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"Dying and Dissaving"

Doctoral Student Presentations

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Presenter: Clara Fernström, PhD student at Stockholm School of Economics

Dying and Dissaving

Research plan

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Abstract

Do people change savings behavior when life expectancy is suddenly reduced? Standard life cycle theory suggests that they should. I study the causal effect of life expectancy on economic choices using Swedish register data that allows me to link health shocks to income, wealth, and savings. Using exogenous variation in the exact timing of a health shock I contribute to understanding (1) the empirical importance of life expectency in decision-making and (2) the causal effect of health on wealth.

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1 Introduction

The idea that people make decisions with respect to life expectancy is a key component in many life cycle models. Economists assume that individuals optimize to smooth consumption over expected life time when they choose how much to save, consume, work, or allocate to risky assets. I study how important this component is empirically.

The strong correlation between health and economic outcomes is well established, but the direction of causality is not. Empirical challenges make it hard to say whether it is lack of wealth that causes bad health, or bad health that limits wealth accumulation. Or is it some unobservable characteristic, such as impatience, that determines choices with negative effects on both health and wealth? Using exogenous variation in the exact timing of a health shock I also contribute to understanding how health affects wealth accumulation. The effect of health on economic choices is crucial from a policy perspective. To understand how policy can be improved we need to understand the implications of worsening health status.

The aim is to document how health shocks affect individuals' economic choices and through which mechanisms. In particular, I study savings and how it is affected by changes in life expectancy and the conditional distribution of survival rate.

In this document, I describe the hypotheses and how I plan to test them. Initially, I will split the data set in two and do the analysis only on one part, in the spirit of Fafchamps and Labonnne (2017). I will update this document if necessary, before I do the analysis on the full population.

1.1 Research question

Does life expectancy have a causal effect on individuals' economic choices?

1.2 Empirical approach

I use Swedish register data to study how health shocks affect individuals' savings through changes to survival probability, and thus to life expectancy. I account for associated changes in income and marginal utility of consumption. The institutional setting in Sweden with a comprehensive social insurance system allows me to abstract from expenses related to medical or long-term care. I use two approaches. First, I have an empirical strategy to identify causality using random timing in the health shock. Second, I use a life cycle model to simulate the expected theoretical effect of drawing a bad health shock that influences survival probability, income, and marginal utility of consumption.

2 Hypotheses

What is the theoretical effect of a health shock on savings? A health shock may have implications for life expectancy, income (if the health shock occurs before retirement), and for marginal utility of consumption.

2.1 Survival probability

A health shock that reduces survival probability should lead to a fall in savings (in the absence of a bequest motive). The reason why mortality risk matters for savings is that it increases the effective discount rate; consumption tomorrow becomes less valuable relative to consumption today. The individual increases consumption and dissaves. In fact, the full distribution of conditional survival may matter for the current saving decision.

- Survival probability $\downarrow \Rightarrow$ Savings $\downarrow$

2.2 Income

The direction of the effect on income depends on timing. If the shock is associated with an immediate fall in income (due to inability to work), there is reason to draw on savings. But if the individual expects income to fall even more in the future, there could be reason to increase current savings.

- Current income $\downarrow \Rightarrow$ Savings $\downarrow$
- Future income $\downarrow \Rightarrow$ Savings $\uparrow$

2.3 Marginal utility of consumption

Marginal utility of consumption may depend on health state. There is evidence that marginal utility of *non-medical* consumption decreases with worsening health (e.g. Finkelstein et al. 2013, Viscusi and Evans 1990). Consumption becomes less enjoyable or physically impossible. Again, the direction of the effect depends on timing. If current marginal utility falls, consumption drops and savings increase. Whereas if the individual expected future marginal utility to go down, current consumption increases and savings fall.

- Current marginal utility $\downarrow \Rightarrow$ Savings $\uparrow$
- Future marginal utility $\downarrow \Rightarrow$ Savings $\downarrow$

2.4 Heterogeneity with respect to family composition

These effects may vary with family composition. For example, the effect of reduced survival probability may vary with the presence and strength of a bequest motive. If there is no bequest motive, a reduction in survival probability implies dissaving. If there is a bequest motive, a reduction in survival probability implies a trade-off between dissaving while alive and increasing saving for bequests. Depending on the strength of the bequest motive, a

reduction in survival probability may imply lower or higher savings. Inheritance taxation and life insurance may alter this trade-off. This means that the effect of a reduction in survival probability should vary with family composition as well as with inheritance taxation and life insurance. As a starting point, I assume that the strength of the bequest motive is constant across health states.

2.5 Net effect

To understand the net effect I simulate a life cycle model of precautionary savings with health state dependent utility and stochastic processes determining survival probability, health state, and income. The idea is to model different health shocks with different implications for these dimensions. I base the model on previous work by e.g. Carroll (1997), Deaton (1991), Domeij and Johannesson (2006), French (2005), De Nardi (2004), De Nardi et al. (2009), and De Nardi et al. (2016), and use parameters as close to the literature as possible.

3 Data and variables

In the reduced-form analysis I use register data with the objective to study the empirical counterparts of the main components in the standard life cycle model. I match individual health data with economic and demographic data for the full Swedish population from National Board of Health and Welfare, Statistics Sweden, and Swedish Twin Registry. In this panel I can follow health events as well as economic outcomes and behaviors over time. With family connections from the Multigeneration Registry I can also trace type of relationship between individuals.

3.1 Outcome variable

The main outcome of interest is savings behavior. The idea is that all type of income is either consumed or saved, both in the model and in the data.

Primarily, I want to capture *active* savings decisions. Savings is defined as the annual changes in different assets and removing any change that is due to price changes during the year. This is possible since I have data on detailed asset holdings from the Wealth Registry 1999–2007, matched to the annual return of each asset. I follow previous literature including Calvet, Campbell, and Sodini (2007); Calvet, Campbell, and Sodini (2009); Calvet and Sodini (2014); Koijen, Van Nieuwerburgh, and Vestman (2015); and Bach, Calvet, and Sodini (2017). The main outcome variable is defined as follows:

$$\begin{aligned} Savings_{it} = {} & \Delta Bank\,account_{it} + \Delta Risky\,assets_{it} + \Delta Housing_{it} \\ & + \Delta Capital\,insurance_{it} + Private\,pension\,contribution_{it}. \end{aligned}$$

Individuals may also target a specific savings to income ratio, or wealth level each year. Therefore I can check savings to income ratio (even though this may be inflated by lower income), financial wealth and total wealth (or their logs) in the robustness analysis. Additionally, I follow the literature and use car purchases as a proxy for consumption.

3.2 Explanatory variables

To test the hypotheses in Section 2 empirically I define measures corresponding to each theoretical mechanism. I use a data driven approach and categorize diagnoses in these dimensions. This allows me to compare different diagnoses with different implications. For example, I can compare one diagnosis with a large effect on survival probability and a small effect on income, with another diagnosis with a small effect on survival probability and large effect on marginal utility. Thus, I can compare fatal and chronic diseases. This approach should give me sufficient variation in the empirical analysis.

I define diagnosis-level characteristics by age, gender, and diagnosis, and assign these to diagnosed individuals. Healthy individuals who are not yet diagnosed are assigned survival probabilities based on life tables or population averages for their age and gender group. For individuals with multiple diagnoses I group those with the same set of diagnoses. As long as these groups are large enough, the contribution of any single individual should not be driving the average. For smaller groups, I exclude the affected individual when calculating the average assigned to her. In case the contributing diagnoses to health complications are hard to pin down from the Patient Registry, I define the main and contributing causes of death from the Cause of Death Registry, and then infer timing of diagnosis from Patient Registry. Additional diagnosis details are available in the National Quality Registries by type of diagnosis. The aim is to consistently define the point in time when the individual learnt about her new survival probability, etc.

The main explanatory variable of interest is survival probability. It is defined as the share of individuals that survive from one year to the next, with the same age, gender, diagnosis, and year of diagnosis.

$$Survival\,probability_t = \frac{\text{Number of survivors in } t+1}{\text{Number of individuals diagnosed in year } t}.$$

Given the panel dimension of the data it is also possible to use the full distribution of conditional survival. In particular, I estimate the historical distribution at different points in time to understand the time trend in parameters, and possibly extrapolate into the future. The idea is that a patient receives information from her physician on the survival prospects, potentially incorporating recent advances in medical treatments.

I also categorize diagnoses in terms of income and marginal utility as potential mechanisms. I call these other diagnosis characteristics $\boldsymbol{Z_{it}}$. Timing of current and expected future changes to income and marginal utility matters. Again, these are averages of all individuals

of the same age, gender, and diagnosis.

To measure the current income change, I use the ratio of income in the year of diagnosis to income in the pre-diagnosis year. I can include labor and capital income, as well as any social transfers. To measure the expectation of future income, I use the ratio of average income in the three years after diagnosis to income in the pre-diagnosis year:

$$Z_{Current\,income,\,t} = \frac{\text{Income}_t}{\text{Income}_{t-1}},$$

$$Z_{Future\,income,\,t} = \frac{\text{Average income}_{t+1\rightarrow t+3}}{\text{Income}_{t-1}}.$$

The choice of using an average over three years is somewhat arbitrary; the meaningful average essentially depends on the average life expectancy of the given diagnosis.

To measure marginal utility of consumption I use a measure of disease severity as a proxy. I create an index of hospitalization spells, treatments, and drug prescriptions. I have one measure of current marginal utility (severity), and one measure of expected marginal utility (severity) in the subsequent years:

$$Z_{Current\,MU,\,t} = \text{Severity index }_t,$$

$$Z_{Future\,MU,\,t} = \text{Average severity index }_{t+1\rightarrow t+3}.$$

These proxies for marginal utility capture some dimensions that might limit the utility of

consumption. As a complement, I include other measures that capture additional dimensions. First, I link diagnoses to subjective limitations on daily life activities and subjective health perception, based on survey data from the Swedish Twin Registry (EQ5D). This gives a mapping of a subjective health measure to different diagnoses, i.e. how a certain diagnosis affects the individuals' normal life. Second, similar to Cesarini et al. (2016), I calculate the Charlson comorbidity index. This overlaps somewhat with severity index defined above, but is a common measure of severity in the literature. Third, one possible reason why marginal utility might fall is a depression following the news of the diagnosis and reduction in survival probability. I define at diagnosis level the share of individuals that are also diagnosed with depression and/or prescribed antidepressant drugs as a control variable. At the individual level I can capture the observed cases of diagnosed depression and/or prescribed antidepressant drugs. Finally, the National Quality Registries and the Cancer Registries provide additional details on the diagnoses that can service as diagnosis-specific measures of severity.

4 Empirical strategy

In the first part of the analysis, I study how these diagnosis-level characteristics affect individuals' savings. By adding controls and fixed effects, I come closer to causal estimates. In the second part, I use quasi-experimental difference-in-differences estimation as the key strategy to identify the causal effect of survival probability on savings.

4.1 Fixed effects

The first step is to describe the effect of survival probability on savings, holding income and severity measures $\boldsymbol{Z_{it}}$ fixed. To understand the relative effect I also use the natural logaritm of savings. Next, to alleviate concerns about selection into bad health due to socioeconomic

factors, I add controls and fixed effects. I do this stepwise to understand what is the driving variation. The baseline specification:

$$\begin{aligned} Savings_{it} = \beta\, Survival\, probability_{it} + \boldsymbol{\gamma}\, \boldsymbol{Z_{it}} \\ + \alpha_i + \boldsymbol{\delta}\, \boldsymbol{X_{it}} + year_t + diagnosis_d + \varepsilon_{it}. \end{aligned} \tag{1}$$

Following the hypotheses in Section 2, I expect the following signs on the estimated parameters (in absence of a bequest motive):

- $\beta_{Survival\, probability} > 0$,
- $\gamma_{Current\, income} > 0$,
- $\gamma_{Future\, income} < 0$,
- $\gamma_{Current\, MU} < 0$,
- $\gamma_{Future\, MU} > 0$.

I control for pre-determined as well as time-varying individual characteristics $\boldsymbol{X_{it}}$ including gender, age, education/human capital, family composition, region of residence/birth, health history (including birth outcomes in the Birth Registry since 1973), family health history (including data from Cancer Registry since 1958), employment status, immigrant status, permanent/transitory income risks, habits, and previous financial (including leverage and portfolio risk) and default behaviors. Additionally, at diagnosis level, the new information on survival probability could be more or less certain. The variance of life expectancy could vary between diagnoses and this new source of uncertainty could also affect the savings decision and could be included in the set of control variables.

I include individual fixed effects to control for any individual-specific selection. The identifying variation is then within individuals over time. The key identifying assumption is that there are no time-varying, unobserved factors correlated with health and savings at the individual level, e.g. financial troubles that are bad both for health and savings, or early disease

symptoms. To test whether some diagnoses are systematically preceded by financial troubles I can map available data from Swedish Enforcement Authority (Kronofogden) to diagnoses. In case of perceived symptoms, it is problematic if the individual actually has internalized a lower survival probability before being diagnosed, and acted accordingly. However, it is unlikely that individuals take far-reaching financial decisions before any condition is confirmed by expertise.

For a smaller sample, I can include twin fixed effects to control for all variation that is common to a twin pair, such as genes, and common environment. The identifying variation is then the difference in survival probability between twins within the same pair.

Year fixed effects help alleviate concerns about market and business cycle factors potentially affecting both savings and health. By including diagnosis fixed effects I use only variation in age and gender, i.e. life cycle factors. This specification is thus not possible together with individual fixed effects or age and gender controls since this is the only variation left. However, diagnosis times year fixed effects controls for any business cycle variation in various diseases.

4.2 Quasi-experimental difference-in-differences estimation

The main specification for identifying causality is a difference-in-differences set-up based on timing of diagnosis. I compare individuals who are diagnosed in year τ to individuals with the same diagnosis Δ years later. The identifying variation is the difference between those diagnosed, and those who will be diagnosed a few years later. The key identifying assumption is that timing of the diagnosis is as good as random, at least within a small window Δ. Similar identification strategies are used by Fadlon and Nielsen (2017) and Martinello and Druedahl (2017).

In the following specification, $Treat$ is an indicator of belonging to the treated (diagnosed) group or not:

$$
\begin{aligned}
Savings_{it} = \tilde{\beta}\, & Treat_i \times Post_t \\
& + \phi\, Post_t \\
& + \alpha_i + \boldsymbol{\delta}\, \boldsymbol{X_{it}} + year_t + diagnosis_d + \varepsilon_{it}.
\end{aligned} \tag{2}
$$

For individuals diagnosed in year τ, $Treat = 1$. For individuals diagnosed with the same diagnosis in year $\tau + \Delta$, $Treat = 0$. The main coefficient of interest is $\tilde{\beta}$ and is the differential effect of a diagnosis on savings, relative to a comparable control group.

The choice of Δ involves a trade-off between comparability of treated and control groups and the desire to follow the outcome for a period that is as long as possible. For example, Fadlon and Nielsen (2017) set $\Delta = 5$, but they also discuss the possibility of using a "rolling window" to increase power. I test parallel trends for the treated and control groups.

Next step in the analysis is to isolate the effect of survival probability on savings in this quasi-experimental set-up. Thus, I split the difference-in-difference estimator $\tilde{\beta}$ in (2) into the respective effect of survival probability and other diagnosis-characteristics $\boldsymbol{Z_{it}}$.

$$
\tilde{\beta} = \tilde{\beta}_0 + \tilde{\beta}_1\, Survival\, probability_{it} + \tilde{\boldsymbol{\beta}}_{\mathbf{2}}\, \boldsymbol{Z_{it}} \tag{3}
$$

Thereby I can identify the differential effect of survival probability on savings ($\tilde{\beta}_1$) in the following specification:

$$
\begin{aligned}
Savings_{it} = \tilde{\beta}_1\, & Survival\, probability_{it} \times Treat_i \times Post_t \\
& + \tilde{\boldsymbol{\beta}}_{\mathbf{2}}\, \boldsymbol{Z_{it}} \times Treat_i \times Post_t \\
& + \tilde{\beta}_0\ \times Treat_i \times Post_t \\
& + \phi\, Post_t \\
& + \alpha_i + \boldsymbol{\delta}\, \boldsymbol{X_{it}} + year_t + diagnosis_d + \varepsilon_{it}.
\end{aligned} \tag{4}
$$

4.3 Heterogeneity

There are some dimensions of heterogeneity that I document across the characteristics described above. These include at least the following three. First, the effect depends on how

expected the diagnosis is. This can be measured as health history of the individual and her family, age at diagnosis, type of diagnosis, and whether hospitalization was planned or not. For example, a severe diagnosis may be more expected had the individual had been on sick leave the period before. Moreover, timing of stroke or heart attack is more random than if the affected individual has had symptoms over a long time. The Multigenerational Register allows me to follow the medical history of biological relatives, which should matter more for diagnoses with larger genetic component. Similarly, the Swedish Twin Registry allows me to follow individuals with identical or very similar genetic setup. For these twins I can also match with life style factors (such as tobacco and alcohol consumption), which should matter more for diagnoses with larger life style component. This helps get a good indication of the expectancy of the given disease.

Second, the effects could also vary with family composition as discussed in Section 2.4. For example, individuals with a spouse, children, or other heirs, may have different incentives to save due to bequest motives, compared to single individuals without heirs. A reduction in survival probability implies a trade-off between dissaving while alive and increasing saving for bequests. By splitting the sample across family composition I study how lower survival probability affects these groups differently. Moreover, the trade-off could be altered by inheritance taxation and life insurance. The abolishment of the inheritance tax in Sweden in 2004 provides an opportunity to study this trade-off. Without full coverage of data on life insurance policies, I can impute the existence of a life insurance policy ex ante by following the life insurance pay-out to survivors ex post.

Third, the effect could vary with level of education and other socioeconomic factors given the strong correlation between health and socioeconomic status (see e.g. Lundborg et al. 2015 and Smith 1999). In particular, a given diagnosis may affect individuals with different educational background differently.

4.4 Potential issues

There are at least two reasons why the estimated effect of survival probability on savings, $\beta_{Survival\,probability}$, could be biased.

First, a shock to life expectancy may change the labor-leisure trade-off. If an individual has some savings at the time when she learns her new, lower survival probability, she may want to quit her job. In the data, I observe the drop in income associated with the diagnosis. In the setup described above, such behavior would increase the effect through the productivity/income mechanism whereas it actually should increase the effect through survival probability mechanism. Thus, the estimated $\beta_{Survival\,probability}$ would be biased downward.

Second, I attempt to capture changes in marginal utility of consumption due to worsening health as described above. However, if my measures are insufficient to capture all dimensions of marginal utility, there might be a reduction in consumption (lack of dissaving) that is due to unobserved changes in marginal utility. Such an omitted variable, i.e. "true marginal utility" could lead to a biased estimate of $\beta_{Survival\,probability}$. The direction of the bias depends on the sign of the correlation of the omitted variable and the dependent variable, and *Survival probability* respectively. Current marginal utility and *Savings* are negatively correlated (following the hypothesis in Section 2). Current marginal utility should be positively correlated with *Survival probability*; the lower survival probability, the larger the risk of a condition (apathy or depression) in which the marginal utility of consumption is lower. Thereby, the resulting omitted variable bias would be negative, i.e the estimated $\beta_{Survival\,probability}$ would be biased downward also for this reason.

5 Extensions

I will expand the analysis in this paper in several directions. First, I will study the effect at *household* level rather than *individual* level, to understand the importance of the unitary

household model. A health shock to one spouse may also alter the power balance in intra-household bargaining process. Second, there are also implications of changes in survival probability on other financial behaviors apart from savings, such as portfolio choice, housing, debt, annuities demand, and so-called investment biases. Third, the fact that the individual's labor-leisure trade-off might change due to changes in survival probability there is also reason to study the effect on labor supply and retirement dynamics of the affected individual and her spouse. Finally, as discussed in Section 4.3, individuals may rationally anticipate a health shock. They might also irrationally anticipate a health shock. There is evidence that individuals' personal experiences influence their decision-making (e.g. Balasubramaniam 2017). For example, if a non-biological relative or colleague receives a diagnosis with negative implications for survival probability, the individual may perceive her own survival probability as reduced, and act accordingly. More empirical work on such social/network (rather than genetic) channels are needed.

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