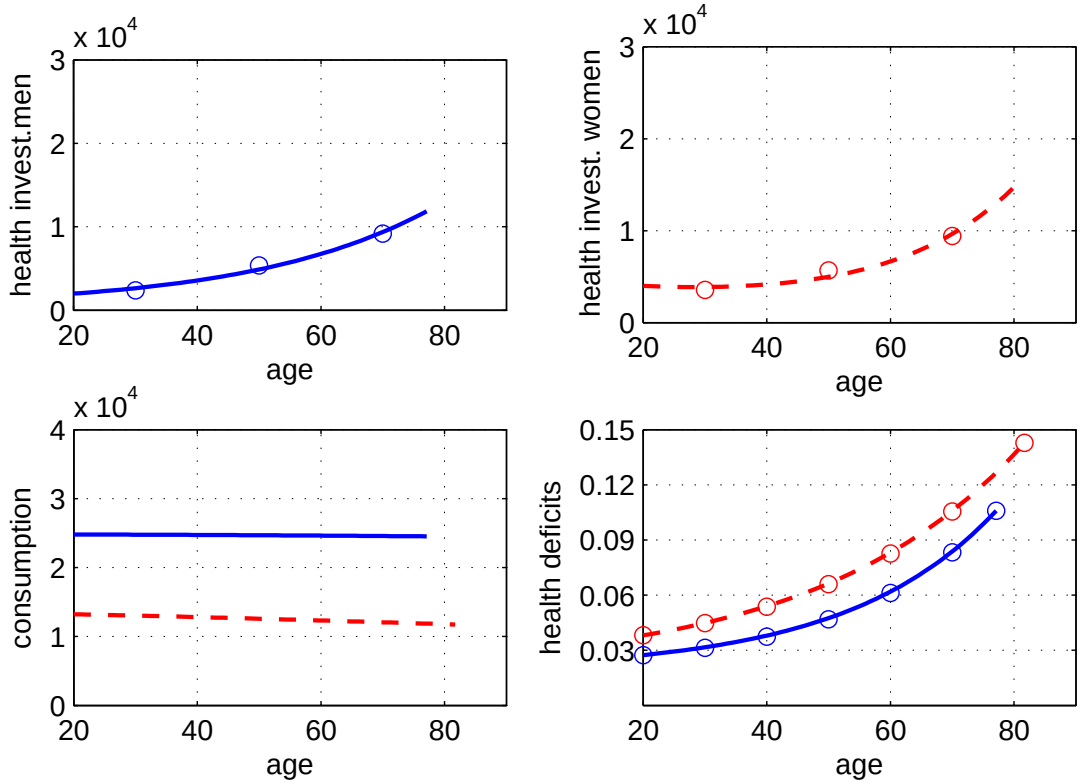
Our estimates of the α's imply that women care more for health in terms of instantaneous utility than men, in line with the evidence presented in the Introduction. A one-standard-deviation increase in health deficits reduces the marginal utility from consumption of women by 4.1% and that of men by 0.4%. Both values are below the point estimate of Finkelstein et al. (2013) of 11% (with a 95% confidence band from 2.7% to 16.8%). However, there are other studies estimating a lower (or higher) effect of health on marginal utility (see Finkelstein et al., 2009, for an overview). Unfortunately, Finkelstein et al. do not report gender-specific results. Gender effects are buried together with the impact of other controls in individual fixed effects. One explanation for our relatively low estimates of α is that we consider the whole adult life whereas Finkelstein et al. focus on individuals above 50. They also use a smaller set of health deficits than we do by calibrating the frailty index of Mitnitski et al. (2002a). The estimates for A imply that in the basic model the health technology of men is estimated to be mildly superior to that of women.

2.3. **Results.** Figure 2 shows the predicted life cycle trajectories for men (solid lines) and women (dashed lines) for the calibrated benchmark case. Data points are indicated by dots. Unlike the Grossman (1972) model, the health deficit model predicts that health expenditure increases with age at all ages. Quantitatively, however, men and women invest differently in their health. As shown in the upper panels, women's health expenditure exceeds that of men in young and middle age. Only near the death of men health expenditures converge. After that, women continue to live for 4.6 more years with further rising health expenditure. As shown in the lower left panel, women finance their extended desire for health care by consuming less. Most of the literature on life-cycle consumption focusses on households to which our results for individuals cannot be compared directly. Our predicted consumption trajectories, however, are in line with the observation that within households women consume on average 67% of men's consumption (Lise and Seiz, 2011). Considering that husband and wife (at least partially) pool their income, the model prediction that single women consume on average 53% of single men's consumption is consistent with this observation. The consumption trajectories are consistent with the observation that age-consumption profiles are essentially flat across the life cycle, once children are controlled for (Browning and Ejrnæs, 2009).[5] The panel at the lower right-hand side

[5] If the interest rate would be somewhat higher than the time preference rate, a hump shaped consumption profile would emerge (see eq. (5) and Strulik, 2015b).

shows that the gerontological parameters together with gender-specific preferences are able to predict the actual accumulation of health deficits of men and women. Women are less healthy at any age yet die 4.6 years later than men. While we imposed that death occurs when $\bar{D}_M$ and $\bar{D}_F$ deficits are reached for men and women, the result that death actually happens at the empirically observed life expectancy of men and women is derived by gender-specific health expenditure. Notice that these results are invariant to the introduction of an exogenous retirement age, as long as life time income stays the same.

FIGURE 2: OPTIMAL AGING AND DEATH OF MEN AND WOMEN: LIFE TRAJECTORIES



Blue (solid) lines: men, red (dashed) lines: women. Dots: data (deficits from Mitnitski et al. 2002a; health expenditure from MEPS, 2010). See main text for details.

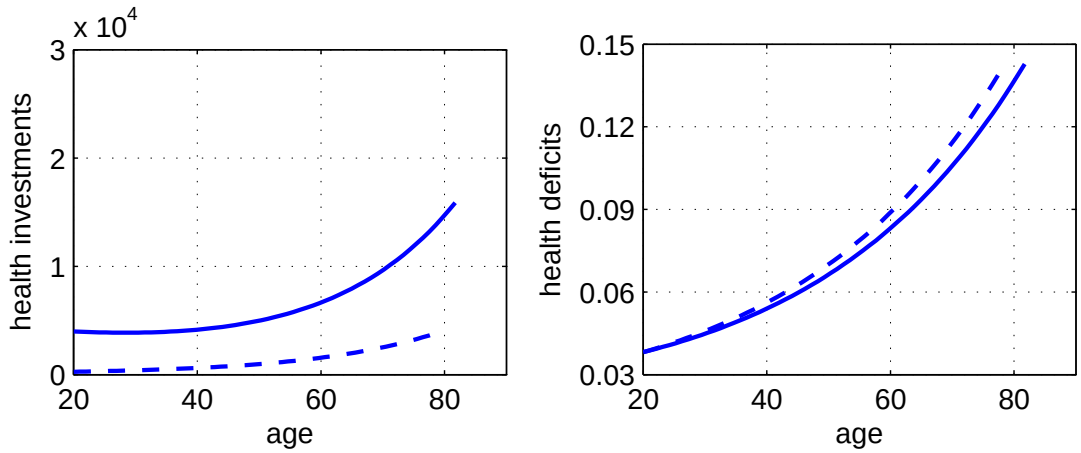
It is worth emphasizing that gender-specific preferences are *essential* to get gender-specific health investment right. The biological parameters, taken for themselves, drive health investment paths in the "wrong direction". Because women develop deficits at a slower pace and these deficits are less severe (since $\bar{D}_F > \bar{D}_M$), a model with unisex preferences would predict that men spend more on health investment than women.

We now come to our main counterfactual experiment. We endow women with male preferences and compute how much this reduces the observed gender gap in life expectancy. For the

benchmark run this means that we put $\sigma_F = \sigma_M = 1.15$ (instead of 1.39) and $\alpha_F = \alpha_M = 0.01$ (instead of 0.125). In other words, women become less risk avers and appreciate a good state of health less. They are in these respects equal to men. We keep everything else from the benchmark scenario, implying that we keep gender differences in income and health technology as well as the gender-specific physiology parameters.

The preference change results in a reduction of the gender gap in life expectancy from 4.6 to 1.4 years, i.e. by 70%. Figure 3 shows why. Solid lines reiterate the trajectories for women's health expenditure and deficit accumulation from Figure 2. Dashed lines reflect the new optimal trajectories obtained when women have men's preferences. Women then spend substantially less on health at any age, and in particular at old age. As a result they accumulate health deficits faster and die at age 78.5 (instead of 81.7) when $\bar{D}_F$ deficits are accumulated.

FIGURE 3: COUNTERFACTUAL: IF WOMEN HAD MEN'S PREFERENCES



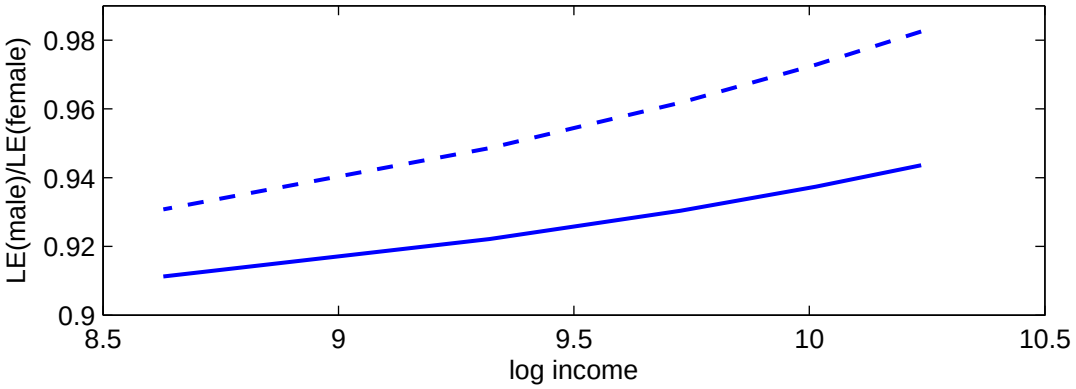
Solid lines: women benchmark run (from Fig. 1); life expectancy at 20: 81.7 years. Dashed lines: women with men's preferences $\sigma_F = \sigma_M$, $\alpha_F = \alpha_M$; life expectancy at 20: 78.5 years.

We next investigate the response of the gender-gap to rising income. For that purpose we successively reduce the labor income of men and women by the same factor until it reaches 20% of benchmark income. The solid line reflects the gender-gap under gender-specific preferences as calibrated above. The abscissa measures income in terms of implied male GDP per worker. In line with the post-war evidence, the gender-gap declines with rising income. Cullen et al. (2015) obtain across developed countries a correlation coefficient of 0.0095 between log income and the gender gap in life-expectancy at age 25 (their Figure 3).[6] Across U.S. counties, they estimate a

[6] Notice that we here consider a different definition on the gender gap as the ratio of male vs. female life expectancy (following Cullen et al., 2015).

partial correlation of 0.059 between log income and the gender gap in terms of survival until age 70 (Table 1). Our benchmark model predicts a slope of 0.017, a value between the two estimates of Cullen et al. Our model thus corroborates the prediction that the gender-gap will decline further in the future, provided that income continues to grow.[7]

FIGURE 4: INCOME AND THE GENDER GAP



Solid line: predicted male/female ratio of life expectancy for alternative income levels and benchmark parameters. Estimated linear slope coefficient 0.017 ($R^2 = 0.986$). Dashed line: predicted gender gap if women had male preferences.

The explanation is straightforward: with rising income the slopes of the strictly concave utility functions $U(c)$ become more similar ($\tilde{u}$ in (3) approaches infinity irrespective of gender). In other words, with rising income current consumption becomes less urgent and the difference between men and women in this regard (captured by $\sigma_M > \sigma_F$) matters less. Moreover, the gender gap declines also simply because rising income causes both men and women to live longer (to see this, consider generally rising life expectancy under the assumption of a constant absolute difference in longevity between men and women). The dashed line in Figure 4 shows the evolution of the gender gap if women had male preferences. The gap is initially smaller and is closed at a somewhat higher speed. Finally it should be noted that there is also a countervailing force (not investigated here), stemming from a declining gender income gap, which would taken for itself, increase the gender-gap in life-expectancy since women care more about their health. In our benchmark scenario, the gender gap would increase from 4.6 to 6.2 if, ceteris paribus, women had male income.

[7] In line with these results, a recent large scale study using individual income data for the U.S. population documents a declining gender gap across social strata, running from about 0.92 at the tenth income percentile to about 0.97 at the 90th percentile (Chetty et al., 2016, Figure 2).

3. Unhealthy Consumption

3.1. **Setup.** As discussed in the Introduction, an important behavioral difference between men and women is the on average higher level of unhealthy consumption of men. To capture this behavioral aspect we next introduce unhealthy consumption into our model. Unhealthy consumption, denoted by u, modifies the model in three dimensions: its consumption brings pleasure, costs money, and increases the speed of health deficits accumulation. In order to capture the pleasure aspect, we replace the instantaneous utility function $\tilde{u}$ in (3) by

$$\tilde{u}(c(t),u(t)) = \begin{cases} \frac{\left(\beta c(t)^{\psi}+(1-\beta)u(t)^{\psi}\right)^{\frac{1-\sigma}{\psi}}-1}{1-\sigma} & \text{for } \sigma \neq 1 \\ \frac{1}{\psi}\log\left(\beta c(t)+(1-\beta)u(t)\right) & \text{for } \sigma = 1. \end{cases} \tag{7}$$

The parameter β reflects the individual's preference for normal and unhealthy goods and ψ governs the elasticity of substitution between normal and unhealthy goods, $\epsilon = 1/(1-\psi)$. Notice that this motivates two gateways for the gender-gap. Men could just like unhealthy consumption more than women (reflected by higher male β) or they could consume more unhealthy goods because they are less risk averse and thus care less about future consumption and longevity (captured by higher male σ).

The equation of motion for health deficits is modified to

$$\dot{D}(t) = \mu(D(t) - Ah(t)^{\gamma} - a + Bu(t)^{\omega}). \tag{8}$$

Unhealthy consumption speeds up the process of health deficits accumulation (as the mirror image of health expenditure). The parameter B measures general unhealthiness of the unhealthy good u and ω measures the marginal return in terms of deficits. Goods that are unhealthy already in small doses would be characterized by a concave shape ($\omega < 1$), while goods that are unhealthy mostly when consumed in excess would be characterized by a convex shape ($\omega > 1$).

Finally, we define q as the relative price of the unhealthy good in terms of the normal good and modify the budget constraint according to

$$\dot{k}(t) = w + rk(t) - c(t) - ph(t) - qu(t). \tag{9}$$

Individuals maximize (2) subject to (7)-(9) and the same boundary conditions as for the simple model. This provides the first order conditions

$$\left(\frac{D_0}{D(t)}\right)^{\alpha}\left(\beta c(t)^{\psi}+(1-\beta)u(t)^{\psi}\right)^{\frac{1-\sigma-\psi}{\psi}}\beta c(t)^{\psi-1}=\lambda_k(t) \tag{10}$$

$$-\lambda_D(t)\mu A\gamma h(t)^{\gamma-1}=p\lambda_k(t) \tag{11}$$

$$\left(\frac{D_0}{D(t)}\right)^{\alpha}\left(\beta c(t)^{\psi}+(1-\beta)u(t)^{\psi}\right)^{\frac{1-\sigma-\psi}{\psi}}(1-\beta)u(t)^{\psi-1}+\lambda_D(t)\mu B\omega u(t)^{\omega-1}=p\lambda_k(t) \tag{12}$$

$$\lambda_D(t)(\rho-\mu)+\frac{\alpha}{D(t)}U(c(t),D(t))=\dot{\lambda}_D(t) \tag{13}$$

$$\lambda_k(t)(\rho-r)=\dot{\lambda}_k(t). \tag{14}$$

3.2. **Model Calibration.** In contrast to the basic model, calibration of the augmented model involves some parameter uncertainty. As a benchmark, we therefore assume that men and women have the same preferences with respect to health-neutral and unhealthy consumption, i.e. $\beta_F = \beta_M$. We thus assume for the moment that differences in unhealthy behavior are governed exclusively by differences in relative risk aversion. Further we assume that unhealthy consumption enters the equation for deficit accumulation linearly, i.e. $\omega = 1$. We later check sensitivity to these assumptions. Finally, we set the relative price of the unhealthy good q equal to 1.

We next explain how the new parameters affect the actually observed behavior regarding unhealthy consumption and present our calibration strategy. The parameters β and ϵ both affect the expenditure share on unhealthy consumption. The effect of β as the preference parameter can be readily seen from the utility function. A larger β means a lower preference for unhealthy goods and a lower expenditure share of these goods. A larger ϵ increases the willingness of individuals to substitute between health-neutral and unhealthy goods. For higher values of ϵ individuals tend to substitute away from unhealthy consumption because it reduces their health and life expectancy. Another effect of ϵ relates to the price elasticity of demand of unhealthy goods. For a higher value of ϵ, any increase of the price of unhealthy goods leads to a larger reduction in unhealthy consumption. Finally, the parameter B governs the extent to which unhealthy consumption translates into the accumulation of deficits and thus controls the resulting loss in life expectancy for given consumption of the unhealthy good.

Since most of the literature on unhealthy consumption focusses on cigarette and tobacco it appears reasonable to use this type of unhealthy consumption as a benchmark case. In 2010, the average expenditure share of tobacco products and smoking supplies in the U.S was 0.6% for single women and 1.2% for single men (BLS, 2012). We set the price elasticity of cigarette demand to -0.4, an intermediate value in the range of available estimates (Chaloupka and Warner, 2010) . Finally, we use from Pijoan-Mas and Rios-Rull (2014) the estimate of an average loss in life expectancy from smoking of 2.4 years for women and 2.7 years for men. We then use these additional data points to pin down the gender-specific parameters ϵ and B as well as $\beta_F = \beta_M = \beta$. The unisex parameters and the gender-specific physiological parameters are the same as for the basic model.

Table 1: Calibration Results

σ_F	σ_M	α_F	α_M	$\beta_F = \beta_M$	ϵ_F	ϵ_M	A_F	A_M	B_F	B_M
1.43	1.16	0.11	0.02	0.48	1.54	2.82	0.00148	0.00146	$7.28 * 10^{-6}$	$1.45 * 10^{-6}$

Calibration results for the augmented model with unhealthy consumption. Assumptions: $\beta_F = \beta_M$, $\omega = 1$, and $q = 1$.

The estimates of the σ's and α's deviate only marginally from those for the basic model. We obtain somewhat higher estimates for the σ's and a slightly lower (higher) estimate for the women's (men's) α once we introduce unhealthy consumption into our model. The health technology (A_F, A_M) is now estimated to be virtually the same for men and women. The estimate of β indicates that individuals have a slightly stronger preference for the unhealthy good. Notice that, in contrast to the standard model of goods demand, β does not coincide with the expenditure share of the unhealthy good. The expenditure share is much lower than $1 - \beta$ because individuals take into account the unhealthiness of the good in their consumption plans. Our estimates for ϵ suggest that men substitute more easily between healthy and unhealthy goods. This result is consistent with the observation that women struggle more in quitting smoking (see Sorensen and Pechacek, 1987, for a review). The estimates of the B's indicate that consumption of the unhealthy good has a larger impact on deficit accumulation of women compared to men. This finding is in line with studies investigating gender differences in the risk of developing smoking-related health disorders. Zang and Wynder (1996), for example, report that women are more likely to develop major lung cancer types at every level of exposure to cigarette smoke. The authors also conclude that these differences do not result from disparities in base-line exposure, smoking history or body size, but most likely from higher susceptibility

to tobacco carcinogens in women. One explanation for the higher sensitivity of women can be found in their relatively lower nicotine metabolism. Beckett et al. (1971) and Benowitz (1984) found that the total plasma clearance of nicotine, normalized by body weight, is lower in women than in men.

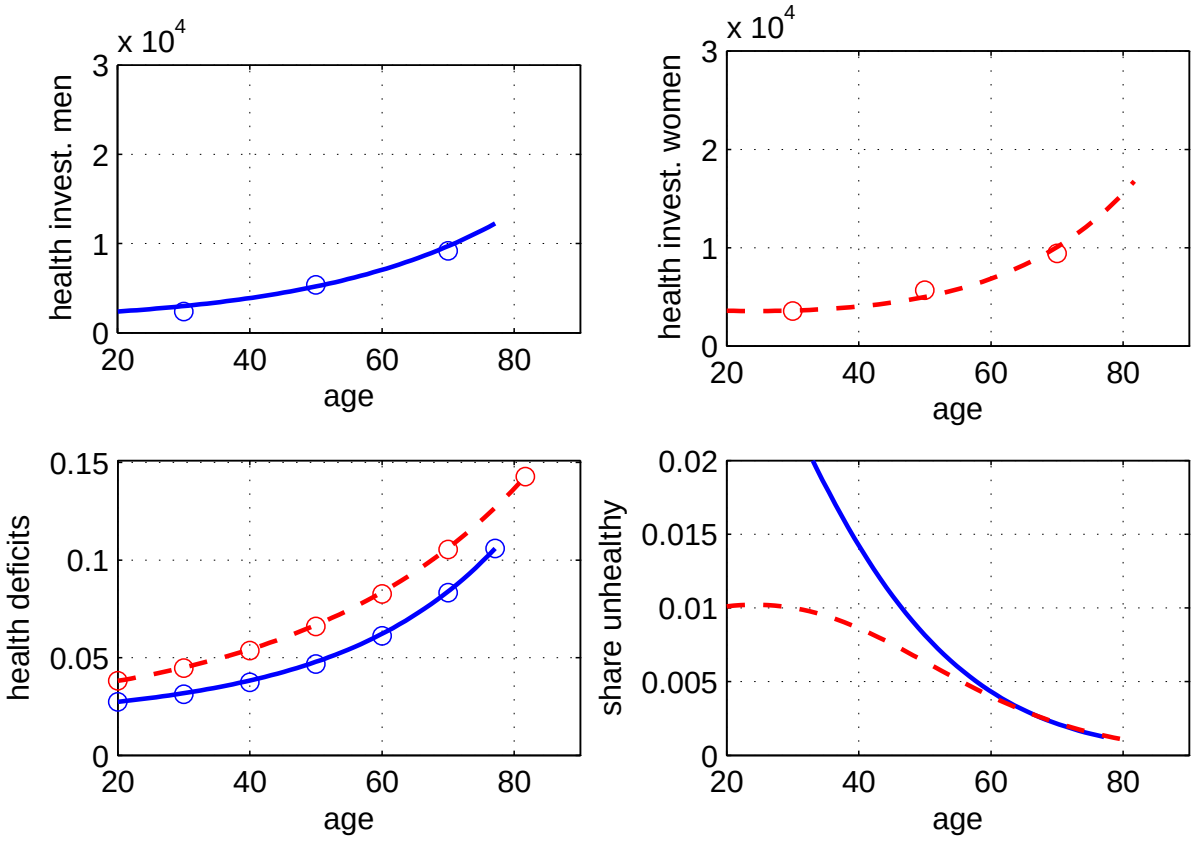
3.3. **Results.** Figure 5 shows the implied trajectories for health investments, deficits, and the share of unhealthy consumption. The age profiles for health-neutral consumption are similar to the basic model (and thus not shown). Again, the model performs reasonably well in matching life cycle health investments and deficits. The share of unhealthy consumption is decreasing in the course of aging, as observed by Strulik (2013), and consistent with the data.[8] By construction, women devote on average a smaller share of their expenditure to unhealthy consumption. The different slopes of the trajectories stem from the fact that women find it harder to substitute away from unhealthy consumption in the course of aging (due to their lower ϵ). Thus when they get older they decrease their level of unhealthy consumption more slowly.

We next provide results for our main counterfactual experiment of endowing women with male preferences. In addition to the same β, men and women are assumed to share the same σ, α and ϵ as well. Figure 6 shows the implied trajectories for health investments and the expenditure share of unhealthy consumption. As before, health investments of women decline substantially at any age. In addition, women now spend on average more on unhealthy consumption (especially at young age). As a result women are expected to die much earlier, at age 77.68. The gender gap in life expectancy declines by 4.02 years, implying that differences in behavior account for 87.5% of the observed gender gap in longevity.

3.4. **Sensitivity Analysis.** Finally we investigate the sensitivity of results to deviations from the benchmark assumptions. In our first sensitivity check we relax the assumption of unisex preferences with respect to unhealthy consumption. By successively increasing β_F, we open up another gateway for gender differences in unhealthy consumption, namely that men simply like unhealthy goods more than do women. Calibration results for female parameters are summarized in Table 2.

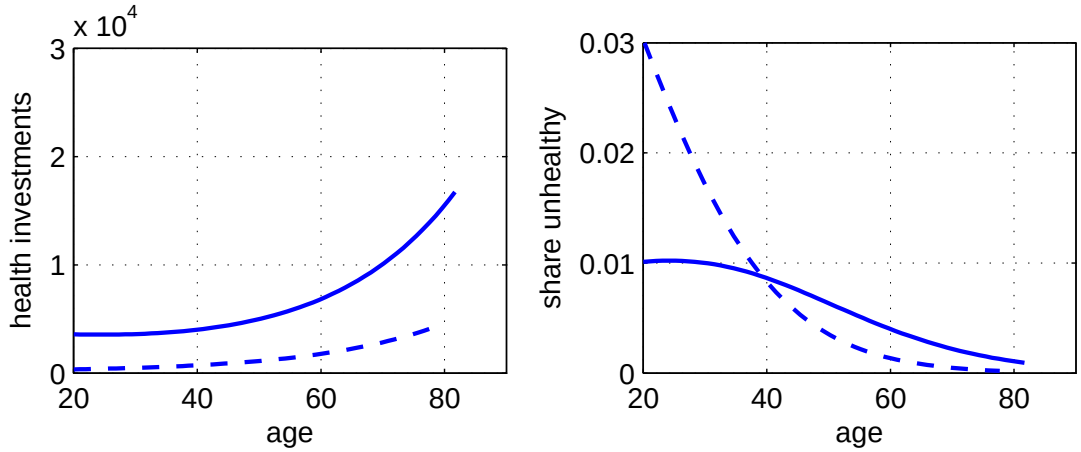
[8] According to the Centers for Disease Control and Prevention (CDCP, 2015), the share of smoking men in 2005 declined from 28 percent in the 18-24 age group to 8.9 percent in the above 65 age group (a decline of 68 percent). For females it declined from 20.7 in the 18-24 age group to 8.9 percent in the above 65 age group (a decline of 59 percent).

FIGURE 5: LIFE TRAJECTORIES FOR MEN AND WOMEN: THE CASE OF UNHEALTHY CONSUMPTION



Blue (solid) lines: men, red (dashed) lines: women. Dots: data (deficits from Mitnitski et al. 2002a; health expenditure from MEPS (2010). See main text for details.

FIGURE 6: COUNTERFACTUAL: IF WOMEN HAD MEN'S PREFERENCES



Solid lines: women benchmark run (from Fig. 5); Life expectancy at 20: 81.7 years. Dashed lines: women with men's preferences $\sigma_F = \sigma_M$, $\alpha_F = \alpha_M$, $\beta_F = \beta_M$, $\epsilon_F = \epsilon_M$. Life expectancy at 20: 77.68 years.

Table 2: Sensitivity Analysis β_F: When Women Like Unhealthy Consumption Less than Men

case	σ_F	α_F	ϵ_F	A_F	B_F	gap reduction
1) $\beta_F = 0.503$	1.43	0.12	1.47	0.00146	$7.5 * 10^{-6}$	3.99 (86.7%)
2) $\beta_F = 0.527$	1.42	0.12	1.43	0.00147	$7.6 * 10^{-6}$	3.91 (85.0%)

All other parameters are kept from the benchmark run ($\beta_M = 0.48$). Gap reduction refers to the reduction in the gender gap in longevity once we endow women with male preferences.

Comparing the results to the benchmark run, we observe a reduction in the estimate of ϵ_F. This response is intuitively plausible. A lower preference for the unhealthy good (higher β_F) drives the incentive for unhealthy consumption down. In order to match the observed expenditure share, ϵ_f needs to decrease to make it more difficult for women to substitute away from unhealthy consumption. The other parameter estimates do not change or change only marginally. Table 2 also shows the result of our counterfactual experiment. Imposing lower preference for unhealthy goods for women, changes the estimated reduction of the gender gap in longevity only marginally (the gap was 4.02 years in the benchmark run with unhealthy consumption).

We next analyze changes in the parameter ω, which measures the marginal return of unhealthy consumption in terms of deficits. Table 3 shows the calibration results for a 5% increase and a 5% decrease in ω and the associated results for our counterfactual experiment. Changes in ω translate into changes in ϵ and, accordingly, into changes in β. The most substantial impact occurs in the estimates for B. When ω increases, deficits accumulate at a higher pace. In order to match the observed loss of life expectancy from smoking, B needs to fall and vice versa. However, the effect of changing ω on the composition of the gender gap remains moderate.

Table 3: Sensitivity Analysis ω

case	σ_F	σ_M	α_F	α_M	$\beta_F = \beta_M$	ϵ_F	ϵ_M	A_F	A_M	B_F	B_M	gap reduction
1) $\omega = 1.05$	1.41	1.15	0.13	0.03	0.41	1.70	3.44	0.00148	0.00150	$4.4 \cdot 10^{-6}$	$1.1 \cdot 10^{-6}$	3.9 (85.6%)
2) $\omega = 0.95$	1.42	1.16	0.10	0.02	0.57	1.39	2.30	0.00148	0.00149	$9.6 \cdot 10^{-6}$	$2.0 \cdot 10^{-6}$	4.1 (88.3%)

All other parameters are kept from the benchmark run. Gap reduction refers to the reduction in the gender gap in longevity once we endow women with male preferences.

4. Conclusion

In this paper we presented a first attempt to explain gender differences in health and mortality by health-economic theory. Our approach, based on the notion of health deficit accumulation,

is suitable to be calibrated with real data. We proposed a calibration of male and female Reference Americans, according to which gender differences in health and mortality are explained by gender-specific physiology (rate of aging, terminal deficits at death) and gender-specific preferences for health and consumption smoothing. We utilized the calibrated model in order to estimate the contribution of preferences and health behavior on the observed gender gap in longevity. We found that preferences for health and health investment can motivate about 70% of the gender gap. Extending the model with gender-specific preferences for unhealthy consumption, our counterfactual experiments suggest that up to 88% of the gender gap can be motivated by gender-specific preferences.

Here we followed the tradition of mainstream health economics and considered health behavior in the context of optimal life-cycle decisions of aging individuals. An interesting future project is to consider the consumption and health spending decisions of aging couples. Such an approach could investigate the impact of altruism and bargaining power on health outcomes and longevity of husband and wife, health care provided by the spouses, and the effect of the death of a partner on individual health and longevity. While such an extension of the theory will certainly add more complexity and computational challenges, we are confident that there will be again a straightforward calibration with data from gerontology and minimal degrees of freedom. Our confidence is based on the theory's foundation on health deficit accumulation, a concept of measuring health and aging which is widely and increasingly used in the natural and medical sciences.

Appendix: Calibration Method

We briefly explain our calibration strategy for estimating the remaining preference parameters and biological parameters. For the benchmark model we have to estimate gender-specific values for the preference parameters α and σ, and for medical health technology A. We use these parameters to fit life expectancy of males and females, and total personal health care per-capita spending by gender and age group (see main text for data references).

The problem we encounter is that we cannot solve the model analytically, which makes it impossible to determine the impact of parameter values on the model's fit to the data analytically. To circumvent this problem we adapt the Method of Simulated Moments from the econometric literature (see McFadden 1989) to our calibration procedure. The idea is, analogous to the MSM, to fit the simulated model's response to the data, instead of the analytically calculated model's response.

In detail our procedure works as follows. We start with a "reasonable" set of parameter values and determine the optimal lifetime trajectories for males and females numerically. We then calculate the deviation of simulated life expectancy from the data, and the deviation of gender specific lifetime trajectories of health spending from the data, aggregated in 20 year intervals. Since, altogether, we have more data points than parameters, the problem is overdetermined. Hence, we use a standard MATLAB procedure to minimize the sum of the squares of the residuals. In each iteration the procedure modifies the parameter values in order to improve the model's fit to the data, and it terminates when a parameter set is found such that the deviation between simulation results and the data has reached its minimum.

For the extended model we apply the same method to a larger number of parameters and data points. In particular, we extend the estimated parameters by gender specific preference parameters (β, ψ) and a gender specific biological parameter (B), both related to unhealthy consumption. We augment the data by data on the price elasticity of cigarette demand, the gender specific shares of cigarette consumption on total consumption, and the average reduction in life expectancy caused by smoking (for references see main text). Finally, we determine parameter values that minimize the extended residuum.

References

ALA (2011). Trends in Tobacco Use, American Lung Association, Research and Program Services, Epidemiology and Statistics Unit

Almond, D., and Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives* 25(3), 153-72.

Arber, S., and Cooper, H. (1999). Gender differences in health in later life: the new paradox? *Social Science & Medicine* 48(1), 61-76.

Austad, S.N. (2006). Why women live longer than men: sex differences in longevity. *Gender Medicine* 3(2), 79-92.

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

Barford, A., Dorling, D., Smith, G.D., and Shaw, M. (2006). Life expectancy: women now on top everywhere. *British Medical Journal* 332(7545), 808.

Beckett, A. H., Gorrod, J. W., and Jenner, P. (1971). The effect of smoking on nicotine metabolism in vivo in man. *Journal of Pharmacy and Pharmacology* 23(S1), 62S-67S.

Benowitz, N. L. (1984). Daily intake of nicotine during cigarette smoking. *Clinical Pharmacology & Therapeutics* 35(4), 499-504.

Bird, C.E., and Rieker, P.P. (1999). Gender matters: an integrated model for understanding men's and women's health. *Social Science & Medicine* 48(6), 745-755.

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

Browning, M., and Ejrns, M. (2009). Consumption and children. The Review of Economics and Statistics 91(1), 93-111.

Case, A., and Deaton, A.S. (2005). Broken down by work and sex: How our health declines. In: *Analyses in the Economics of Aging*, University of Chicago Press, 185-212.

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

Chaloupka, F.J. and Warner, K.E. (2000). The economics of smoking. In: *Handbook of Health Economics* Vol. 1, Elsevier, Amsterdam, 1539-1627.

Chetty, R. (2006). A new method of estimating risk aversion. *American Economic Review* 96, 1821-1834.

Chetty, R., Stepner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., ... and Cutler, D. (2016). The association between income and life expectancy in the United States, 2001-2014. *Journal of the American Medical Association* 315(16), 1750-1766.

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

CDCP (2015) Centers for Disease Control and Prevention. Current Cigarette Smoking Among AdultsUnited States, 20052014. Morbidity and Mortality Weekly Report 2015; 64(44):123340.

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

Cullen, M.R., Baiocchi, M., Eggleston, K., Loftus, P., and Fuchs, V. (2015). The Weaker Sex? Vulnerable Men, Resilient Women, and Variations in Sex Differences in Mortality since 1900. NBER Working Paper No. w21114.

Dalgaard, C-J. and Strulik, H. (2012). The genesis of the golden age: Accounting for the rise in health and leisure. Working Paper, University of Copenhagen.

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

Dalgard, C.-J., Strulik, H. (2015). The Economics of Health Demand and Human Aging: Health Capital vs. Health Deficits, Discussion Paper, University of Goettingen.

Ehrlich, I., and Chuma, H. (1990). A model of the demand for longevity and the value of life extension. *Journal of Political Economy* 98(4), 761-82.

Felder, S. (2006). The gender longevity gap: explaining the difference between singles and couples. *Journal of Population Economics* 19(3), 543-557.

Finkelstein, A., Luttmer, E. F., and Notowidigdo, M. J. (2009). Approaches to Estimating the Health State Dependence of the Utility Function. *The American Economic Review* 99(2), 116-121.

Finkelstein, A., Luttmer, E.F., and Notowidigdo, M.J. (2013). What good is wealth without health? The effect of health on the marginal utility of consumption. *Journal of the European Economic Association* 11(1), 221-258.

Forster, M. (2001). The meaning of death: some simulations of a model of healthy and unhealthy consumption. *Journal of Health Economics* 20(4), 613-638.

Fries, J.F. (1980). Aging, natural death, and the compression of morbidity. *New England Journal of Medicine* 303(3), 130-135.

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

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy* 80, 223-255.

Grossman, M. (2000). The human capital model. *Handbook of Health Economics*, Vol. 1, 347-408.

Grossmann, V. and Strulik, H. (2015). Optimal Social Insurance and Health Inequality, CESifo WP No. 5604.

Hall, R.E. and Jones, C.I. (2007). The value of life and the rise in health spending. *Quarterly Journal of Economics* 122, 39–72.

Harttgen, K., Kowal, P., Strulik, H., Chatterji, S., Vollmer, S. (2013). Patterns of frailty in older adults: comparing results from higher and lower income countries using the Survey of Health, Ageing and Retirement in Europe (SHARE) and the Study on Global AGEing and Adult Health (SAGE). *PLOS One* 8(10), e75847.

Holden, C. (1987). Why do women live longer than men? *Science* 238(4824), 158-160.

HDR (2013). United nations development program. Human Development Report.

Jones, C.I., and Williams, J.C. (2000). Too much of a good thing? The economics of investment in R&D. *Journal of Economic Growth* 5(1), 65-85.

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.

Layard, R., Mayraz, G., and Nickell, S. (2008). The marginal utility of income. *Journal of Public Economics* 92, 1846-1857.

Lise, J., and Seitz, S. (2011). Consumption inequality and intra-household allocations. *Review of Economic Studies* 78(1), 328-355.

Luy, M. (2003). Causes of male excess mortality: insights from cloistered populations. *Population and Development Review* 29(4), 647-676.

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

Macintyre, S., Ford, G., and Hunt, K. (1999). Do women over-report morbidity? Men's and women's responses to structured prompting on a standard question on long standing illness. *Social Science & Medicine* 48(1), 89-98.

McFadden, D. (1989). A Method of Simulated Moments for Estimation of Discrete Response Models without Numerical Integration. *Econometrica* 57(5), 995-1026

Mehra, R. Prescott, E.C. (1985) The equity premium: A puzzle. *Journal of Monetary Economics* 15, 145-161.

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.jsp?component=1&subcomponent=0&year=-1&tableSeries=1&searchText=&searchMethod=1&Action=Search`)

Mitnitsiki, A., Bao, L., and Rockwood, K. (2006). Going from bad to worse: a stochastic model of transitions in deficit accumulation, in relation to mortality, *Mechanisms of ageing and development* 127, 490–493.

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

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002b). The mortality rate as a function of accumulated deficits in a frailty index. *Mechanisms of ageing and development* 123, 1457-1460.

Mitnitski, A., Song, X., Skoog, I., Broe, G.A., Cox, J.L., Grunfeld, E. and Rockwood, K. (2005). Relative fitness and frailty of elderly men and women in developed countries and their relationship with mortality. *Journal of the American Geriatrics Society* 53, 2184–2189.

Mitnitski, A., Lang, I.A., Rockwood, K. (2011). Measuring aging in individuals and populations: A deficit accumulation approach, Lepas Working Papers

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

NVSS (1980). National Center for Health Statistics: Vital Statistics of the United States, 7980, Vol. 11, Sec. 6, Life Tables. DHHS Pub. No. (PHS) 84-1104. Public Health Service, Washington.

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

Oksuzyan, A., Juel, K., Vaupel, J.W., and Christensen, K. (2008). Men: good health and high mortality. Sex differences in health and aging. *Aging Clinical and Experimental Research* 20(2), 91-102.

Pijoan-Mas, J. and Rios-Rull, J.-V. (2014). Heterogeneity in Expected Longevities. *Demography* 51, 2075-2102.

Preston, S.H., and Wang, H. (2006). Sex mortality differences in the United States: The role of cohort smoking patterns. *Demography* 43(4), 631-646.

Preston, S.H., D.A. Glei, and J.R. Wilmoth (2010), Contribution of smoking to international differences in life expectancy, in: E.M. Crimmins, S.H. Preston, and B. Cohen (eds.), *International Differences in Mortality at Older Ages: Dimensions and Sources*, Washington, D.C.: National Academies Press, pp. 105-131.

Rockwood, K., and Mitnitski, A. (2007). Frailty in relation to the accumulation of deficits. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 62(7), 722-727.

Robson, A.J. and Kaplan, H.S. (2007). Why do we die? Economics, biology, and aging, *American Economic Review* 97, 492-495.

Schünemann, J., Strulik, H., Trimborn, T. (2015). Going from Bad to Worse: Adaptation to Poor Health, Health Spending, Longevity, and the Value of Life, Cege Discussion paper 268.

Sindelar, J.L. (1982). Differential Use of Medical Care by Sex. *Journal of Political Economy* 90(5), 1003-19.

Skirbekk, V. (2004). Age and individual productivity: A literature survey. *Vienna Yearbook of Population Research*, 133-153.

Sorensen, G. and Pechacek, T. F. (1987). Attitudes toward smoking cessation among men and women. *Journal of Behavioral Medicine* 10(2), 129-137.

Strulik, H. (2013). Health and education: Understanding the gradient. Discussion Paper, University of Goettingen.

Strulik, H. (2015a). A closed-form solution for the health capital model. *Journal of Demographic Economics* 81(3), 301-316.

Strulik, H. (2015b). Frailty, mortality, and the demand for medical care, *Journal of the Economics of Ageing* 6, 5-12.

Strulik, H. (2015c). The Health Hump, Discussion Paper, University of Goettingen.

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

Sunden, A. E. and Surette, B.J. (1998). Gender differences in the allocation of assets in retirement savings plans. *The American Economic Review* 88(2), 207211.

Trimborn, T., Koch, K.-J. and Steger, T.M. (2008). Multidimensional transitional dynamics: A simple numerical procedure. *Macroeconomic Dynamics* 12, 301319.

Verbrugge, L.M. (1988). A life and death paradox. *American Demographics* 11, 34-37.

Verbrugge, L.M. (1985). Gender and health: an update on hypotheses and evidence. *Journal of Health and Social Behavior* 26, 156-182.

Wagstaff, A. (1986). The demand for health: some new empirical evidence. *Journal of Health Economics* 5 195-233.

Waldron, I. (1993). Recent trends in sex mortality ratios for adults in developed countries. *Social Science & Medicine* 36(4), 451-462.

Wardle, J., Haase, A.M., Steptoe, A., Nillapun, M., Jonwutiwes, K., and Bellisie, F. (2004). Gender differences in food choice: the contribution of health beliefs and dieting. *Annals of Behavioral Medicine* 27(2), 107-116.

World Life Expectancy (2015). `http://www.worldlifeexpectancy.com/history-of-life-expectancy`

Zang, E. A., and Wynder, E. L. (1996). Differences in lung cancer risk between men and women: examination of the evidence. *Journal of the National Cancer Institute* 88(3-4), 183-192.

Zweifel, P. and Breyer, F. (1997). *Health Economics*, Oxford University Press, Oxford.

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