# Essays on the Effects of Shocks in Childhood and Later Life on Health

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*To Anna, Magdalena, and Valentin*

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# Contents

# Chapter 1

## Introduction

A healthy population is the engine for economic growth in modern societies (World Health Organization (2002)). It has been shown that healthy individuals have a better educational attainment, a better labor market performance, are wealthier and happier (see for instance Smith (1999), Frijters *et al.* (2011), Smith (2009a), Case and Paxson (2010)). Whether people are healthy or not, is determined by single and combined factors that affect individuals over the whole life course. There exists a large literature that examines the consequences of adverse circumstances early in life on health. These studies have documented that children exposed to health shocks and adverse life conditions such as undernutrition or poor parental socioeconomic status fall into poorer health as they age (Barker (1992), Case *et al.* (2005), Neelsen and Stratman (2011)). However, negative life events that occur later in life are also associated with worse physical and psychological health status of individuals. Unemployment and wealth shocks, a personal injury, or the death of a family member negatively affect different dimensions of an individual's health, well-being and mortality (Frijters *et al.* (2011), Espinosa and Evans (2008), Van den Berg *et al.* (2011)).

Facing an aging population, policy makers have recently recognized the importance of a healthy population as a mechanism for stimulating and protecting economic growth, and reducing poverty (World Health Organization (2002)). Managing and financing health care costs associated with these health investments are challenging and important issues of the today's political agenda of high-income countries in Europe and the US. Public health care spending has significantly risen in the last decades, and will further increase. A key factor is the demographic change: The first wave of baby boomers turned 65 in 2010, bringing an additional boost into health care spending (Olhansky *et al.* (2009)). Thus, comprehensive reforms of the European and US-American health systems are required. Addressing these problems, policy makers need to understand the causes and mechanisms that involve poor health in the elderly population in order to assess the costs of poor health and, eventually, guarantee a stable economic growth.

This dissertation provides insights into causes and consequences of several early and late life shocks on different dimensions of later life health. The second chapter of this dissertation focuses on the effects of the partner's death on the surviving spouse's mental health among the elderly US-population. Chapter three deals with the consequences of World War II as an early life shock on late life health and socioeconomic outcomes in Western continental Europe. Chapter four examines the effect of the

hunger episodes after World War II in Germany on late life health and identifies food consumption behavior as one possible pathway of this link. In all three chapters I focus on the population aged 50+. An important aspect of my thesis research is the analysis of large data sets based on longitudinal surveys that are linked with external historic, administrative or process data. These data typically contain very strategic and exhaustive information due to continuity, completeness, and representation of small area statistics. Since they are the result of a registration process and used for official statistics, they can provide an excellent enrichment of survey data. In my dissertation, these data linkages thus are the basis for the identification of causal effects and the characterization of channels through which such life shocks work.

The second chapter of my dissertation, the essay *"The Effects of Bereavement on Mental Health - An Analysis of Anticipation Patterns Surrounding the Death of a Partner"* examines the effect of bereavement on the surviving partner's mental health taking account of the anticipation of the partner's death. The partner's death is characterized as one of the most stressful events in life affecting many dimensions of psychological and physical health (Martikainen and Valkonen (1996), Espinosa and Evans (2008), Van den Berg *et al.* (2011)). The identification of a causal bereavement effect requires that any health outcomes of the surviving partner are only affected after the death of their spouse, but not before. However, individuals might anticipate the partner's future death leading to a change in mental health outcomes prior to the actual death event. If this is the case, the partner's death is considered to be endogenous. As a consequence, the detrimental effects of bereavement on mental health have been underestimated.

To study the effect of bereavement, I use data from seven waves of the Health and Retirement Study (HRS) and supplemental Exit interview data. I link this information to the non public National Death Index (NDI) file on causes and dates of the partner's death, and to exact interview dates. I test the assumption of an exogenous death event by using information on whether the time of the partner's death was expected or not. I exploit the differences in the number of days between the interview and the death date in order to determine the onset and extent of anticipation and adaptation associated with bereavement. Moreover, I use information on the duration of final illness and official causes of death allowing me to separate the effects of bereavement from those of the partner's illness on mental health. Parsing out this caregiver effect reveals an exact picture of the effect of bereavement on mental health.

I find strong anticipation effects of future bereavement on mental health if the partner died expectedly, starting about 18 months before the partner's death. Instantaneous bereavement effects are large for the expected time group, but significantly stronger for an unexpected time of death. The process of adapting to an initial level of mental health after the death event is similar for both groups, with higher probability levels of depression up to one year after the partner's unexpected death. A more detailed analysis of the bereavement effect suggests that a part of these changes in mental health can be attributed to a caregiver burden effect.

The findings of this study reflect that there is an underestimation of the bereavement effect in terms of mental health. The high prevalence of depression I find for an unexpected time of death implies an additional risk of becoming poor for widowed individuals who already have a high level of exposure to poverty (McGarry and Schoeni (2005)). Therefore, mourning support and intervention programs might attenuate the most adverse effects of bereavement and help to control public health care expenditures.

In chapter three of my dissertation, *"The Effects of World War II on Economic and Health Outcomes across Europe"*, my co-authors and I focus on the World War II as an adverse life event investigating its long-run effects on late-life economic outcomes, specifically health, education, labor market outcomes and marriage, in Western continental Europe. We explore several channels through which this war might have influenced individual lives, and document which groups of the population were most affected.

Our research relies on a retrospective survey as part of European Survey of Health, Aging, and Retirement in Europe (SHARE) in 2009 that have recently become available. SHARE consists of 20,000 observations that cover representative samples of the population ages 50 and over in 13 European countries. SHARE measures major contemporaneous economic and health outcomes of adults over age 50 in these European countries and SHARELIFE additionally includes retrospective modules meant to capture salient parts of early life experiences, including those related to the war. For us, information on exposure to hunger, dispossession, persecution and moving histories is of greatest interest. We also collect external data on casualties, timing and location of combat action, yearly GDP by country, population movements, and male-female population ratios (Ellis (1994)). To our individual-level analysis of the multidimensional effects of a major shock that affected life circumstances, we add new dimensions to a rapidly increasing literature aimed at explaining the causes of health and wealth gradients in labor and health economics (see Deaton (2007), Smith (2009a), Heckman (2012)).

Our analysis shows that experiencing war increases the probability of suffering from diabetes, depression and to a lesser degree from heart disease. Moreover, individuals exposed to war or combats have significantly lower self-rated health as adults. Experiencing war is moreover associated with lower years of education, and life satisfaction, and it decreases the probability for women of ever being married, while increasing this probability for men. We find strong effects along the explored pathways of wartime effects, specifically for hunger, dispossession, persecution, childhood immunizations and having an absent father. While a war of the magnitude of World War II affected all social classes to some degree, our work suggests that the worst effects were on the middle class with the lower class right below them in size of impact.

This study highlights advantages of having life-histories in prospective studies such as SHARE. Population-based economic panels are relatively recent, but combining them with life-histories covering salient past personal and macro events opens up many new research opportunities of which World

War II is only one illustration. This is especially the case in Western Europe where the political and economic history of the past four decades is particularly rich and varied.

In chapter four, *"Individual Behavior as Pathway Between Early Life Shocks and Adult Health – Evidence from Hunger Episodes in Post World War II Germany"*, my co-authors and I investigate the long-run effects of episodes of hunger experienced as a child on health status and behavioral outcomes in later life. We argue that an early-life event - in our case a prolonged experience of severe hunger during childhood - is not only an immediate negative biological shock to the stock of health but also affects subsequent behaviors that will impact health at older ages. A one-time shock changes the entire future path of health investments over time and thus levels of health later in life. We show that individual behavior is a pathway between early life shocks and adult health.

Our research relies again on retrospective life data from the European Survey of Health, Aging, and Retirement in Europe (SHARE). We concentrate on Germany that was among those countries most affected by World War II, but also by hunger (Kesternich *et al.* (2012)). One key aspect of this study is the collection of official caloric rations in Germany between 1930 and 1957. These monthly rations for each occupation zone provide a reliable measure of food supply available per capita.

We find a behavioral mechanism of how a childhood shock translates into adult health by showing that early-life hunger experiences also predict food consumption later in life. Engel curves for food consumption are significantly different for individuals who suffered from hunger and those who did not. This effect is strongest for low-income individuals suggesting that poorer individuals are more at risk of ever becoming hungry again and thus eat more as a type of precautionary measure. Our results moreover suggest that having suffered from hunger during childhood increases BMI, the probability of having overweight and diabetes. The hunger effects are again significantly stronger for low levels of gross household income. The coefficient for hunger bundles all possible pathways through which hunger exposure affects late life health outcomes. This includes biological links and non-biological or behavioral mechanisms (see Hamelin *et al.* (1999), Cameron and Demerath (2002)). Linking the experience of hunger, the behavioral path and health outcomes in an simultaneous equation framework, we find that late life outcomes are not solely affected by the experience of hunger during childhood through any biological or not further identified mechanisms, but also through a change in food consumption behavior.

Our study contributes to the recent literature by showing that in particular age 11 to 16 is a sensitive period in childhood suggesting that policy interventions focusing on this group of children might be particularly useful. More importantly, our findings indicate that preferences are not only influenced by preferences of parents or role models as in Dohmen *et al.* (2011), but also by large shocks experienced during childhood and youth. This result strengthens the case for interventions in early childhood and adolescence, as summarized by Heckman (2012).

# Chapter 2

## The Effects of Bereavement on Mental Health
## An Analysis of Anticipation Patterns Surrounding the Death of the Spouse

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### 2.1 Introduction

The death of a spouse is characterized as one of the most stressful events in life, affecting many dimensions of individual health and well-being. Recently widowed individuals show a higher occurrence of immediate physical and psychological health risks, and the death of a spouse is associated with increased mortality and a higher probability of various chronic physical as well as psychological diseases (Martikainen and Valkonen (1996), Van den Berg *et al.* (2007), Espinosa and Evans (2008) and Van den Berg *et al.* (2011)). The consequences of bereavement vary widely, depending on the personal characteristics of the deceased and the bereaved, and the circumstances of death such as its cause (Lee and Carr (2007)). The observed association between bereavement and health might work through various channels, such as the loss of social support, psychological stress of caregiving, nutritional deficits, but also the loss of welfare and a greater poverty risk (see Martikainen and Valkonen (1996), Rosenbloom and Whittington (1993), McGarry and Schoeni (2005)).

While there are several studies that document the association of spousal bereavement with health and other outcomes, the identification of the causal effects of bereavement is challenging. In particular, a causal effect would imply that health outcomes of the survivor are only affected after the spouse's death, but not before. Most of the studies on bereavement assume that the time of the spouse's death is not anticipated, so that the death event is exogenous in econometric models. However, this identifying assumption fails if the time of the partner's demise is anticipated, at least to the extent that behavioral or health adjustments begin before the partner dies. Thus, neglecting the potential anticipation of the death event causes endogeneity, which typically implies that any detrimental effects of bereavement on physical and psychological health are underestimated.

In this paper, we study the effects of conjugal bereavement on mental health taking account of potential anticipation and adaptation effects. Using survey information on whether the time of the partner's death was expected or not, we test the assumption that the death event is exogenous. On the methodological side, an important innovation of our study is the analysis of linked survey and administrative data. We exploit official death records and exact interview dates to determine the date of death on a monthly basis rather than only by one year or longer, which allows for a much

sharper analysis of anticipation and adaptation effects. Moreover, we use information on the duration of final illness and the official cause of death, which allows us to separate the mental health effects of bereavement from those of the partner's illness. The latter is sometimes referred to as the "caregiver burden" effect (Christakis and Allison (2006)), and we use this term here as well. Our study is the first to separate out this caregiver effect, providing a much more detailed picture of the effects of bereavement on mental health.

Our analysis is based on data from the Health and Retirement Study (HRS), a large, representative panel survey of the elderly US population which collects detailed information about the daily life of the respondents and their spouses. A supplemental survey of recently widowed individuals, the HRS Exit Interviews, additionally informs about the circumstances of the partner's death, changes in daily life, and finances of the surviving spouse. Moreover, the HRS provides a link to administrative data from the National Death Index File (NDI), which contains information on official causes of death and death dates. Using the exact interview and death dates, we can construct monthly data on the adjustment of mental health. To our knowledge, we are the first to use these linked data sources in a joint analysis.

This study provides strong evidence for the anticipation of bereavement. While we find an increasing probability of depression in the pre-death period if the partner died expectedly, this effect is absent when the death of the partner was unexpected. This implies that the death event cannot generally assumed to be exogenous in an econometric analysis, leading to a smaller bereavement effect than for a completely unanticipated time of death. Analyzing respondents who were interviewed within 24 months surrounding the death event, we find that the deterioration of mental health starts about 18 months prior to the partner's expected death, and has adapted about 20 months after this event. Respondents who did not expect the time of their partner's death do not show any health adjustments, but they take longer to achieve the pre-death level of mental health. By separating out the bereavement effect from the caregiving effect, we find suggestive evidence that a higher probability of depression before the partner's death is also driven by the illness of the dying partner. However, factoring out the caregiving effect does not incrementally change the patterns of anticipation and adaptation in mental health which is consistent with other findings from the literature (Christakis and Allison (2006), Christakis and Allison (2009)). To check the robustness of our results, we conduct a subsample analysis, stratifying the data by age and gender, which confirms the effect of bereavement on mental health (see also studies of Lee and Carr (2007) and Christakis and Elwert (2008)).

The results of this study have important implications for the health care expenditures of the elderly and public health care costs. Older widowed individuals face higher risks of mental disorders and depression (see Hansson and Stroebe (2007) for a comprehensive review). They incur considerable out-of-pocket medical expenditures which are even higher than those for chronic diseases such as hypertension or arthritis. These costs are mainly driven by the structure of the Medicare system

which requires higher patient cost sharing for mental health services than for general medical services (Harman *et al.* (2004)). Moreover, widowed individuals are more likely to live in poverty than their married counterparts, due to the deceased's out-of-pocket costs and death expenses (Hurd and Wise (1989), Sevak *et al.* (2004), McGarry and Schoeni (2005)). The combination of own and the deceased spouse's health care costs thus puts widowed individuals at a higher risk of becoming poor. In order to prevent individuals from developing serious illnesses due to bereavement, we suggest an early intervention and health-related support after the death of a partner. It might countervail excess individual out-of-pocket expenditures and moderate public health care costs due to long-lasting and cost-intensive rehabilitations and medical treatments (Reynolds III *et al.* (2012)).

Besides the economic costs, such as health care expenditures, the death of the partner involves intangible costs (Ferrer-i Carbonell and van Praag (2002)). In assessing the monetary value of different life events, Frijters *et al.* (2011) show that the death of a partner or a child is associated with the highest costs (and with an extremely high amount of monetary compensation). If the effect of bereavement on mental health is underestimated, this life event is valued too low. As a consequence, health policy decisions that are based on a cost-benefit analysis might be incorrectly evaluated. The resulting negative consequences would particularly affect the elderly population, because their lower remaining life expectancy puts them at a superior risk of becoming widowed. An accurate assessment of the non-pecuniary costs of the partner's death thus is of great importance for health policy.

Section 2 summarizes the literature on bereavement and gives an overview over the analysis of anticipation and adaptation in the well-being research. In Section 3, we introduce the HRS data as well as the restricted data resources analyzed in this study. We outline our empirical strategy and the procedure for identifying a causal bereavement effect. Section 4 presents the results of the baseline specifications of static and dynamic models. We moreover characterize the onset of anticipation and the duration of adaptation using monthly differences between the interview date and the death date. Section 5 provides results from the joint analysis of the caregiving effect and the bereavement effect. In Section 6, we stratify the sample by age and gender and check the robustness of our main findings. The concluding Section 7 summarizes the findings and discusses implications for health policy.

### 2.2 Literature Review

This study is broadly related to two strands of the literature. A first group of papers to which we refer is the analysis of bereavement and health outcomes. Researchers define heightened mortality, morbidity and health risks of the recently widowed as "bereavement effect" (among others, examples can be found in Martikainen and Valkonen (1996), Espinosa and Evans (2008), Van den Berg *et al.* (2011) and Hansson and Stroebe (2007), for a comprehensive overview). Thompson *et al.* (1984) report that older bereaved spouses have an about 40 times higher risk of getting new or

worse illness two months after the loss of their spouse. Self-reported medication and perceived ill health is significantly higher than for non widowed counterparts. The death of a spouse leads to more cardiovascular diseases (Buckley *et al.* (2010)), a higher probability of arthritis and diabetes, cancer, heart trouble, and high blood pressure (Van den Berg *et al.* (2011)).

The bereavement research has identified several channels through which the death of the partner and health outcomes are associated. The loss of social and material support leads to longer duration of bereavement and directly increases mortality (Martikainen and Valkonen (1996)). Heightened mortality and morbidity is also generated by the psychological stress of caregiving for the dying (Christakis and Allison (2006)). Rosenbloom and Whittington (1993) suggest that the loss of a spouse also changes the eating behavior of the surviving partner resulting in poor nutrition and deteriorating health. Other consequences of the partner's death are higher levels of poverty and reduced welfare of widowed individuals (Sevak *et al.* (2004), McGarry and Schoeni (2005)).

The effects of bereavement vary with socioeconomic factors such as gender, age and race, but also with the duration of the illness and the causes of death. Elwert and Christakis (2006) find heightened mortality among widowers whose wives died of lung cancer or heart diseases. Other causes of death, such as Alzheimer, are not associated with these higher hazards. Women face higher mortality risks if the husband's death was caused by COPD or influenza, and pneumonia. Furthermore, Carr *et al.* (2001) analyze the impact of death forewarning on multiple indicators of mental health on 210 widowed individuals from the Changing Lives of Older Couples (CLOC) study. The authors do not find a significant impact of an unexpected death on depression, anger or overall grief. A sudden death is only associated with higher levels of yearning for women, but not for men. A more recent study of Burton *et al.* (2006) analyzes the impact of an unexpected partner's death and caregiving experiences on several measures of psychological and physical health, also using the CLOC survey data. An unexpected death of the partner is associated with worsening depression. The authors do not find a significant impact of caregiving experiences on mental health outcomes for the expected death group. The results of this study suggest that anticipation of the death event plays an important role for mental health changes, while caregiving experiences seem to be less relevant.

Our study contributes to the bereavement research in several ways. First, we analyze the bereavement effect accounting for health adjustments in the pre-death period, and show that anticipation is actually a source of endogeneity in bereavement research. According to this, we provide evidence for the underestimation of the bereavement effect. Second, we use a large data base for our analysis, which allows not only to control for individual heterogeneity, but also to draw a detailed pattern of mental health surrounding death date. Third, we study the bereavement and the caregiving effect in concert in order to get an insightful picture of the possible interdependence between these two phenomena.

Another area of research to which we can relate our paper to is well-being and life satisfaction. Typically, these studies explore the impact of life events such as unemployment (Clark and Oswald

(1994), Winkelmann and Winkelmann (1998), Blanchflower (2001), Di Tella *et al.* (2001)), divorce and marriage (e.g. Laporte and Windmeijer (2005)) or disability (Oswald and Powdthavee (2008), Dolan *et al.* (2008)) on happiness and life satisfaction outcomes. It has been shown that these life events not only have an instantaneous effect on well-being, but also have an impact in the periods before and/or after their actual occurrence. While most of these studies have focused on adaptation as a hedonic concept or hedonic treadmill [1], a lesser number of studies have also analyzed anticipation effects. In an analysis of BHPS data, Clark *et al.* (2008) find significant anticipation effects for women in the wave before the partner's death, but no effects for men. Moreover, men completely recover within one year, while women take up to two years for adaptation. A recent study by Frijters *et al.* (2011) on quarterly event data from the Australian survey HILDA suggests that anticipation of the partner's death starts in the year before death. The instantaneous effect of widowhood is very large, but offsets about two years after the death event occurred.

We adopt the concept of anticipation and adaptation in happiness research for our study of mental health. We show that the effects examined by Clark *et al.* (2008) and Frijters *et al.* (2011) are similar to those we find for respondents whose spouse died expectedly, but not for an unexpected time of death[2]. The similarity of these findings might be explained by the dependent variable we use to measure mental health status, which can also be interpreted as a measure of mental well-being. However, we did not find studies on well-being and life satisfaction which investigate the effects of an adverse life event on a monthly basis. Moreover, our study is based on data that provide more detailed information on the circumstances of the partner's death. Together with our estimation strategy, we expect to contribute to the literature with some interesting new findings on the effects of the partner's death and patterns of individual behavior surrounding the death event.

## 2.3 Empirical Strategy and Data

### 2.3.1 Data

The Health and Retirement Study (HRS) is a longitudinal panel study that biennially surveys a representative sample of US-Americans aged 50+. It collects information about various topics of US daily life, including physical and mental health, housing, family structures, employment, and retirement as well as expectations and individual decision making. We use seven consecutive waves of the RAND version of HRS from 1996–2008, which originally contain 213,829 observations from

[1] The hedonic treadmill describes the tendency of individuals to quickly return to a relatively stable level of happiness after a life event (Graham and Oswald (2010))

[2] The term "unexpectedly" refers to an unexpected time of death in this paper.

30,547 respondents[3]. During the observation period, 8,261 spouses died, and 2,777 of them were participants in the HRS.

We link the information from the HRS main survey with HRS exit data which are collected with supplemental interviews from respondents whose spouse died between two waves[4]. Exit data provide information on the circumstances of the partner's death as well as on economic and social changes in the surviving spouse's life that come along with this event. The most important variable for our study is a binary measure of the respondent's expectation about the time of the partner's death.

***"Was the death expected at about the time it occurred, or was it unexpected?"***

Merging HRS core and exit interview leaves 2,710 death events. We restrict the data to respondents with one dead spouse only (30,064 respondents with 2,694 death events) and to those with valid interview dates. Since we would like to assess anticipation and adaptation effects, respondents have to be observed before and after the partner's death at least once in our sample. We finally get 12,463 observations for 2,096 respondents with a maximum of seven waves.

A third source of data is the restricted National Death Index (NDI) file. It provides detailed information on causes of death and exact death dates for respondents whose interview status is known [5]. We link HRS core and exit interviews to the NDI file. The original NDI cause of death variable consists of 113 categories according to ICD 9 and ICD 10 codes. We collapse these two codes to their highest level and harmonize them. Since the number of categories is still too high, we aggregate the 3-digit information to the broader 2-digit level obtaining eleven categories of causes of death for 12,450 respondents. We link NDI dates with the corresponding exact interview dates which also are not publicly available. This allows us to get an accurate measure of the time elapsed between an interview and the death of a spouse. This measure is crucial for us, since the magnitude of anticipation and adaptation behavior presumably depends on time elapsed between the interview and the death event. We obtain 12,268 time differences between death and interview dates.

The respondent's mental health status is measured by the "Center of Epidemiologic Studies Depression" scale (CESD). This indicator is the most common screening test for helping individuals to determine their depression quotient (Radloff (1977)). It consists of eight items measuring depressive feelings and behaviors during the past week with yes/no questions. The CESD score ranges from zero to eight, with higher values corresponding to a higher depression quotient. Single items are: feeling depressed, everything is an effort, being sleepless, being happy, feeling alone, enjoying life, feeling sad, and feeling unmotivated. The responses for positive items (happy and enjoy life) enter the CESD score in reverse order. The indicator has an additive nature and consists of nine categories,

[3] The RAND institute provides a revised and imputed version of the complete HRS from 1992–2008. We decided to exclude the first two waves, since Exit interview information relevant to our study was not collected.

[4] 98% of the individuals from the core interview could be merged to exit interviews.

[5] For an extended description of the NDI file see http://hrsonline.isr.umich.edu/index.php?p=resprodinfo&iyear=102

including zero. Figure 2.1 presents the frequency of answers of the CESD score over all waves. The distribution is highly left skewed with a mean response at 1.8 and median at 1. Due to the highly skewed distribution of the score and the binary nature of its single items, we propose to collapse this variable into a binary indicator measuring the prevalence of depression (see Winkelmann and Winkelmann (1998)). The CESD score is coded as one if it is above one, and as zero otherwise[6]. Accordingly, individuals who report below-average are treated as mentally healthy. A CESD score above the average indicates the potential incidence of depressive symptoms. A CESD score of one thus implies a lower level of mental health[7].

The HRS has the advantage of collecting data about both spouses, allowing us to control for a large part of individual heterogeneity. In our baseline specification, we account for the dead partner's age at death, years of education, race, health conditions and health behaviors (BMI, ADL, smoking). As respondent characteristics, we include measures of age and age squared, years of education, race, sex, health insurance status, new health conditions and health behaviors (BMI, ADL, smoking, drinking). In addition, we control for the total length of marriage and whether the surviving partner has a new spouse. Besides, we add liquid and non liquid wealth to the baseline specification. To control for any caregiving effects, we moreover include the official causes of death and the duration of final illness. The HRS also provides information on the household's portfolio risk which is a measure of the household's risk aversion. In the second specification we thus account for the household's financial risk measured as the fraction of risky assets (stocks) the household holds in its portfolio, and for debts. A third specification instead includes controls for the existence of a will, the sum of death expenses, and out-of-pocket expenses of the dead spouse. We hypothesize that the household's risk attitude as well as the measures of death expenses have deleterious effects on the surviving spouse's mental health. If this is the case, the additional measures in specifications (2) and (3) capture additional effects of individual heterogeneity in mental health. Table 3.1 reports summary statistics for current prevalence of depression (CESD), the restricted data measures, and all other variables of interest.

### 2.3.2 Econometric Models

The purpose of this study is to identify the effect of conjugal bereavement on the prevalence of depression by analyzing linked longitudinal survey and administrative data with panel-based methods. We start off our empirical framework with a rather basic specification that neither takes account of dynamic mental health effects nor of a potential endogeneity problem in the timing of the death event. Apart from that, potential unobserved individual heterogeneity is eliminated by specifying a

[6] We decided to take the average as threshold value. We also defined a binary CESD score taking the median as the threshold value suggesting that only those are considered as completely mentally healthy that do not report any negative symptoms, but are happy and enjoy life. The corresponding results can be obtained upon request.

[7] We are aware that this is accompanied by a loss of efficiency. Nevertheless the binary probit estimator is consistent and this result is independent of the chosen threshold (Crouchley (1995)).

correlated random effects (CRE) framework. In a next step, we add measures for anticipation and adaptation to our model. With this specification we can find out if the surviving partner's expectation about the time of death is a reliable measure. An addition, it provides us with a test on the assumption of an exogenous time of the partner's death. We also set up these specifications in a dynamic framework.

We measure the respondent's probability of depression with a binary variable generated from the CESD score. This allows us to estimate a series of probit models motivated by a latent variable specification. The static model with unobserved heterogeneity can be written as

$$y_{it}^{*} = \beta G_i + \phi D_{it} + \theta (D * G)_{it} + \mathbf{x'}_{it}\boldsymbol{\gamma} + \alpha_i + u_{it} \tag{1}$$

with $i = 1, ..., N$ and $t = 1, ..., T$. $y_{it}^{*}$ represents the underlying, unobservable mental health status of respondent $i$ at wave $t$. $D_{it}$ is a treatment variable which takes on the value one at the period at which the death event occurs, and is zero otherwise. $G_i$ specifies whether the respondent belongs to the expected or unexpected time of death group. $(D * G)_{it}$ is an interaction term computed from $G_i$ and $D_{it}$ that takes on the value one if we observe the partner's death for the unexpected time group at a specific wave $t$. $\alpha_i$ is the unobserved individual-specific effect, $u_{it}$ represents an idiosyncratic error term and $\mathbf{x}_{it}$ contains the $M$ covariates.

Given that the unexpected time group is not subject to any anticipating behavior and there is no unobserved heterogeneity left, equation (1) identifies the causal bereavement effect. However, if there actually are anticipation effects, the bereavement effect is underestimated. In order to identify such a behavior in any of the groups, we introduce measures for anticipation and adaptation into our model by computing a number of pulse variables which measure the impact of the partner's death in the periods before and after death (see Laporte and Windmeijer (2005)). This leads to the following extended model

$$\begin{aligned} y_{it}^{*} =& \beta G_i + \phi_0 D_{it,0} + \theta_0 (D * G)_{it,0} + \mathbf{x'}_{it}\boldsymbol{\gamma} + \alpha_i \\ &+ \sum_{k=1}^{K} \phi_{-k} D_{it,-k} + \sum_{p=1}^{P} \phi_p D_{it,p} + \sum_{k=1}^{K} \theta_{-k} (D * G)_{it,-k} + \sum_{p=1}^{P} \theta_p (D * G)_{it,p} + u_{it}. \end{aligned} \tag{2}$$

Now, the coefficients $\phi_0$ and $\theta_0$ measure the instantaneous impact of the partner's death and the corresponding interaction term. $D_{it,p}$ and $(D * G)_{it,p}$ represent pulse variables for the treatment and the interaction term which take on the value one in the $p$th period after the death event, and are zero otherwise. The corresponding parameters $\phi_p$ and $\theta_p$ measure the impact of the death event $p$ periods after the treatment, indicating the degree of adaptation. In a similar fashion, $D_{it,-k}$ and $(D * G)_{it,-k}$ are dummy variables that are zero except in the $k$th period before the death event. The parameters $\phi_{-k}$ and $\theta_{-k}$ capture the impact of the death event on mental health $k$ periods before the treatment. They represent the degree of anticipation of the partner's death.

The specification in equation (2) allows us to carry out simple tests of the degree of anticipation and adaptation in mental health (Clark *et al.* (2008)). Without anticipation in any group, all coefficients

$\phi_{-k}$ and $\theta_{-k}$ take on insignificant values and are close to zero. If anticipating behavior exists, these coefficients are positive and significantly different from zero suggesting that a future death event leads to worse mental health in the pre-death period. Without any adaptation, all coefficients $\phi_p$ and $\theta_p$ take on positive values different from zero, suggesting a long run impact of the treatment on mental health status. If these coefficients become insignificant, respondents have fully adapted and mental health status has reverted to the pre-treatment level. We can also conduct a joint test on group differences. If the coefficients $\phi_{-k}$ are negative, but $\theta_{-k}$ are positive, and this difference is not significantly different from zero, anticipation in the unexpected time group is offset relatively to the expected time group.

Typically, there are two ways of estimating a causal effect as specified in equations (1) and (2) with panel data. Fixed effects models allow for a completely unrestricted relation between the unobserved heterogeneity term $\alpha_i$ and time-varying covariates. However, time-constant variable coefficients such as differences between expected and unexpected spousal death groups are not estimated. Random effects models allow for the estimation of time constant variables, but are based on the assumption that the unobserved individual-specific heterogeneity is purely random, a situation which is rarely satisfied. We therefore suggest a correlated random effects framework which allows for the estimation of time-varying covariates and imposes restrictions on the unobserved heterogeneity term.

For the implementation of the nonlinear CRE model for unbalanced panel data, we first define a vector $\mathbf{w}_{it} = \{D_{it}, G_i, (D * G)_{it}, \mathbf{x}_{it}\}$ which represents the treatment, the group variable, the interaction term, and potentially observed covariates. The unbalancedness of the data moreover requires the definition of a selection indicator $(s_{it} : t = 1, ..., T; i = 1, ..., N)$ which is equal to one if and only if observation $(i, t)$ is used, and zero otherwise. Thus, the number of time periods available for observation $i$ is $T_i = \sum_{r=1}^{T} s_{ir}$ (Wooldridge (1995)). With respect to $\mathbf{w}_{it}$, $s_{it}$, and $\alpha_i$, the exogeneity assumption is

$$E[u_{it}|\mathbf{w}_i, \alpha_i, \mathbf{s}_i] = 0, \quad t = 1, ..., T \tag{3}$$

which implies strict exogeneity of selection and all covariates. $\mathbf{s}_i = (s_{i1}, ...s_{iT})$ is allowed to be arbitrarily correlated with potential covariates $\mathbf{w}_i$ and the unobserved heterogeneity term $\alpha_i$ (Wooldridge (2009)). In order to satisfy equation (3), the standard random effects model assumes that $\alpha_i$ is independent of covariates and $s_{it}$, which is often questionable in applied work. The CRE approach deals with individual-specific effects that are correlated with $\mathbf{w}_{it}$ by explicitly specifying the relation $E[\alpha_i|\mathbf{w}_{it}]$ (Jones *et al.* (2007)). We define the dependence via a linear regression function

$$\alpha_i = \pi_0 + \bar{\mathbf{w}}_i' \boldsymbol{\pi}_\mathbf{1} + e_i \tag{4}$$

in which $\bar{\mathbf{w}}_i'$ are time averages of time-varying covariates and $e_i$ is a purely random part (Mundlak (1978)). We can carry over this specification to our unbalanced panel data case by defining $\bar{\mathbf{w}}_i =$

$T_i^{-1} \sum_{r=1}^{T} s_{ir} w_{ir}$ [8]. If the relation between the individual-specific effect and the explanatory variables is correctly specified, unobserved heterogeneity is captured by time-means, leading to a consistent and efficient estimator for the death event on mental health status of the surviving partner.

For sake of simplicity, we define a linear index

$$\begin{aligned}\mathbf{r'}_{it}\boldsymbol{\delta} =& \pi_0 + \bar{\mathbf{w}}_i'\boldsymbol{\pi_1} + \beta G_i + \phi_0 D_{it,0} + \theta_0 (D * G)_{it,0} + \mathbf{x'}_{it}\boldsymbol{\gamma} \\ &+ \sum_{k=1}^{K} \phi_{-k} D_{it,-k} + \sum_{p=1}^{P} \phi_p D_{it,p} + \sum_{k=1}^{K} \theta_{-k} (D * G)_{it,-k} + \sum_{p=1}^{P} \theta_p (D * G)_{it,p},\end{aligned} \tag{5}$$

which contains all observed components in the data, and their corresponding parameters. Given the conditional probability for the CRE probit model, $Pr[y_{it} = 1|\mathbf{r_{it}}, \boldsymbol{\delta}, e_i] = \Phi[\mathbf{r_{it}'}\boldsymbol{\delta} + e_i]$ and $u_{it} \sim N(0,1)$, the joint density for the $i$th individual $(y_{i1}, ..., y_{iT})$ under conditional independence is

$$f(\mathbf{y}_i|\mathbf{R}_i, e_i, \boldsymbol{\delta}) = \prod_{t=1}^{T} \Phi(\mathbf{r'}_{it}\boldsymbol{\delta} + e_i)^{y_{it}} \times [1 - \Phi(\mathbf{r'}_{it}\boldsymbol{\delta} + e_i)]^{1-y_{it}} \tag{6}$$

if $s_{it} = 1$. Assuming that the remaining individual effect defined in equation (4) is normally distributed, with $e_i \sim N(0, \sigma_e^2)$, integrating out $e_i$ gives the density

$$f(\mathbf{y}_i|\mathbf{R}_i, \boldsymbol{\delta}, \sigma_e^2) = \int_{-\infty}^{\infty} f(\mathbf{y}_i|\mathbf{R}_i, e_i, \boldsymbol{\delta}) \frac{1}{\sqrt{2\pi\sigma_e^2}} exp\left(\frac{-e_i}{2\sigma_e^2}\right)^2 de_i. \tag{7}$$

The random effects maximum likelihood estimator of $\boldsymbol{\delta}$ and $\sigma_e^2$ maximizes the log-likelihood $\sum_N^{i=1} lnf(\mathbf{y}_i|\mathbf{R}_i, \boldsymbol{\delta}, \sigma_e^2)$. Since there is no closed form solution, the integral in (7) is evaluated numerically using adaptive Gauss-Hermite quadrature (Rabe-Hesketh *et al.* (2005)).

In health economics, state dependence typically is a concern. The current mental health level might partly be the result of an accumulation of previous health events or problems. Moreover, mental health problems might be generated by unobserved factors, such as risk aversion or time preferences (Jones *et al.* (2006)). If persistent poor mental health is correlated with the respondent's report about the time of the partner's death, we would face a selection problem in our model. A possible scenario could be that respondents with poor mental health in the previous wave might be more pessimistic about future events, thus expecting the partner's death. We check this possibility by introducing the first lag of the dependent variable into equation (2) [9].

Consistent estimation requires solving the initial condition problem. We follow the suggestion of Wooldridge (2005) by directly modeling the distribution of the unobserved effect conditional on

[8] In addition to averages of covariates we also include time averages of wave dummies, since they are no longer constant in an unbalanced panel (Wooldridge (1995))

[9] In the evaluation literature such a situation is referred as to the Ashenfelter's dip which describes selection on idiosyncratic temporary shocks (Blundell and Costa Dias (2009)). If selection actually takes place, we expect our coefficients to be downward biased.

$(y_{i0}, \mathbf{x}_{it})$, the initial value and exogenous explanatory variables [10]. The Mundlak specification defined in equation (4) then becomes

$$\alpha_i = \pi_0 + \bar{\mathbf{w}}_i' \boldsymbol{\pi_1} + \pi_2 y_{i0} + e_i \tag{8}$$

in which the parameter $y_{i0}$ now represents the initial value of mental health. Since our panel is unbalanced, we allow for heterogeneous initial conditions, that is, each individual has his own initial period (Contoyannis *et al.* (2004b)). The conditional maximum likelihood (CML) approach is based on a modified joint density of (6)

$$\begin{aligned} &f(\mathbf{y}_i|\mathbf{R}_i, \boldsymbol{\delta}, \rho, y_{it-1}\pi_2, y_{i0}, e_i) = \\ &\prod_{t=1}^{T} \{\Phi(\mathbf{r'}_{it}\boldsymbol{\delta} + \rho y_{it-1} + \pi_2 y_{i0} + e_i)^{y_{it}} \times [1 - \Phi(\mathbf{r'}_{it}\boldsymbol{\delta} + \rho y_{it-1} + \pi_2 y_{i0} + e_i)]^{1-y_{it}}\}. \end{aligned} \tag{9}$$

The corresponding likelihood function has the same structure as in the static model with exception to the additional terms $(y_{it-1}, y_{i0})$ [11].

In the next section we will present the results from estimating the specified static and dynamic CRE probit models using the (conditional) maximum likelihood approach described above.

### 2.4 Results

In this section we consider the relationship between bereavement and mental health. We implement this by estimating static and dynamic CRE probit models based on equations (1) and (2) and accounting for important covariates (see table 3.1). For the sake of clarity, we will only report the most important coefficients from the main and interaction terms as well as from the pulse variables. Before presenting the estimation results, we illustrate the key relationship between the probability of depression of the surviving spouse and the (un)expected time of death. Figure 2.2 shows that both, the expected and the unexpected time of death group, follow a similar trend in mental health over time which indicates that there are no different pre-death patterns (Lechner (2011)). For both groups, we find a large jump in the probability of depression at the time of the partner's death. For an unexpected time of death, an additional impact of the death event on mental health is observed, indicating a larger bereavement[12]. The figure also shows group differences at the wave before the death event. In this period, the expected time of death group shows an increasing probability of

[10] please note that the specification of this auxiliary conditional distribution only results in consistent parameter estimates if we assume that the conditional distribution of the unobserved effect is not misspecified (Wooldridge (2005)).

[11] Simulation studies show that this estimator performs equally well with other estimators (Heckman (1981)) for panels with moderate long durations (see Arulampalam and Stewart (2009), Akay (2012)).

[12] For instance, these respondents lost their spouse in an accident or they were just married and still newly in love.

depression, while this remains relatively stable for the other group[13]. This supports our assumption that the partner's death is anticipated. Even though adaptation in mental health seems to take place in both groups, the probability of depression is higher for the unexpected time of death group in all observed periods after the death event. Moreover, these respondents take about one wave longer to achieve their initial probability level.

The figure gives three important implications for bereavement research: First, an unexpected time of death seems to be accompanied by a stronger bereavement effect; second, there exist strong anticipation effects for respondents whose spouse died expectedly; and third, adaptation exists and seems to have a different speed for expected and unexpected time of death group.

### 2.4.1 Basic Specifications

In table 2.2, we present the results from static and dynamic correlated random effects (CRE) probit model estimation of the probability of depression, each for three specifications[14]. The static model reveals strong bereavement effects in all specifications. Respondents experiencing the demise of their spouse have a significantly higher probability of depression after the death event. The coefficient measuring the treatment impact is relatively similar in all specifications (0.445–0.558), but significantly differ by group. The coefficients of the interaction term are positive and significantly different from zero suggesting a higher probability of depression if the time of death was not expected (between 0.161 and 0.167).

The estimation of the dynamic model provides significantly positive coefficients of the lagged dependent variable. The size of the coefficients ranges between 0.243 and 0.269, revealing a dependency structure of current and previous mental health status. Respondents with poor mental health in the previous period thus seem to leave the state of poor mental health relatively quickly in the subsequent period. These findings are in line with the literature on health dynamics which finds evidence for a substantial mobility in mental health (Hauck and Rice (2004), Yoon (2010)). As expected, the initial mental health status is strongly correlated with current mental health. Coefficient values range from 1.039 to 1.052, suggesting that respondents with a poor mental health status at the beginning of the observation period have a significantly higher probability of depression in subsequent waves. Accounting for dynamics and initial conditions in our model moderately increases the size of the coefficients related to the death event (between 3% and 8%). This suggests that neither the death event nor the time of the partner's death is remarkably influenced by the initial or previous mental health status. Nevertheless, unobserved individual heterogeneity left in the model is almost halved

[13] Please note that we use the terms "depression" and "poor mental health" interchangeably.

[14] We also estimated this relationship using a linear model. The results are available upon request.

as the additional measures for mental health are taken into account [15].

Table 2.2 reveals a significantly stronger reaction to the partner's death in terms of mental health if the time of death was unexpected. We assume that these differences are mainly caused by anticipatory behavior in the expected time of death group. In order to detect this health pattern, we estimate equation (2) and the corresponding dynamic model accounting for possible anticipation and adaptation effects around the partner's death date. Several studies on life satisfaction suggest that anticipation of a spousal death begins about one year prior to the death event, while the bereavement effect dissipates after a maximum of two years (Clark *et al.* (2008), Frijters *et al.* (2011)). Since the HRS collects data on a biennial basis, we propose to include one pre- and one post-treatment dummy for the main and the interaction term. We moreover control for anticipation and adaptation in a simultaneous way in order to prevent our estimates from an omitted variable bias (Powdthavee (2009))[16].

The estimation results are displayed in table 2.3. The first three columns refer to the static model estimations. Again, we find a large and significantly positive impact of bereavement on the mental health outcome. The probability of depression increases by 0.512–0.623, subject to the specifications. An unexpected time of the partner's death additionally increases the effect of bereavement by 0.170–0.176. For respondents whose spouse died at an expected time, we find significantly positive effects of the pre-death pulse dummies. The existence of anticipation one wave prior to death increases the current probability of depression by 0.256–0.271. In contrast, the coefficients which measure the impact of an unexpected time of death one period before the treatment are significantly negative (between -0.258 and -0.288). This implies that the anticipation effect examined for the expected time of death group is completely offset for those whose partner died unexpectedly (e.g. for specification (1): 0.256–0.258 = -0.002). An $F$-test on the net effect of the pre-treatment pulse dummies of main and interaction terms does not reject the null hypothesis for any specification (see Appendix table A.2).

The degree of adaptation one period after the death event is measured with pulse dummies. The coefficients range between 0.073 and 0.084 and are not significantly different from zero. Following Di Tella *et al.* (2010) we calculate long-run effects of bereavement for each group separately. Only a small fraction of 12% to 15% of the initial bereavement effect is left over the ensuing two years if the partner died at an expected time, suggesting that adaptation to the pre-death mental health level has completed within one period. This corresponds to findings by Clark *et al.* (2008) and

[15] Coefficients of covariates measuring the influence on health/health risks, causes of death and duration of final illness, socioeconomics for respondent and spouse, wealth, financial risk, and inheritance/death expenses are not presented in table 2.2, but are available upon request.

[16] Estimation results with more pre- and post-treatment dummies can be found in the Appendix table A.1. These results indicate that anticipation takes place only in the last pre-treatment period. For other periods the common trend assumption is satisfied.

Frijters *et al.* (2011) who examined a maximum period of two years for completing adaptation. The unexpected time of death group does not completely adapt to the pre-death level within two years. The coefficients lie between 0.161 and 0.167 (significant at the 10% level), suggesting that over 90% of the initial bereavement effect is still left over the ensuing two years. For both groups together the fraction of impact left after two years is about 30%.

The coefficients which measure the instantaneous impact of bereavement do not change if we account for dynamics in mental health. Moreover, we still find significant anticipation for those who expected the time of death and no anticipation for an unexpected time of death (see Appendix table A.2 for the corresponding hypotheses tests.). The coefficients for post-treatment dummies have lower values and are not significantly different for both death groups. The remaining fraction of the current impact of bereavement decreases to about 9% for the expected time of death group, and 75% for the other group. The lagged dependent variable as well as the initial value is highly significant and similar in size as in table 2.2. The intra-class correlation drops as the first lag of the binary CESD score measure is introduced, suggesting a lower fraction of unobserved individual heterogeneity left in the dynamic model.

Finally, we compare the coefficients from the estimations with and without accounting for pre- and post-treatment dummy effects. Controlling for anticipation and adaptation results in main effects (for the expected death group) that are between 8% and 14% higher than in table 2.2. For the unexpected time of death interaction term the size of the coefficients do only slightly change (a maximum of 5%)[17]. Our results underline the assumption about the relation of bereavement and mental health giving important implications: on the one hand, it suggests that the unexpected time of death variable actually provides a consistent estimate of the bereavement effect. On the other hand, an expected time of death rules out exogeneity in the death event due to the anticipation of future bereavement. Moreover, the changing size in the coefficients suggests an admittedly small, but additional downward bias if pre- and post-treatment dummies are not included.

### 2.4.2 Patterns Surrounding Death

The previous analysis indicates that those respondents show anticipating behavior whose spouse died expectedly. For an unexpected time of death, we do not find an anticipation effect, but a higher immediate bereavement effect. Moreover, differences in mental health between the expected and unexpected time group become significant in the wave before the death event and disappear in the wave after the partner's death.

Since the time period between two waves refers to about two years, we could not scale the probability of depression by the length of time elapsed in the previous analysis. However, we can use the

[17] We conducted Likelihood-Ratio tests and checked the information criteria for each specification. They show that accounting for dynamics always leads to a better fit (see A.3)

administrative NDI file and the restricted interview file, and construct a scale by using differences between the exact interview date and the exact death date. This allows us to determine the beginning of anticipation and adaptation. We restrict the corresponding analysis on the two waves immediately before and after the death event.

In figure 2.3 we plot the probability of depression for the expected and unexpected time group against daily differences between interview and death dates, for 365 days before and after death. The vertical dashed line indicates the date of death. Since only a few or none respondents are observed on each given day, the graph is based on a kernel smoothed regression. Even though the lines are quite volatile, the figure clearly shows a divergence of group-specific depression probabilities starting around 100 days before the partner's death. While those who expect the time of the partner's death show a higher prevalence of depression prior to death, the average probability level is fairly constant for an unexpected time of death at this time. As expected, we find a large jump in the probability of depression just as the timeline crosses the dashed line[18]. After the death event both groups seem to behave similarly in adaptation.

We replicate the above graph using observations on a monthly basis. Figure 2.4 illustrates that differences in the trend of the mental health measure between the expected and the unexpected time group start about six months before the death event. While those who expect their spouse's death show a steadily increasing probability of depression, the unexpected time group again remains on a fairly constant level of mental health. The highest probability of depression for the unexpected death group is found about one month after the spouse's death, exceeding that of the other group by more than 10%. Both groups show a similar adaptation process with a slightly lower probability of poor mental health for the expected time of death group.

We underline these findings by estimating probit models for specification (2)[19]. One drawback of the analysis is the low number of monthly observed respondents, in particular one month before the partner's death. We thus decided to collapse interviews from months one and two. We obtain one bi-monthly pulse dummy for the first two months, and monthly pulse dummies for all other interview months. We separately estimate the probit models for the expected and unexpected death groups for one wave before and after death, and plot the obtained predictions against a monthly timeline (The corresponding coefficients from the probit estimations can be found in the Appendix table A.4).

The upper graph in figure 2.5 illustrates the development of anticipation. The dashed line indicates the month of death. The overall pattern suggests that there is a trend of an increasing probability of poor mental health for the expected time group, starting about 18 months before the partner's

[18] The lines expected and unexpected deaths are not completely vertical at zero, since we do not observe respondents exactly at the day of the partner's death. Obviously nobody was willing to give an interview on the day their partner died

[19] Specification (3) does not provide sufficient variation from the additional death expenses and inheritance variables in the pre-death wave.

death. Between the 7th and the 18th month the probability levels for the two groups differ to a high extent. Starting from the sixth month onwards, the probability of depression for the expected time of death group increases from month to month, resulting in a probability more than 40% higher at the month of death.

The lower graph of figure 2.5 plots the predictions for 24 months after the spousal death. The probability of depression for the unexpected time group is higher in the month immediately following death. It drops until month four to a similar level as the other group, then increases again at month five and remains on a higher level until month 20, where it converges to the level of the expected time group. Taking a value of 0.5 as a reference probability of depression, figure 2.5 suggests that those who were interviewed about five to eight months after the expected spousal death are back at this level. Respondents who did not expect the time of their partner's death, however, take up to about 20 months to regress to the reference probability of 0.5.

The predictions illustrated in figure 2.5 reveal the mechanisms that are responsible for the findings of significant anticipation and differential adaptation in table 2.3. About 20 months before and after the death event the probability of depression of both groups are similar, implying a common trend in mental health apart from the partner's death.

## 2.5 Separating the Caregiving from the Bereavement Effect

The previous results provide evidence for the existence of anticipation and adaptation around the time of the partner's death event. With respect to our results, a higher pre-death probability of depression is caused by the anticipation of future bereavement. Besides, the literature provides an alternative explanation for this pattern: worse levels of mental health in the pre-death period can also be caused by the burden of being a care giver. This refers to the impact the final illness of the spouse typically has on the partner's health (Christakis and Allison (2006), Christakis and Allison (2009)). With the following analysis we aim to separate the caregiving from the bereavement effect using information on the duration of the final illness and causes of death from official death records.

### 2.5.1 Duration of the Partner's Final Illness

The duration of the final illness is defined as the time elapsed between the onset of a spouse's illness and his death. In the HRS, this information is collected in six categories: the duration of terminal illness took one to two hours, less than one day, less than one week, less than one month, less than one year, and more than one year. According to these categories, we generate a dummy variable which takes on the value one if the duration of the final illness was less than one month, and zero

otherwise [20].

We expect that long lasting terminal diseases are accompanied by a caregiver burden, affecting the respondent's mental health prior to the actual death event. In contrast, short durations are less likely to be associated with caregiving. Figure 2.6 shows the assumed relation between the expected/unexpected death reports of respondents and mental health by the duration of the final illness. If the spouse suffers from a final illness longer than one month, both groups show higher probabilities of depression before the actual occurrence of death. These health adjustments might be explained by the caregiver burden effect: while expectations about the time of death might differ, the onset of a long lasting terminal disease results in care giving of both groups and a decline in the survivor's mental health. Albeit to only a small extent, the unexpected time of death is associated with a higher current impact of the death event which could be attributed to the additional effect of an unanticipated bereavement. A short duration of the final illness reveals anticipation only for those whose partner's time of death was expected. The unexpected time group holds a constant probability level of depression from two waves before death onwards up to the death event. Assuming that a quick death is not attended by care giving, the higher probability of depression for the expected time group might only be due to the anticipation of future bereavement.

We verify figure 2.6 by estimating dynamic and static CRE probit models with specifications (1)–(3). In the static as well as the dynamic models of table 2.4, the coefficients for the death event are positive and highly significant. For a short duration of final illness the values of these coefficients are constantly larger than for long durations. The coefficients mostly differ in specification (3), in which the impact of the death event increases by over 40% if we switch from a short to a long duration of final illness (from 0.402 to 0.711 in the static model, 0.420 to 0.692 in the dynamic model). For an unexpected time of death, we find higher probabilities of poor mental health for long rather than short durations, which is consistent with the pattern found in figure 2.6. However, they are never significantly different from zero, suggesting no different instantaneous impact of bereavement for an unexpected time of death relative to those whose partner died at an expected date. For long lasting as well as quick deaths, the pre-treatment coefficients of the main effect are significantly different from zero at the 10% level. While this impact is moderate for long durations with values ranging from 0.175 to 0.199 over all specifications, it doubles for short term final illnesses (the values vary from 0.351 to 0.381 by specifications). This indicates that caregiving effects decrease the probability of poor mental health in the pre-death wave, while anticipation revealed for a short duration of final illness considerably increases the probability of depression before the partner's actual death. Consistent with figure 2.6, anticipation is ruled out for an unexpected time of death after a short duration of final illness. The adaptation coefficients are positive for both groups, but not significantly different from zero, suggesting a completed adaptation one wave after the partner's death, regardless

[20] We used "less than a month" as the threshold category, since the time period between this and the next category is very large.

of the duration of the terminal illness.

The results suggest that neither anticipation of future bereavement nor caregiving effects exist if the spouse died at an unexpected date and within one month after the diagnosis of final illness. A higher probability of depression in the pre-death period for those with an expected time of death can be referred to anticipation effects of future bereavement. Long durations of final illness are always associated with a higher probability of depression before the actual death event induced by a caregiver burden, and an unexpected time of death has an additional detrimental effect. Moreover, caregiving seems to attenuate bereavement effects for both expectation groups which is in line with findings from the literature (Christakis and Allison (2009)).

### 2.5.2 Causes of Death

The extent of the bereavement effect is often associated with the partner's cause of death. Some diseases, such as Alzheimer's or cancer, might be long lasting and intense, while others hit the bereaved often completely unprepared. This suggests an additional variation in the impact of the partner's death following from different lethal diseases (see for instance Martikainen and Valkonen (1996)). Predictions of the probability of depression for different causes of death are interesting, since they provide a more detailed picture of how anticipation effects depend on the causes of death.

The main illnesses from which spouses die in our sample are cancer, heart diseases and respiratory disease. Other diseases are of neurological, psychological, digestive, nutritional, or musculo-skeletal nature. They are on average considered to be long-term illnesses, thus collapsing them into one category. Spouses who died of infections, accidents and suicides, or of natural causes are defined as other causes (see Appendix table A.5 for causes of death and the duration of illness).

Figure 2.7 represents the change in the probability of having depression by expected and unexpected time of death and for five different categories of causes of death. Death events caused by cancer or respiratory diseases do not clearly impose differential impacts on the probability of depression for an expected or unexpected time of the partner's death. Anticipation seems not to be very pronounced in both groups, even though these causes of death do not necessarily lead to a quick death.

A death from heart diseases reveals different probabilities of depression for the expected and unexpected time group. While respondents who lost their spouse unexpectedly hold a constant level of mental health two waves before the death event, an expected time of death is associated with an increasing probability of depression at this time. Regardless of any health adjustments, the death of a spouse puts both groups at a similar probability level of depression after the spouse's demise. The different effects of the death event thus are considered to be driven by anticipating future bereavement.

For long-term causes of death, the expected time group is characterized by a steep increase in the probability of depression about four waves prior to the partner's demise. Afterwards, their mental

health status remains stable up to the death event. This is in line with findings from the literature which do not identify effects of the partner's death for Alzheimer's or Parkinson's disease (Christakis and Elwert (2008)). A natural explanation is that adverse health consequences evolve with the onset of the partner's illness and as caregiving starts. The long duration of these diseases however absorbs a large fraction of the caregiver burden effect, leading to a smaller instantaneous bereavement effect. For respondents who experienced an unexpected time of their partner's death, this explanation does not work, since there is in fact a large impact of the death event on the probability of depression.

For other causes of death, figure 2.7 illustrates differences between the expected and unexpected time of death group before and after the death event. While there is only a moderate effect of the death event on the probability of depression for the expected time group, this effect is large if the partner died at an unexpected time. The differences can partly be explained by deaths following from accidents and suicides which presumably occur almost always unexpectedly.

We underline the findings from figure 2.7 by estimating static and dynamic CRE probit models by causes of death (see tables A.6– A.8 in the Appendix). For spousal deaths following from cancer, heart or respiratory diseases the effects of the partner's death on the probability of depression are positive and highly significant. Whether the spouse died expectedly or unexpectedly does not make any significant difference for these causes of death. For long-term and other causes of death, the differences in mental health probabilities before and after death are only significant for the unexpected time of death group. For deaths from long-term causes we additionally find group-specific differences in the probability of depression which is lower for an unexpected time of death. For heart disease and cancer we find significant anticipation effects for the wave before death. The coefficients of the post-period main and interaction terms suggest that respondents adapt to their initial probability level of depression within one wave for almost all causes of death. There is one exception: if the spouse died of other diseases, the probability of depression one wave after death is significantly lower than its initial value, suggesting better mental health levels after rather than before the partner's death.

The analysis of death causes partly reflects the findings from the analysis of the duration of final illness. Heart diseases, for instance, often occur suddenly and do not involve a long duration of final illness. In this case, the higher probability of depression in the pre-death period for an expected time of death might represent the anticipation of future bereavement. In contrast, a higher probability of depression in the periods prior to death might reflect the caregiver burden[21].

[21] For a more detailed analysis of how anticipation and adaptation vary by final disease, a finer classification of causes of death and exact courses of disease entities would be required. Given the low number of observations for some causes of death, the results provide only suggestive evidence about how different causes of death might be related to the expectation about the time of the partner's death and how mental health is affected by bereavement or caregiver burden.

## 2.6 Analysis of Subsamples

This section studies the previous analysis with smaller samples stratified by gender and by two age groups. Studies on the bereavement effect suggest that men and women react differently to the death of a partner. Due to their mortality advantage women might more likely anticipate the death of their partner, while men might not expect their wife to die earlier. Moreover, older people know that their remaining life expectancy is low and thus expect to die with a higher probability than young individuals (see for instance Carr *et al.* (2001), Lee and Carr (2007)). We examine these potential differences in the respective subsamples and check whether our previous findings are robust to the smaller number of observations.

### 2.6.1 Men and Women

The literature suggests that the relationship between the unexpected death of the spouse and health differs by sex (Carr *et al.* (2001)). Women might more likely anticipate a sudden spousal death due to their mortality advantage which is apparent in the official statistics: 39.9% of American women aged 65 and older are widowed, compared with 12.7% of men (U.S. Bureau of the Census (2012)). If older women are more likely to anticipate the spousal death, an unexpected death might have more deleterious effects on men (Smith and Zick (1996), Van den Berg *et al.* (2007))[22]. Carr *et al.* (2001) partly found a reverse effect: women seem to have significantly higher levels of yearning than men if spousal death occurred suddenly. They infer from these findings that men and women have different strategies of coping with widowhood.

Table 2.5 reports the results of static and dynamic CRE probit estimations for men and women separately. For both sexes, we find significantly positive and large differences in the probability of depression before and after death. While female individuals show significantly stronger effects of bereavement if the partner dies unexpectedly, the effects for men do not differ by the time of death. We find significant anticipation effects for the expected, but not for the unexpected time of the husband's death. The adaptation pattern neither differs by gender nor by the expectation of death. These findings are in line with the assumption men do typically not anticipate the wife's time of death. The patterns found for the female subsample are similar to those of the main sample which is not surprising. About 70% of the respondents are women. However, the nature of the data we are analyzing imposes a high fraction of female participants (see U.S. Bureau of the Census (2012)). The age pattern of the HRS motivates us to analyze subsamples split by the respondent's age at partner's death.

[22] Widowers who loose their wife without forewarning might have to adapt to multiple roles, e.g., breadwinner and housekeeper.

### 2.6.2 The Young and the Elderly

Older individuals hold a lower remaining life expectancy than younger persons. Therefore, even an unexpected time of death might be viewed as timely for the elderly. For the younger, however, the life expectancy is higher and the partner's death more likely happens unexpectedly (Carr *et al.* (2001)). Accordingly, we expect a stronger bereavement effect for younger respondents. Moreover, we assume that any anticipation effects for the elderly that do not depend on the expectation of the partner's death. In contrast, anticipation effects are only significant for the younger if the partner's death actually was expected.

We divide our sample at an age at death of 70. This threshold corresponds to the median age at which the respondents in the sample loose their partner[23]. Table 2.6 displays the results from the static and dynamic CRE probit estimations. We find significant bereavement effects for the elderly and the younger, with larger coefficients at older ages. The unexpected time of death coefficient is large and highly significant for respondents up to age 69, but small and insignificant for those aged 70+. Anticipation exists for both age groups. While anticipation is independent of whether the time of death was expected or not in the old age subsample, anticipation at younger ages is offset if the partner's death occurred unexpectedly. Adaptation patterns do not significantly differ by age group and the time of death.

These results underline the conjecture that circumstances of death play almost no role for older individuals and that the effects of bereavement are more pronounced for younger individuals. The explanation for our findings is obvious: younger individuals have their whole life before them and simply do not expect that the spouse dies at an earlier stage of life (Van den Berg *et al.* (2007)). The elderly, however, recognize that remaining longevity is short and therefore anticipate death, no matter whether it is expected or not.

## 2.7 Discussion and Conclusion

This paper examines the effect of bereavement on mental health using linked survey and administrative data for U.S. individuals aged 50+ and their spouses. We test the assumption of an exogenous death event using information on the time of the spouse's death. Moreover, we examine the patterns of anticipation and adaptation 24 months around the death date. We also investigate whether changes in mental health can be attributed to a caregiver burden effect.

Our findings suggest that anticipation effects exist if the spouse's demise was expected. An unexpected time of the partner's death, however, is not associated with anticipation. Anticipation of

[23] Census data indicate that proportions of marital status sharply change in the population at age 70 (Kreider and Ellis (2011))

future bereavement leads to an increased probability of depression starting about 18 months before the death event. Adaptation lasts for about 20 months after death when it was unexpected, but only seven months if the death was expected. The extended analysis of the caregiver burden effect shows that a partner's death following from a long-lasting illness is associated with a higher probability of depression in the pre-death period, even if the exact time of death was not expected. We attribute these health changes to the caregiver burden. For a short duration of the final illness, a higher probability of depression before death is related to the anticipation of future bereavement. The analysis of the causes of death provides additional suggestive evidence for the existence of both, a bereavement effect and a caregiver burden effect. We furthermore find that reactions to the partner's death vary by gender and age. While the relation between mental health and bereavement found for the whole sample is mostly confirmed for women and younger individuals, the elderly always and men never seem to anticipate their wife's death.

The results of this study suggest that anticipation cannot be ignored when investigating the effects of bereavement. Anticipation leads to a higher probability of depression in the pre-death period, thus instantaneous bereavement effects might be underestimated. Our results indicate that the effect of the partner's death is not solely responsible for deteriorating mental health; a caregiver burden effect is also present in specific situations.

This study contributes to the current research on bereavement and well-being by exploiting a large longitudinal data set linked to non-public information which allows us to zoom in on mechanisms of anticipation and adaptation. Moreover, the econometric method and its specification had not yet been applied in this setting. Our paper also is one of the few studies that analyze the caregiver burden effect and the bereavement effect on mental health in concert.

Nevertheless, the study has potential limitations. First, we might face selective panel attrition. Those who report very poor health are most likely to leave the panel (see Contoyannis *et al.* (2004b)). Accordingly, widowed individuals have a higher probability of becoming nonrespondents because of their deteriorating mental health, being institutionalized, or even because of their own death (Martikainen and Valkonen (1996)). Similarly, the death of a spouse may lead to a situation in which individuals are not capable of participating in a two-hour interview. In both cases, the bereavement effect on mental health might be underestimated. However, studies on panel attrition do not find substantial differences in the magnitude of the estimated coefficients with and without correcting for health-related attrition (Contoyannis *et al.* (2004b), Hauck and Rice (2004)). We therefore do not expect attrition to cause substantial bias in the effects found in our study.

A second potential problem is the low number of observations associated with specific causes of death, and the lack of more detailed data. Unfortunately, we do not have enough observations to compare the effects of sudden causes of death, such as accidents, and with those of otherwise unexpected deaths. Moreover, a more reliable separation of bereavement and caregiving effects would require having both death records and Medicare claims data, the latter providing information on specific

health conditions and the associated out-or-pocket costs (see studies of Christakis and Allison (2006) and Christakis and Allison (2009)).

Finally, our estimations of static and dynamic CRE probit models are based on the assumption of equicorrelated errors. In a random effects framework, the error covariance matrix is normalized to one, implying that any serial error correlation within respondents over time is ruled out. Accounting for serially correlated errors would require simulation-based methods which are not readily available for unbalanced data. Future research might develop and implement such models using maximum simulated likelihood methods.

Turning to policy implications, this study highlights that a group of individuals – those who face their partner's death – is at considerable risk of developing severe mental and physical health disorders. These individuals often suffer from poor mental health, often over several years, and thus are likely to incur large out-of-pocket medical expenditures which are only partly covered by Medicare (Harman *et al.* (2004)). If the bereavement effect has been underestimated, as our study suggests, the risk and the extent of poverty might be even higher than assumed. Mental health prevention and intervention programs might attenuate these adverse effects of bereavement. Even though evidence for the efficacy of these services is limited (see Currier *et al.* (2008) for an overview), intervention programs targeted at such high risk groups might reduce the risk of mental health disorders and health care costs for the elderly.

Finally, the findings of our study imply that the death of one's spouse is associated with large non-pecuniary costs, primarily arising from changes in mental health. When evaluating policies that affect individuals in the years surrounding their spouse's death using cost-benefit analysis, it is of great importance to account for these nun-pecuniary costs.

## Tables and Figures

Table 2.1: Summary statistics

| label | N | mean | std.dev. | min | max |
|---|---|---|---|---|---|
| CESD score | 12166 | 0.418 | 0.493 | 0 | 1 |
| | | | | | |
| waves dead | 12463 | 0.492 | 0.500 | 0 | 1 |
| death unexpected | 12463 | 0.428 | 0.495 | 0 | 1 |
| number waves dead | 6195 | 2.360 | 1.323 | 1 | 6 |
| number waves alive | 6268 | 2.415 | 1.372 | 1 | 6 |
| days between interviews and death | 12268 | -77.588 | 1743.253 | -4506 | 4526 |
| | | | | | |
| **cause of death** | | | | | |
| cancer | 12463 | 0.318 | 0.466 | 0 | 1 |
| musculo-skeletal system | 12450 | 0.007 | 0.080 | 0 | 1 |
| heart, blood failure | 12450 | 0.377 | 0.485 | 0 | 1 |
| respiratory system | 12450 | 0.119 | 0.324 | 0 | 1 |
| endocrine, metabolic, nutrition | 12450 | 0.038 | 0.192 | 0 | 1 |
| digestive system | 12450 | 0.059 | 0.235 | 0 | 1 |
| neurological, sensory system | 12450 | 0.045 | 0.207 | 0 | 1 |
| emotional, psychological | 12450 | 0.006 | 0.075 | 0 | 1 |
| infection | 12450 | 0.012 | 0.108 | 0 | 1 |
| accident, suicide | 12450 | 0.012 | 0.111 | 0 | 1 |
| natural cause | 12450 | 0.007 | 0.081 | 0 | 1 |
| | | | | | |
| duration of final illness | 12354 | 4.144 | 1.641 | 1 | 6 |
| | | | | | |
| **health and health risk** | | | | | |
| respondent: new major condition | 12463 | 0.079 | 0.270 | 0 | 1 |
| respondent: new minor condition | 12463 | 0.133 | 0.339 | 0 | 1 |
| respondent: BMI | 12287 | 27.244 | 5.609 | 9.6 | 82.7 |
| respondent: ADL | 12456 | 0.153 | 0.360 | 0 | 1 |
| respondent: ever smoked | 12340 | 0.556 | 0.497 | 0 | 1 |
| respondent: drink | 12460 | 0.420 | 0.494 | 0 | 1 |
| spouse: ever had major condition | 12463 | 0.325 | 0.468 | 0 | 1 |
| spouse: ever had minor condition | 12463 | 0.426 | 0.495 | 0 | 1 |
| spouse: ever smoked | 12212 | 0.752 | 0.432 | 0 | 1 |
| spouse: ADL | 12463 | 0.144 | 0.351 | 0 | 1 |
| spouse: ADL missing | 12463 | 0.476 | 0.499 | 0 | 1 |
| spouse: BMI | 12463 | 0.111 | 0.314 | 0 | 1 |
| spouse: BMI missing | 12463 | 0.481 | 0.500 | 0 | 1 |

*continued on next page*

Table 2.1 – *continued from previous page*

| label | N | mean | std.dev. | min | max |
|---|---|---|---|---|---|
| **socioeconomic information** | | | | | |
| respondent: age | 12293 | 69.694 | 9.770 | 40 | 90 |
| respondent: age squared | 12463 | 4996.991 | 1411.015 | 1156 | 10000 |
| respondent: race | 12463 | 0.127 | 0.334 | 0 | 1 |
| respondent: gender | 12463 | 0.741 | 0.438 | 0 | 1 |
| respondent: years schooling | 12463 | 11.922 | 3.038 | 0 | 17 |
| respondent: health insured | 12463 | 0.928 | 0.259 | 0 | 1 |
| respondent: new spouse | 12463 | 0.084 | 0.278 | 0 | 1 |
| spouse: age at death | 10692 | 72.944 | 9.288 | 40 | 104 |
| spouse: race | 12374 | 0.127 | 0.333 | 0 | 1 |
| spouse: years schooling | 12314 | 11.719 | 3.350 | 0 | 17 |
| total years of marriage | 12240 | 41.179 | 15.447 | 0.1 | 72.6 |
| **financial information** | | | | | |
| log liquid wealth | 12463 | 7.560 | 6.528 | -13.813 | 17.542 |
| log non liquid wealth | 12463 | 10.316 | 3.514 | -12.826 | 16.531 |
| **inheritances & expenditures** | | | | | |
| spouse made a will | 12463 | 0.294 | 0.456 | 0 | 1 |
| amount death expenses | 12212 | 889.248 | 2959.604 | 0 | 50000 |
| amount o-o-p medical expenses | 12463 | 604.126 | 5220.511 | 0 | 217102 |
| **household's financial risk** | | | | | |
| portfolio risk | 12109 | .134 | .268 | 0 | 1 |
| debts | 12463 | .252 | .434 | 0 | 1 |

Table 2.2: Correlated random effects probit models (dependent variable: binary CESD score)

| | **static model** | | | **dynamic model** | | |
|---|---|---|---|---|---|---|
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | | | 0.243*** | 0.246*** | 0.269*** |
| | | | | [0.043] | [0.044] | [0.044] |
| initial value | | | | 1.042*** | 1.039*** | 1.052*** |
| | | | | [0.051] | [0.052] | [0.052] |
| before/after death | 0.551*** | 0.558*** | 0.445*** | 0.566*** | 0.573*** | 0.470*** |
| | [0.059] | [0.060] | [0.074] | [0.057] | [0.058] | [0.071] |
| unexpected death | -0.037 | -0.044 | -0.035 | -0.039 | -0.045 | -0.038 |
| | [0.062] | [0.063] | [0.063] | [0.051] | [0.052] | [0.051] |
| unexpected×before/after death | 0.161** | 0.165** | 0.167** | 0.172** | 0.178** | 0.181** |
| | [0.079] | [0.080] | [0.084] | [0.076] | [0.078] | [0.081] |
| constant | 1.067 | 1.206 | 1.148 | -0.408 | -0.226 | -0.371 |
| | [1.048] | [1.067] | [1.066] | [0.926] | [0.945] | [0.935] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.417 | 0.419 | 0.424 | 0.243 | 0.246 | 0.239 |
| observations | 9,634 | 9,373 | 9,410 | 9,634 | 9,373 | 9,410 |
| number of hhidpn | 1,962 | 1,961 | 1,959 | 1,962 | 1,961 | 1,959 |

0.95Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, causes of death and the duration of final illness, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses.

Table 2.3: Correlated random effects probit models with pulse dummies one period before/after treatment (dependent variable: binary CESD score)

| | **static model** | | | **dynamic model** | | |
|---|---|---|---|---|---|---|
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | | | 0.223*** | 0.229*** | 0.253*** |
| | | | | [0.044] | [0.044] | [0.044] |
| initial value | | | | 1.051*** | 1.046*** | 1.060*** |
| | | | | [0.052] | [0.053] | [0.052] |
| before/after death | 0.620*** | 0.623*** | 0.512*** | 0.616*** | 0.617*** | 0.514*** |
| | [0.068] | [0.069] | [0.082] | [0.066] | [0.067] | [0.079] |
| unexpected death | -0.045 | -0.050 | -0.042 | -0.041 | -0.045 | -0.040 |
| | [0.067] | [0.067] | [0.067] | [0.057] | [0.057] | [0.057] |
| unexpected×before/after death | 0.170** | 0.173** | 0.176** | 0.174** | 0.177** | 0.182** |
| | [0.082] | [0.084] | [0.087] | [0.080] | [0.082] | [0.085] |
| (unexpected×before/after death)$_{+1}$ | 0.161* | 0.167* | 0.161* | 0.138 | 0.138 | 0.138 |
| | [0.091] | [0.093] | [0.092] | [0.089] | [0.091] | [0.089] |
| (before/after death)$_{+1}$ | 0.084 | 0.073 | 0.075 | 0.057 | 0.045 | 0.044 |
| | [0.068] | [0.069] | [0.068] | [0.067] | [0.068] | [0.067] |
| (unexpected×before/after death)$_{-1}$ | -0.258** | -0.288** | -0.258** | -0.257** | -0.282** | -0.256** |
| | [0.114] | [0.116] | [0.114] | [0.112] | [0.115] | [0.113] |
| (before/after death)$_{-1}$ | 0.256*** | 0.271*** | 0.261*** | 0.249*** | 0.266*** | 0.252*** |
| | [0.082] | [0.083] | [0.082] | [0.080] | [0.082] | [0.081] |
| constant | 1.091 | 1.217 | 1.168 | -0.385 | -0.223 | -0.357 |
| | [1.055] | [1.074] | [1.073] | [0.936] | [0.954] | [0.944] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.419 | 0.422 | 0.426 | 0.250 | 0.253 | 0.246 |
| observations | 9,634 | 9,373 | 9,410 | 9,634 | 9,373 | 9,410 |
| number of hhidpn | 1,962 | 1,961 | 1,959 | 1,962 | 1,961 | 1,959 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, causes of death and the duration of final illness, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses.

Table 2.4: Correlated random effects probit models with pulse dummies one period before/after treatment stratified by the duration of partner's final illness (dependent variable: binary CESD score)

| | (1) | | (2) | | (3) | |
|---|---|---|---|---|---|---|
| VARIABLES | long | short | long | short | long | short |
| **static model** | | | | | | |
| before/after death | 0.556*** | 0.754*** | 0.571*** | 0.728*** | 0.402*** | 0.711*** |
| | [0.087] | [0.115] | [0.089] | [0.117] | [0.105] | [0.137] |
| unexpected death | -0.120 | 0.054 | -0.127 | 0.042 | -0.127 | 0.061 |
| | [0.098] | [0.096] | [0.099] | [0.098] | [0.098] | [0.097] |
| unexpected×before/after death | 0.172 | 0.058 | 0.180 | 0.083 | 0.099 | 0.095 |
| | [0.130] | [0.122] | [0.133] | [0.124] | [0.138] | [0.129] |
| (unexpected×before/after death)$_{+1}$ | 0.090 | 0.128 | 0.115 | 0.148 | 0.086 | 0.133 |
| | [0.145] | [0.136] | [0.147] | [0.138] | [0.146] | [0.136] |
| (before/after death)$_{+1}$ | 0.071 | 0.139 | 0.075 | 0.097 | 0.072 | 0.118 |
| | [0.085] | [0.117] | [0.086] | [0.120] | [0.086] | [0.118] |
| (unexpected×before/after death)$_{-1}$ | -0.052 | -0.420** | -0.120 | -0.437** | -0.056 | -0.424** |
| | [0.177] | [0.172] | [0.182] | [0.175] | [0.178] | [0.173] |
| (before/after death)$_{-1}$ | 0.192* | 0.359** | 0.199* | 0.379*** | 0.183* | 0.374*** |
| | [0.101] | [0.144] | [0.103] | [0.147] | [0.102] | [0.145] |
| constant | 2.510 | -0.030 | 2.748* | 0.169 | 2.872* | -0.213 |
| | [1.538] | [1.490] | [1.549] | [1.501] | [1.563] | [1.528] |
| portfolio risk | | | ✓ | ✓ | | |
| will & death expenses | | | | | ✓ | ✓ |
| rho | 0.414 | 0.410 | 0.416 | 0.413 | 0.419 | 0.417 |
| observations | 5,030 | 4,604 | 4,913 | 4,460 | 4,927 | 4,483 |
| number of hhidpn | 1,012 | 950 | 1,012 | 949 | 1,010 | 949 |
| **dynamic model** | | | | | | |
| $y_{t-1}$ | 0.211*** | 0.222*** | 0.219*** | 0.221*** | 0.237*** | 0.252*** |
| | [0.061] | [0.065] | [0.061] | [0.066] | [0.061] | [0.065] |
| initial value | 1.070*** | 1.046*** | 1.064*** | 1.051*** | 1.077*** | 1.063*** |
| | [0.070] | [0.079] | [0.070] | [0.080] | [0.070] | [0.079] |
| before/after death | 0.562*** | 0.733*** | 0.575*** | 0.702*** | 0.420*** | 0.692*** |
| | [0.084] | [0.111] | [0.085] | [0.114] | [0.101] | [0.132] |
| unexpected death | -0.091 | 0.020 | -0.100 | 0.012 | -0.095 | 0.017 |
| | [0.082] | [0.083] | [0.083] | [0.085] | [0.082] | [0.083] |
| unexpected×before/after death | 0.154 | 0.090 | 0.161 | 0.116 | 0.080 | 0.130 |
| | [0.127] | [0.119] | [0.129] | [0.121] | [0.134] | [0.126] |
| (unexpected×before/after death)$_{+1}$ | 0.052 | 0.129 | 0.074 | 0.142 | 0.046 | 0.136 |
| | [0.142] | [0.133] | [0.144] | [0.135] | [0.143] | [0.133] |
| (before/after death)$_{+1}$ | 0.060 | 0.088 | 0.062 | 0.046 | 0.058 | 0.059 |
| | [0.084] | [0.116] | [0.085] | [0.118] | [0.084] | [0.117] |
| (unexpected×before/after death)$_{-1}$ | -0.048 | -0.421** | -0.099 | -0.442** | -0.052 | -0.419** |
| | [0.175] | [0.169] | [0.179] | [0.172] | [0.175] | [0.170] |
| (before/after death)$_{-1}$ | 0.183* | 0.351** | 0.188* | 0.381*** | 0.175* | 0.360** |
| | [0.100] | [0.142] | [0.101] | [0.145] | [0.100] | [0.143] |
| constant | 1.192 | -1.694 | 1.103 | -1.083 | 1.295 | -1.942 |
| | [1.346] | [1.340] | [1.361] | [1.376] | [1.355] | [1.362] |
| portfolio risk | | | ✓ | ✓ | | |
| will & death expenses | | | | | ✓ | ✓ |
| rho | 0.229 | 0.256 | 0.229 | 0.260 | 0.223 | 0.253 |
| observations | 5,030 | 4,604 | 4,913 | 4,460 | 4,927 | 4,483 |
| number of hhidpn | 1,012 | 950 | 1,012 | 949 | 1,010 | 949 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses. A short duration of final illness is defined to last less than one month; long-term final illnesses have a suffering period of more than one month.

Table 2.5: Correlated random effects probit models with pulse dummies one period before/after treatment stratified by gender (dependent variable: binary CESD score)

| | **static model** | | | **dynamic model** | | |
|---|---|---|---|---|---|---|
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| **men** | | | | | | |
| $y_{t-1}$ | | | | 0.330*** | 0.331*** | 0.377*** |
| | | | | [0.096] | [0.097] | [0.097] |
| initial value | | | | 1.229*** | 1.242*** | 1.252*** |
| | | | | [0.123] | [0.125] | [0.126] |
| before/after death | 0.486*** | 0.458*** | 0.293* | 0.470*** | 0.443*** | 0.296* |
| | [0.134] | [0.136] | [0.170] | [0.129] | [0.131] | [0.162] |
| unexpected death | -0.196 | -0.167 | -0.185 | -0.098 | -0.068 | -0.089 |
| | [0.152] | [0.154] | [0.156] | [0.128] | [0.130] | [0.130] |
| unexpected×before/after death | 0.198 | 0.203 | 0.059 | 0.219 | 0.219 | 0.087 |
| | [0.178] | [0.181] | [0.188] | [0.173] | [0.176] | [0.181] |
| (unexpected×before/after death)$_{+1}$ | 0.199 | 0.167 | 0.189 | 0.172 | 0.124 | 0.163 |
| | [0.199] | [0.204] | [0.201] | [0.193] | [0.198] | [0.195] |
| (before/after death)$_{+1}$ | 0.042 | 0.013 | 0.017 | -0.017 | -0.044 | -0.049 |
| | [0.134] | [0.136] | [0.136] | [0.132] | [0.134] | [0.133] |
| (unexpected×before/after death)$_{-1}$ | -0.264 | -0.352 | -0.267 | -0.287 | -0.362 | -0.299 |
| | [0.255] | [0.261] | [0.258] | [0.251] | [0.256] | [0.254] |
| (before/after death)$_{-1}$ | 0.195 | 0.271 | 0.190 | 0.228 | 0.301* | 0.227 |
| | [0.176] | [0.179] | [0.179] | [0.173] | [0.175] | [0.175] |
| constant | 4.712* | 3.997 | 5.049* | 0.833 | 0.445 | 0.869 |
| | [2.604] | [2.690] | [2.677] | [2.317] | [2.405] | [2.363] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.450 | 0.452 | 0.467 | 0.254 | 0.259 | 0.262 |
| observations | 2,362 | 2,307 | 2,315 | 2,362 | 2,307 | 2,315 |
| number of hhidpn | 523 | 523 | 523 | 523 | 523 | 523 |
| **women** | | | | | | |
| $y_{t-1}$ | | | | 0.189*** | 0.194*** | 0.213*** |
| | | | | [0.050] | [0.051] | [0.050] |
| initial value | | | | 1.018*** | 1.010*** | 1.027*** |
| | | | | [0.058] | [0.059] | [0.058] |
| before/after death | 0.672*** | 0.680*** | 0.573*** | 0.676*** | 0.680*** | 0.583*** |
| | [0.081] | [0.082] | [0.095] | [0.078] | [0.080] | [0.092] |
| unexpected death | 0.001 | -0.012 | 0.003 | -0.015 | -0.027 | -0.015 |
| | [0.075] | [0.076] | [0.075] | [0.064] | [0.065] | [0.064] |
| unexpected×before/after death | 0.162* | 0.164* | 0.209** | 0.162* | 0.165* | 0.209** |
| | [0.094] | [0.095] | [0.099] | [0.092] | [0.093] | [0.097] |
| (unexpected×before/after death)$_{+1}$ | 0.158 | 0.171 | 0.162 | 0.138 | 0.149 | 0.141 |
| | [0.104] | [0.105] | [0.104] | [0.102] | [0.103] | [0.102] |
| (before/after death)$_{+1}$ | 0.105 | 0.101 | 0.098 | 0.090 | 0.082 | 0.079 |
| | [0.080] | [0.081] | [0.080] | [0.079] | [0.080] | [0.079] |
| (unexpected×before/after death)$_{-1}$ | -0.272** | -0.289** | -0.274** | -0.267** | -0.282** | -0.267** |
| | [0.128] | [0.131] | [0.129] | [0.127] | [0.129] | [0.127] |
| (before/after death)$_{-1}$ | 0.260*** | 0.256*** | 0.267*** | 0.241*** | 0.242** | 0.246*** |
| | [0.094] | [0.095] | [0.094] | [0.092] | [0.094] | [0.092] |
| constant | 0.010 | 0.325 | -0.102 | -1.079 | -0.794 | -1.160 |
| | [1.216] | [1.236] | [1.240] | [1.082] | [1.101] | [1.094] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.406 | 0.407 | 0.410 | 0.245 | 0.247 | 0.239 |
| observations | 7,272 | 7,066 | 7,095 | 7,272 | 7,066 | 7,095 |
| number of hhidpn | 1,439 | 1,438 | 1,436 | 1,439 | 1,438 | 1,436 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, causes of death and the duration of final illness, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses. The variable measuring gender is excluded from any specification.

Table 2.6: Correlated random effects probit models with pulse dummies one period before/after treatment stratified by age groups (dependent variable: binary CESD score)

| | **static model** | | | **dynamic model** | | |
|---|---|---|---|---|---|---|
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| **age 40-69** | | | | | | |
| $y_{t-1}$ | | | | 0.195*** | 0.201*** | 0.225*** |
| | | | | [0.063] | [0.064] | [0.063] |
| initial value | | | | 1.080*** | 1.088*** | 1.092*** |
| | | | | [0.072] | [0.073] | [0.072] |
| before/after death | 0.557*** | 0.567*** | 0.454*** | 0.579*** | 0.585*** | 0.494*** |
| | [0.099] | [0.102] | [0.118] | [0.096] | [0.098] | [0.114] |
| unexpected death | -0.002 | -0.028 | -0.005 | -0.031 | -0.053 | -0.037 |
| | [0.097] | [0.099] | [0.098] | [0.081] | [0.082] | [0.080] |
| unexpected×before/after death | 0.249** | 0.275** | 0.297** | 0.247** | 0.275** | 0.309** |
| | [0.121] | [0.124] | [0.128] | [0.118] | [0.121] | [0.124] |
| (unexpected×before/after death)$_{+1}$ | 0.193 | 0.218 | 0.189 | 0.165 | 0.178 | 0.161 |
| | [0.132] | [0.134] | [0.132] | [0.129] | [0.132] | [0.129] |
| (before/after death)$_{+1}$ | 0.025 | -0.002 | 0.014 | 0.015 | -0.014 | -0.005 |
| | [0.101] | [0.103] | [0.101] | [0.099] | [0.101] | [0.099] |
| (unexpected×before/after death)$_{-1}$ | -0.307** | -0.327** | -0.308** | -0.303** | -0.325** | -0.302** |
| | [0.137] | [0.140] | [0.138] | [0.136] | [0.139] | [0.137] |
| (before/after death)$_{-1}$ | 0.236** | 0.247** | 0.247** | 0.217** | 0.234** | 0.227** |
| | [0.103] | [0.105] | [0.104] | [0.102] | [0.104] | [0.103] |
| constant | -0.024 | 0.065 | 0.103 | -0.418 | -0.347 | -0.246 |
| | [0.764] | [0.771] | [0.778] | [0.662] | [0.668] | [0.669] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.381 | 0.382 | 0.385 | 0.204 | 0.203 | 0.195 |
| observations | 4,754 | 4,586 | 4,673 | 4,754 | 4,586 | 4,673 |
| number of hhidpn | 809 | 809 | 809 | 809 | 809 | 809 |
| **age 70-90** | | | | | | |
| $y_{t-1}$ | | | | 0.233*** | 0.237*** | 0.263*** |
| | | | | [0.061] | [0.062] | [0.062] |
| initial value | | | | 1.050*** | 1.037*** | 1.063*** |
| | | | | [0.076] | [0.077] | [0.077] |
| before/after death | 0.661*** | 0.647*** | 0.547*** | 0.652*** | 0.639*** | 0.542*** |
| | [0.097] | [0.098] | [0.117] | [0.093] | [0.094] | [0.112] |
| unexpected death | -0.070 | -0.057 | -0.063 | -0.037 | -0.025 | -0.029 |
| | [0.093] | [0.094] | [0.094] | [0.081] | [0.082] | [0.081] |
| unexpected×before/after death | 0.057 | 0.033 | 0.030 | 0.061 | 0.037 | 0.031 |
| | [0.113] | [0.114] | [0.120] | [0.110] | [0.111] | [0.117] |
| (unexpected×before/after death)$_{+1}$ | 0.084 | 0.076 | 0.089 | 0.076 | 0.067 | 0.076 |
| | [0.126] | [0.128] | [0.127] | [0.124] | [0.125] | [0.124] |
| (before/after death)$_{+1}$ | 0.106 | 0.098 | 0.102 | 0.072 | 0.063 | 0.066 |
| | [0.094] | [0.094] | [0.095] | [0.092] | [0.093] | [0.093] |
| (unexpected×before/after death)$_{-1}$ | -0.154 | -0.190 | -0.143 | -0.164 | -0.182 | -0.159 |
| | [0.212] | [0.217] | [0.213] | [0.208] | [0.213] | [0.209] |
| (before/after death)$_{-1}$ | 0.307** | 0.316** | 0.309** | 0.317** | 0.328** | 0.320** |
| | [0.142] | [0.144] | [0.143] | [0.140] | [0.142] | [0.140] |
| constant | -0.738 | -0.904 | -0.855 | -1.120* | -1.240* | -1.212* |
| | [0.731] | [0.739] | [0.750] | [0.638] | [0.647] | [0.648] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.458 | 0.458 | 0.466 | 0.306 | 0.308 | 0.304 |
| observations | 5,013 | 4,919 | 4,863 | 5,013 | 4,919 | 4,863 |
| number of hhidpn | 1,163 | 1,162 | 1,160 | 1,163 | 1,162 | 1,160 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, causes of death and the duration of final illness, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses.

Figure 2.1: Distribution of CESD scores index with nine categories

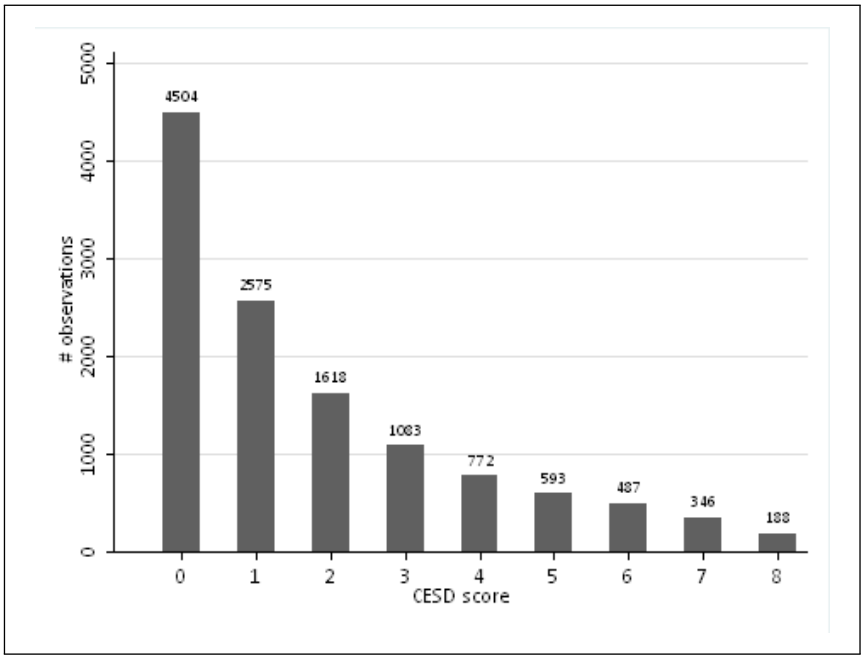



Figure 2.2: Change in CESD scores by expected/unexpected time of death, five waves surrounding death

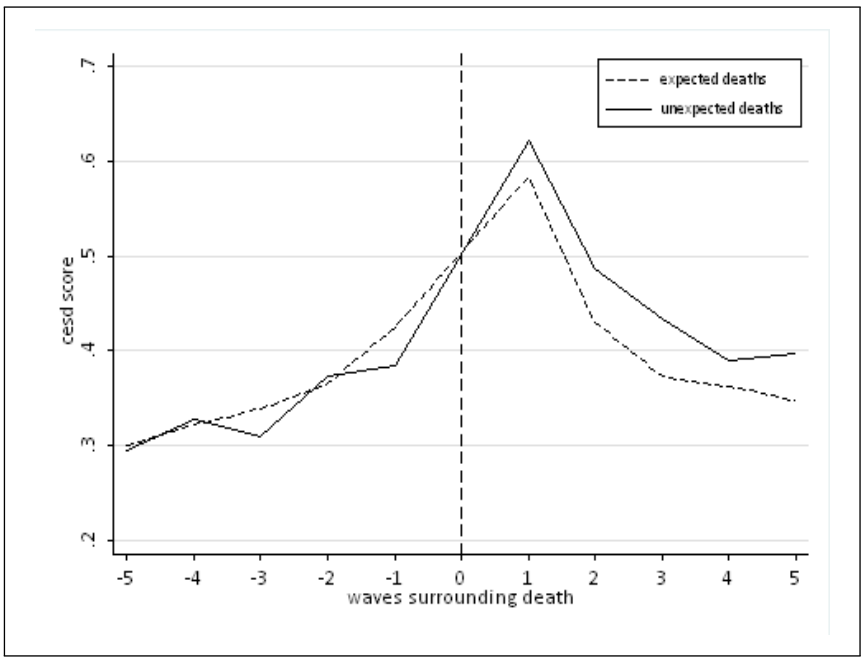


Figure 2.3: Change in CESD scores by expected/unexpected time of death, 365 days surrounding the partner's death; the CESD score ranges from 0 to 1

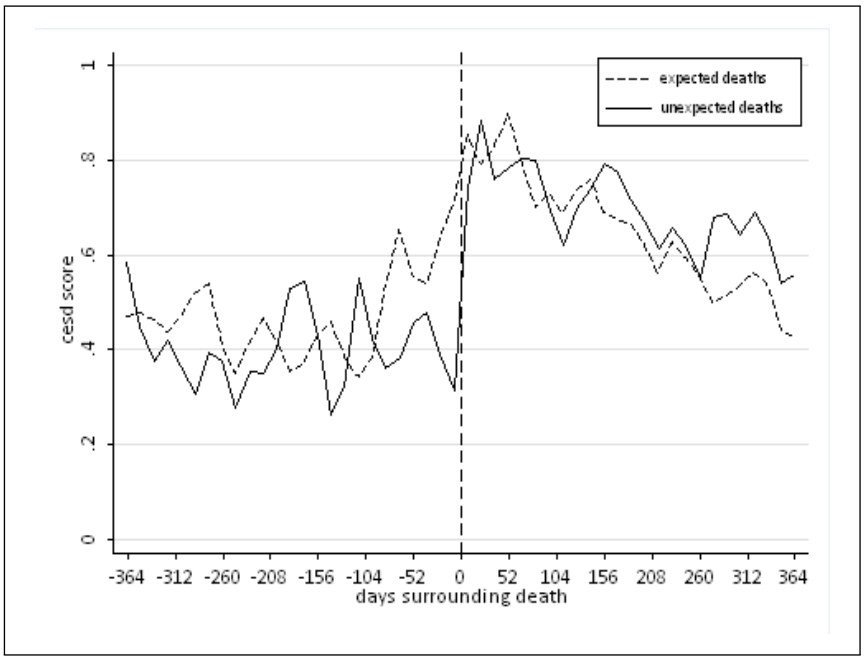



Figure 2.4: Change in CESD scores by expected/unexpected time of death, 12 months surrounding the partner's death; the CESD score ranges from 0 to 1

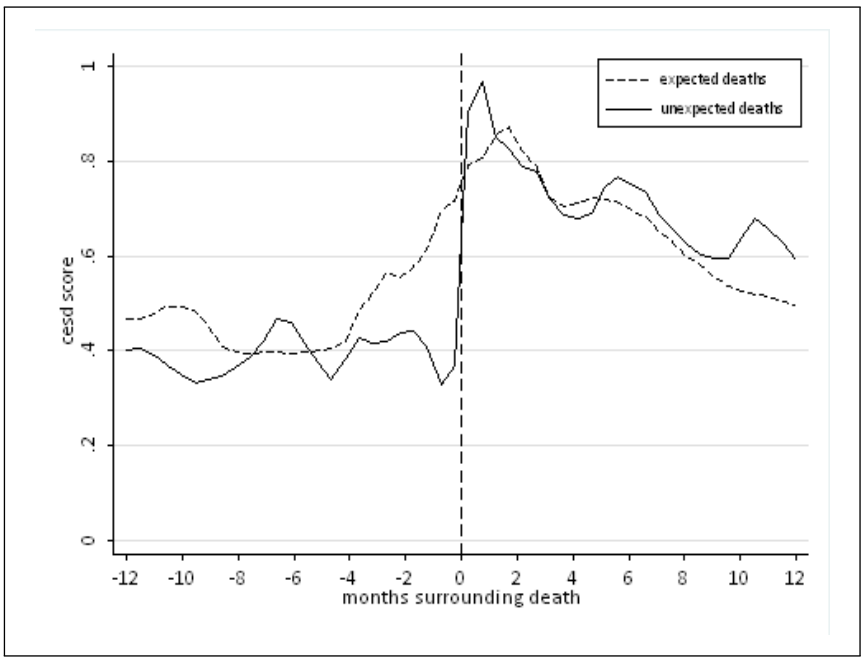


Figure 2.5: Change in predicted CESD scores by expected/unexpected time of death, 12 months surrounding death (predictions are based on specification (2))

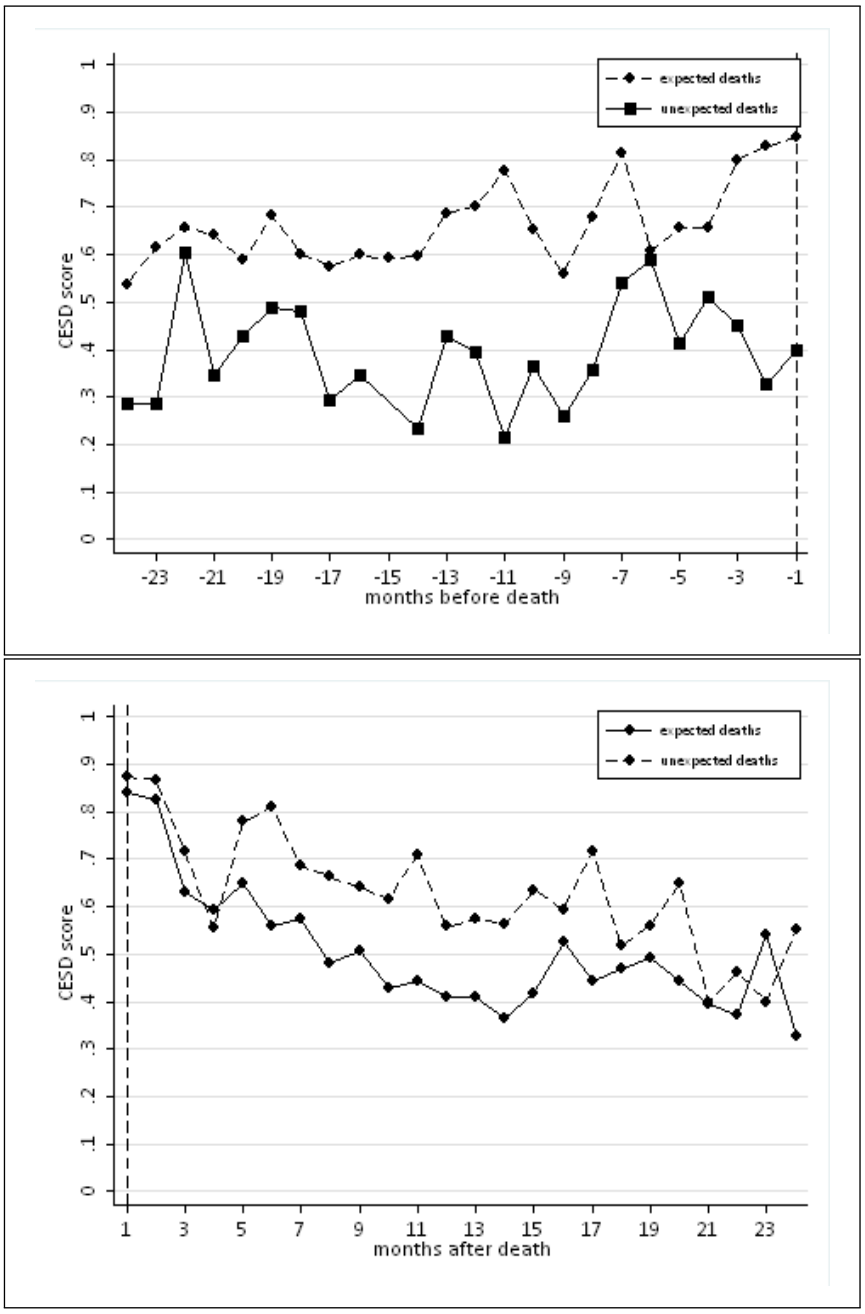


Figure 2.6: Change in CESD scores by expected/unexpected time of death for short (< 1 month) and long (> 1 month) durations of the partner's final illness, five waves surrounding death

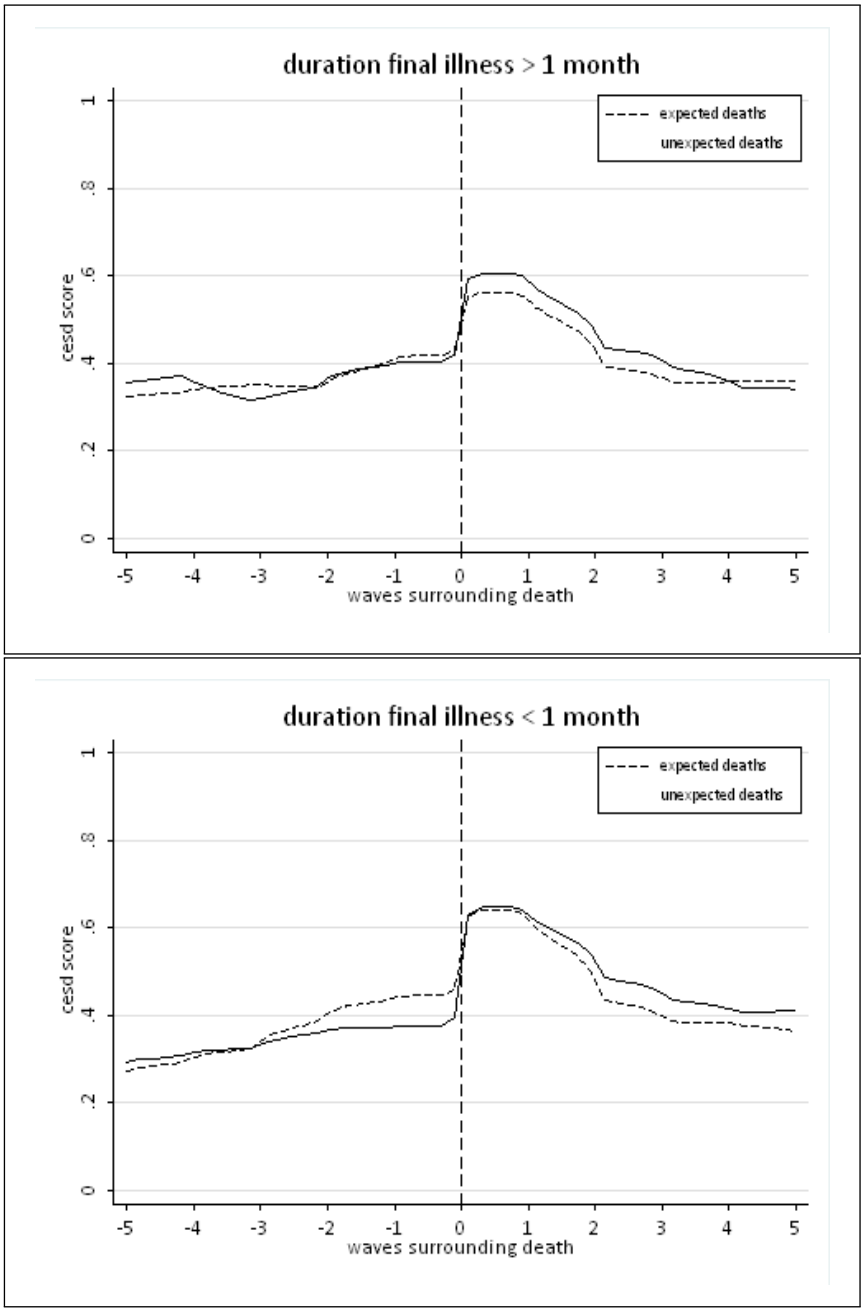
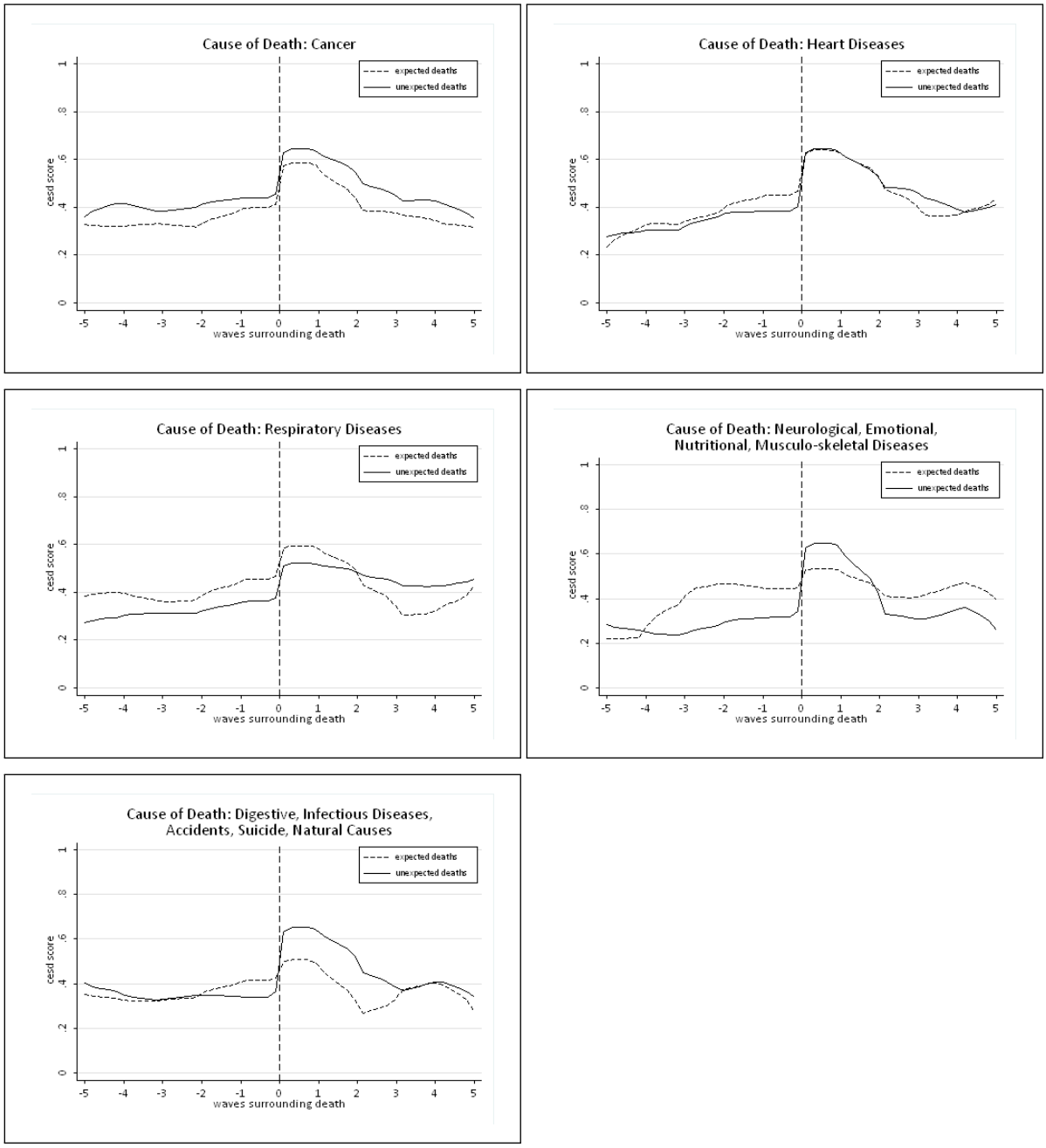


Figure 2.7: Change in CESD scores by expected/unexpected time of death for five groups of causes of the partner's death, five waves surrounding death



## Appendix

Table A.1: Correlated random effects probit models with pulse dummies three periods before/after treatment (dependent variable: binary CESD score)

| | **static model** | | | **dynamic model** | | |
|---|---|---|---|---|---|---|
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | 0.218*** | | 0.223*** | | 0.246*** |
| | | [0.044] | | [0.045] | | [0.045] |
| initial value | | 1.059*** | | 1.054*** | | 1.068*** |
| | | [0.052] | | [0.053] | | [0.053] |
| before/after death | 0.611*** | 0.608*** | 0.573*** | 0.573*** | 0.492*** | 0.496*** |
| | [0.093] | [0.090] | [0.094] | [0.092] | [0.104] | [0.100] |
| unexpected death | -0.056 | -0.040 | -0.077 | -0.054 | -0.057 | -0.041 |
| | [0.096] | [0.088] | [0.098] | [0.090] | [0.097] | [0.088] |
| unexpected×before/after death | 0.182* | 0.174* | 0.200* | 0.187* | 0.193* | 0.185* |
| | [0.107] | [0.105] | [0.110] | [0.107] | [0.111] | [0.108] |
| (unexpected×before/after death)$_{+1}$ | 0.175 | 0.139 | 0.195* | 0.148 | 0.179 | 0.142 |
| | [0.116] | [0.113] | [0.118] | [0.115] | [0.116] | [0.113] |
| (unexpected×before/after death)$_{+2}$ | 0.133 | 0.109 | 0.130 | 0.099 | 0.140 | 0.112 |
| | [0.130] | [0.126] | [0.132] | [0.128] | [0.130] | [0.126] |
| (unexpected×before/after death)$_{+3}$ | -0.061 | -0.070 | -0.038 | -0.059 | -0.061 | -0.072 |
| | [0.147] | [0.143] | [0.150] | [0.145] | [0.148] | [0.143] |
| (before/after death)$_{+1}$ | 0.078 | 0.052 | 0.026 | 0.003 | 0.057 | 0.028 |
| | [0.094] | [0.092] | [0.096] | [0.093] | [0.095] | [0.093] |
| (before/after death)$_{+2}$ | -0.046 | -0.044 | -0.094 | -0.087 | -0.064 | -0.059 |
| | [0.101] | [0.099] | [0.103] | [0.100] | [0.102] | [0.099] |
| (before/after death)$_{+3}$ | -0.019 | -0.021 | -0.073 | -0.063 | -0.027 | -0.028 |
| | [0.110] | [0.107] | [0.112] | [0.108] | [0.111] | [0.107] |
| (unexpected×before/after death)$_{-1}$ | -0.255* | -0.265** | -0.271** | -0.280** | -0.252* | -0.263** |
| | [0.132] | [0.130] | [0.135] | [0.133] | [0.133] | [0.131] |
| (unexpected×before/after death)$_{-2}$ | -0.012 | -0.025 | 0.023 | 0.005 | -0.011 | -0.023 |
| | [0.117] | [0.115] | [0.119] | [0.117] | [0.118] | [0.115] |
| (unexpected×before/after death)$_{-3}$ | -0.042 | -0.066 | -0.022 | -0.051 | -0.027 | -0.057 |
| | [0.152] | [0.150] | [0.157] | [0.154] | [0.153] | [0.150] |
| (before/after death)$_{-1}$ | 0.329*** | 0.318*** | 0.332*** | 0.323*** | 0.339*** | 0.325*** |
| | [0.100] | [0.098] | [0.102] | [0.100] | [0.101] | [0.098] |
| (before/after death)$_{-2}$ | 0.105 | 0.102 | 0.087 | 0.082 | 0.113 | 0.108 |
| | [0.084] | [0.082] | [0.085] | [0.084] | [0.085] | [0.083] |
| (before/after death)$_{-3}$ | 0.140 | 0.138 | 0.134 | 0.128 | 0.139 | 0.138 |
| | [0.102] | [0.101] | [0.104] | [0.103] | [0.103] | [0.101] |
| constant | 1.082 | -0.393 | 1.214 | -0.222 | 1.148 | -0.372 |
| | [1.059] | [0.940] | [1.078] | [0.958] | [1.077] | [0.948] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.420 | 0.251 | 0.422 | 0.254 | 0.426 | 0.247 |
| observations | 9,634 | 9,634 | 9,373 | 9,373 | 9,410 | 9,410 |
| number of hhidpn | 1,962 | 1,962 | 1,961 | 1,961 | 1,959 | 1,959 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, causes of death and the duration of final illness, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses.

Table A.2: $F$-tests on the degree of anticipation and adaptation within and between groups using coefficients from table 2.3

| hypotheses | (1) | (2) | (3) |
|---|---|---|---|
| **static model** | | | |
| **anticipation**: differences between groups | | | |
| (unexp×after death)$_{-1}$+ (after death)$_{-1}$ = 0 | 0.00 (0.98) | 0.00 (0.69) | 0.00 (0.99) |
| **adaptation**: differences within groups | | | |
| (unexp×after death) - (unexp×after death)$_{+1}$ = 0 | 0.01 (0.91) | 0.01 (0.94) | 0.03 (0.87) |
| (after death) - (after death)$_{+1}$ = 0 | 56.87 (0.00) | 56.55 (0.00) | 14.38(0.00) |
| **adaptation**: differences between groups | | | |
| (after death) -(after death)$_{+1}$ | | | |
| = (unexp×after death) - (unexp×after death)$_{+1}$ | 10.57 (0.00) | 10.70 (0.00) | 3.83 (0.05) |
| **dynamic model** | | | |
| **anticipation**: differences between groups | | | |
| (unexp×after death)$_{-1}$+ (after death)$_{-1}$ = 0 | 0.03 (0.87) | 0.02 (0.88) | 0.02 (0.88) |
| **adaptation**: differences within groups | | | |
| (unexp×after death) - (unexp×after death)$_{+1}$ = 0 | 0.14 (0.71) | 0.10 (0.75) | 0.21 (0.65) |
| (after death) - (after death)$_{+1}$ = 0 | 65.03 (0.00) | 64.60 (0.00) | 18.14 (0.00) |
| **adaptation**: differences between groups | | | |
| (after death) -(after death)$_{+1}$ | | | |
| = (unexp×after death) - (unexp×after death)$_{+1}$ | 11.09 (0.00) | 11.23 (0.00) | 4.12 (0.04) |

p-values in brackets; columns (1)-(3) correspond to specifications (1)-(3) from table 2.3

Table A.3: Comparison of the model fit without and with pulse dummies one period before/after treatment

| specification | AIC | LR |
|---|---|---|
| **static model** | | |
| specification 1 | | |
| without pulse dummies | 10736.88 | |
| with pulse dummies | 10721.44 | 23.44 (0.000) |
| specification 2 | | |
| without pulse dummies | 10449.09 | |
| with pulse dummies | 10433.42 | 23.67 (0.000) |
| specification 3 | | |
| without pulse dummies | 10480.92 | |
| with pulse dummies | 10466.11 | 22.82 (0.000) |
| **dynamic model** | | |
| specification 1 | | |
| without pulse dummies | 10214.56 | |
| with pulse dummies | 10203.81 | 18.75 (0.001) |
| specification 2 | | |
| without pulse dummies | 9940.303 | |
| with pulse dummies | 9921.471 | 18.83 (0.001) |
| specification 3 | | |
| without pulse dummies | 9948.232 | |
| with pulse dummies | 9938.328 | 17.90 (0.001) |

p-values in brackets; the test statistics without pulse dummies refer to the estimation results of table 2.2, the test statistics with pulse dummies to the estimation results of table 2.3

Table A.4: Probit model estimations for impact of timing of interview on mental health status, by expected and unexpected timing of death (dependent variable: binary CESD score)

| | **24 months before death** | | **24 months after death** | |
|---|---|---|---|---|
| VARIABLES | (exp) | (unexp) | (exp) | (unexp) |
| month 1+2 | 1.206*** | -0.243 | 1.235*** | 1.151*** |
| | [0.339] | [0.420] | [0.241] | [0.297] |
| month 3 | 1.018** | 0.020 | 0.686*** | 0.548* |
| | [0.420] | [0.409] | [0.233] | [0.295] |
| month 4 | 0.464 | 0.124 | 0.499** | 0.062 |
| | [0.354] | [0.403] | [0.223] | [0.293] |
| month 5 | 0.574* | 0.038 | 0.716*** | 0.832*** |
| | [0.345] | [0.466] | [0.222] | [0.311] |
| month 6 | 0.446 | 0.525 | 0.569** | 0.948*** |
| | [0.339] | [0.345] | [0.232] | [0.299] |
| month 7 | 1.203*** | 0.288 | 0.440* | 0.481 |
| | [0.398] | [0.401] | [0.249] | [0.294] |
| month 8 | 0.645* | -0.221 | 0.304 | 0.433 |
| | [0.374] | [0.403] | [0.230] | [0.298] |
| month 9 | 0.247 | -0.555 | 0.309 | 0.350 |
| | [0.388] | [0.382] | [0.231] | [0.305] |
| month 10 | 0.715* | -0.118 | 0.151 | 0.233 |
| | [0.372] | [0.463] | [0.232] | [0.376] |
| month 11 | 0.906** | -0.883* | 0.055 | 0.564* |
| | [0.401] | [0.529] | [0.235] | [0.291] |
| month 12 | 0.800** | -0.044 | 0.015 | 0.111 |
| | [0.344] | [0.459] | [0.245] | [0.291] |
| month 13 | 0.896** | 0.043 | 0.040 | 0.157 |
| | [0.382] | [0.464] | [0.253] | [0.326] |
| month 14 | 0.404 | -0.686 | 0.002 | 0.249 |
| | [0.432] | [0.441] | [0.284] | [0.300] |
| month 15 | 0.489 | | 0.042 | 0.300 |
| | [0.384] | | [0.260] | [0.323] |
| month 16 | 0.424 | -0.306 | 0.362 | 0.197 |
| | [0.363] | [0.502] | [0.254] | [0.289] |
| month 17 | 0.184 | -0.527 | 0.117 | 0.475 |
| | [0.429] | [0.403] | [0.276] | [0.306] |
| month 18 | 0.382 | 0.093 | 0.121 | -0.095 |
| | [0.355] | [0.468] | [0.235] | [0.278] |
| month 19 | 0.792** | 0.171 | 0.471* | 0.097 |
| | [0.310] | [0.477] | [0.278] | [0.294] |
| month 20 | 0.467 | 0.036 | 0.176 | 0.316 |
| | [0.375] | [0.460] | [0.276] | [0.294] |
| month 21 | 0.466 | -0.294 | -0.004 | -0.323 |
| | [0.423] | [0.469] | [0.263] | [0.323] |
| month 22 | 0.785** | 0.680 | 0.102 | -0.091 |
| | [0.350] | [0.437] | [0.292] | [0.390] |
| month 23 | 0.410 | -0.522 | 0.630** | -0.323 |
| | [0.451] | [0.399] | [0.297] | [0.327] |
| month 24 | 0.227 | -0.455 | -0.200 | 0.085 |
| | [0.474] | [0.493] | [0.315] | [0.357] |
| observations | 485 | 390 | 1,066 | 782 |

clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; estimation results refer to specification (2) only; separate estimations for 24 months before and after the death event; Reference group for month-dummies: more than 24 months before/after the spousal death.

Table A.5: Mean and median duration of final illness for 11 categories of official causes of death

| **cause of death** | **mean** | **p50** | **N** |
|---|---|---|---|
| cancer | 4.90 | 5 | 3947 |
| musculo-skeletal diseases | 5.10 | 5 | 81 |
| heart, circulatory system | 3.42 | 3 | 4642 |
| respiratory system | 4.36 | 4 | 1472 |
| endocrine, metabolic, nutrition | 4.09 | 5 | 472 |
| digestive system | 4.12 | 4 | 732 |
| neurological, sensory system | 4.92 | 6 | 561 |
| emotional, psychological | 3.91 | 5 | 58 |
| infectious diseases | 3.97 | 4 | 146 |
| accidents, Suicide | 1.86 | 1 | 154 |
| natural cause | 4.09 | 4 | 76 |
| | | | |
| total | 4.14 | 5 | 12341 |

Categories refer to 2-digit ICD 10 codes from the National Death Index File.

Table A.6: Correlated random effects probit models with pulse dummies one period before/after treatment for single causes of death (dependent variable: binary CESD score)

| | **cause of death: cancer** | | | | | | **cause of death: heart diseases** | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **static model** | | | **dynamic model** | | | **static model** | | | **dynamic model** | | |
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | | | 0.199** | 0.215*** | 0.226*** | | | | 0.303*** | 0.306*** | 0.330*** |
| | | | | [0.079] | [0.080] | [0.080] | | | | [0.071] | [0.073] | [0.073] |
| initial value | | | | 1.115*** | 1.094*** | 1.123*** | | | | 1.002*** | 0.994*** | 1.026*** |
| | | | | [0.093] | [0.092] | [0.094] | | | | [0.084] | [0.086] | [0.085] |
| before/after death | 0.661*** | 0.660*** | 0.536*** | 0.684*** | 0.680*** | 0.579*** | 0.795*** | 0.776*** | 0.695*** | 0.784*** | 0.767*** | 0.674*** |
| | [0.111] | [0.111] | [0.136] | [0.106] | [0.107] | [0.131] | [0.132] | [0.134] | [0.160] | [0.128] | [0.129] | [0.153] |
| unexpected death | 0.067 | 0.075 | 0.059 | 0.050 | 0.054 | 0.043 | 0.017 | -0.016 | 0.017 | 0.031 | 0.013 | 0.029 |
| | [0.130] | [0.130] | [0.132] | [0.110] | [0.110] | [0.111] | [0.104] | [0.105] | [0.105] | [0.089] | [0.091] | [0.089] |
| unexpected×before/after death | 0.138 | 0.168 | 0.116 | 0.130 | 0.166 | 0.083 | -0.003 | 0.021 | -0.030 | 0.027 | 0.043 | 0.010 |
| | [0.178] | [0.181] | [0.188] | [0.173] | [0.176] | [0.182] | [0.140] | [0.142] | [0.150] | [0.136] | [0.138] | [0.145] |
| (unexpected×before/after death)$_{+1}$ | 0.179 | 0.174 | 0.179 | 0.138 | 0.136 | 0.131 | -0.084 | -0.075 | -0.079 | -0.071 | -0.080 | -0.066 |
| | [0.197] | [0.198] | [0.197] | [0.193] | [0.193] | [0.193] | [0.154] | [0.156] | [0.155] | [0.151] | [0.152] | [0.151] |
| (before/after death)$_{+1}$ | 0.059 | 0.070 | 0.049 | 0.056 | 0.061 | 0.038 | 0.303** | 0.289** | 0.287** | 0.233* | 0.224* | 0.213 |
| | [0.106] | [0.107] | [0.107] | [0.105] | [0.105] | [0.105] | [0.135] | [0.136] | [0.136] | [0.133] | [0.134] | [0.133] |
| (unexpected×before/after death)$_{-1}$ | -0.071 | -0.098 | -0.072 | -0.094 | -0.108 | -0.100 | -0.472** | -0.434** | -0.471** | -0.497** | -0.474** | -0.497** |
| | [0.220] | [0.224] | [0.222] | [0.218] | [0.220] | [0.219] | [0.205] | [0.210] | [0.206] | [0.202] | [0.207] | [0.203] |
| (before/after death)$_{-1}$ | 0.226* | 0.239* | 0.231* | 0.206* | 0.213* | 0.213* | 0.429** | 0.395** | 0.434** | 0.432** | 0.417** | 0.433** |
| | [0.124] | [0.126] | [0.125] | [0.122] | [0.123] | [0.123] | [0.177] | [0.182] | [0.178] | [0.175] | [0.179] | [0.175] |
| constant | 0.215 | -0.045 | 0.260 | 0.328 | 0.132 | 0.406 | 1.597 | 1.789 | 1.639 | -0.017 | 0.181 | -0.060 |
| | [1.907] | [1.916] | [1.937] | [1.696] | [1.699] | [1.709] | [1.693] | [1.741] | [1.724] | [1.502] | [1.549] | [1.515] |
| portfolio risk | | ✓ | | | ✓ | | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ | | | ✓ | | | ✓ |
| rho | 0.428 | 0.422 | 0.435 | 0.250 | 0.240 | 0.248 | 0.414 | 0.416 | 0.424 | 0.244 | 0.249 | 0.241 |
| observations | 3,204 | 3,134 | 3,142 | 3,204 | 3,134 | 3,142 | 3,611 | 3,498 | 3,515 | 3,611 | 3,498 | 3,515 |
| number of hhidpn | 620 | 620 | 619 | 620 | 620 | 619 | 756 | 755 | 756 | 756 | 755 | 756 |

Clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses.

Table A.7: Correlated random effects probit models with pulse dummies one period before/after treatment for single and groups of causes of death (dependent variable: binary CESD score)

| | **cause of death: respiratory diseases** | | | | | | **cause of death: long-term diseases** | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **static model** | | | **dynamic model** | | | **static model** | | | **dynamic model** | | |
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | | | 0.236* | 0.224* | 0.250* | | | | 0.054 | 0.093 | 0.099 |
| | | | | [0.128] | [0.130] | [0.128] | | | | [0.156] | [0.161] | [0.157] |
| initial value | | | | 1.146*** | 1.167*** | 1.154*** | | | | 0.840*** | 0.874*** | 0.880*** |
| | | | | [0.148] | [0.153] | [0.149] | | | | [0.208] | [0.217] | [0.208] |
| before/after death | 0.659*** | 0.724*** | 0.702*** | 0.692*** | 0.759*** | 0.704*** | 0.190 | 0.121 | 0.160 | 0.181 | 0.101 | 0.136 |
| | [0.206] | [0.214] | [0.240] | [0.199] | [0.207] | [0.233] | [0.213] | [0.220] | [0.248] | [0.210] | [0.216] | [0.244] |
| unexpected death | -0.114 | -0.098 | -0.093 | -0.000 | 0.005 | 0.016 | -0.570** | -0.616** | -0.596** | -0.517** | -0.568** | -0.534** |
| | [0.182] | [0.186] | [0.182] | [0.152] | [0.156] | [0.151] | [0.229] | [0.241] | [0.232] | [0.219] | [0.228] | [0.217] |
| unexpected×before/after death | -0.226 | -0.317 | -0.115 | -0.260 | -0.349 | -0.139 | 0.840*** | 0.927*** | 0.899*** | 0.835*** | 0.928*** | 0.940*** |
| | [0.246] | [0.253] | [0.261] | [0.241] | [0.248] | [0.255] | [0.309] | [0.322] | [0.333] | [0.307] | [0.317] | [0.327] |
| (unexpected×before/after death)$_{+1}$ | -0.081 | -0.087 | -0.105 | -0.112 | -0.118 | -0.130 | 0.451 | 0.486 | 0.468 | 0.451 | 0.485 | 0.458 |
| | [0.278] | [0.283] | [0.278] | [0.270] | [0.275] | [0.270] | [0.355] | [0.373] | [0.357] | [0.356] | [0.374] | [0.356] |
| (before/after death)$_{+1}$ | 0.299 | 0.294 | 0.318 | 0.264 | 0.263 | 0.274 | -0.235 | -0.376* | -0.207 | -0.247 | -0.399* | -0.219 |
| | [0.195] | [0.199] | [0.196] | [0.192] | [0.196] | [0.193] | [0.211] | [0.222] | [0.212] | [0.209] | [0.220] | [0.210] |
| (unexpected×before/after death)$_{-1}$ | -0.073 | -0.262 | -0.100 | -0.059 | -0.243 | -0.077 | 0.369 | 0.334 | 0.396 | 0.336 | 0.307 | 0.365 |
| | [0.369] | [0.385] | [0.369] | [0.359] | [0.376] | [0.359] | [0.445] | [0.464] | [0.448] | [0.441] | [0.458] | [0.441] |
| (before/after death)$_{-1}$ | 0.334 | 0.331 | 0.357 | 0.287 | 0.291 | 0.313 | -0.077 | -0.000 | -0.084 | -0.036 | 0.039 | -0.037 |
| | [0.239] | [0.243] | [0.240] | [0.235] | [0.239] | [0.235] | [0.267] | [0.279] | [0.267] | [0.266] | [0.277] | [0.265] |
| constant | -0.619 | -0.877 | -0.562 | -3.068 | -3.311 | -2.928 | 3.798 | 4.282 | 4.049 | 2.205 | 2.516 | 2.093 |
| | [3.367] | [3.437] | [3.413] | [2.891] | [2.972] | [2.928] | [3.565] | [3.742] | [3.623] | [3.459] | [3.601] | [3.461] |
| portfolio risk | | ✓ | | | ✓ | | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ | | | ✓ | | | ✓ |
| rho | 0.401 | 0.411 | 0.397 | 0.185 | 0.205 | 0.175 | 0.352 | 0.378 | 0.355 | 0.292 | 0.307 | 0.273 |
| observations | 1,139 | 1,110 | 1,115 | 1,139 | 1,110 | 1,115 | 875 | 845 | 856 | 875 | 845 | 856 |
| number of hhidpn | 240 | 240 | 239 | 240 | 240 | 239 | 190 | 190 | 189 | 190 | 190 | 189 |

clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses. Long term causes of death are: neurological, emotional/psychological, nutritional/endocrine/metabolic, and musculo-skeletal conditions.

Table A.8: Correlated random effects probit models with pulse dummies one period before/after treatment for other causes of death (dependent variable: binary CESD score)

| | **cause of death: other diseases** | | | | | |
|---|---|---|---|---|---|---|
| | **static model** | | | **dynamic model** | | |
| VARIABLES | (1) | (2) | (3) | (1) | (2) | (3) |
| $y_{t-1}$ | | | | -0.088 | -0.074 | 0.001 |
| | | | | [0.159] | [0.161] | [0.162] |
| initial value | | | | 1.241*** | 1.268*** | 1.218*** |
| | | | | [0.201] | [0.200] | [0.194] |
| before/after death | 0.348 | 0.380 | -0.034 | 0.219 | 0.225 | -0.132 |
| | [0.259] | [0.264] | [0.307] | [0.254] | [0.259] | [0.297] |
| unexpected death | 0.237 | 0.251 | 0.242 | 0.223 | 0.226 | 0.215 |
| | [0.210] | [0.211] | [0.208] | [0.190] | [0.188] | [0.183] |
| unexpected×before/after death | 0.538* | 0.505* | 0.760** | 0.562** | 0.531* | 0.768** |
| | [0.283] | [0.290] | [0.308] | [0.279] | [0.285] | [0.300] |
| (unexpected×before/after death $)_{+1}$ | 0.852** | 0.870** | 0.835** | 0.862** | 0.891** | 0.834** |
| | [0.352] | [0.358] | [0.354] | [0.352] | [0.357] | [0.351] |
| (before/after death $)_{+1}$ | -0.574* | -0.598* | -0.580* | -0.584* | -0.634** | -0.614** |
| | [0.307] | [0.313] | [0.309] | [0.305] | [0.310] | [0.305] |
| (unexpected×before/after death $)_{-1}$ | -0.518 | -0.577 | -0.481 | -0.534 | -0.601 | -0.470 |
| | [0.402] | [0.410] | [0.406] | [0.400] | [0.408] | [0.401] |
| (before/after death $)_{-1}$ | 0.159 | 0.220 | 0.163 | 0.235 | 0.320 | 0.229 |
| | [0.300] | [0.301] | [0.304] | [0.296] | [0.298] | [0.298] |
| constant | 4.772 | 5.377 | 5.584 | 1.325 | 1.600 | 1.604 |
| | [3.709] | [3.750] | [3.741] | [3.459] | [3.481] | [3.409] |
| portfolio risk | | ✓ | | | ✓ | |
| will & death expenses | | | ✓ | | | ✓ |
| rho | 0.384 | 0.379 | 0.370 | 0.263 | 0.245 | 0.217 |
| observations | 878 | 856 | 852 | 878 | 856 | 852 |
| number of hhidpn | 173 | 173 | 173 | 173 | 173 | 173 |

clustered standard errors in brackets; *** p< 0.01; ** p< 0.05; * p< 0.1; Mundlak-equation includes time averages of wave dummies, respondent's new conditions, ADL, BMI, spouse's conditions, liquid and non liquid wealth. All specifications control for wave dummies, health and health risks, and socioeconomic variables of respondent and spouse; specification (2) additionally controls for the household's portfolio risk; specification (3) includes information on wills and death expenses. Other causes of death are: infectious diseases, accidents/suicides, and natural causes.

# Chapter 3

## The Effects of World War II on Economic and Health Outcomes across Europe*

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### 3.1 Introduction

The Second World War (World War II) was one of the major transformative events of the 20th century, with 39 million deaths in Europe alone. Large amounts of physical capital were destroyed through six years of ground battles and bombing. Many individuals were forced to abandon or give up their property without compensation and to move on to new lands. Periods of hunger became more common even in relatively prosperous Western Europe. Families were separated for long periods of time, and many children lost their fathers. Many, including young children, would personally witness the horrors of war as battles and bombing took place in the very areas where they lived. Horrendous crimes against humanity were committed. Due to World War II, political and economic systems in many countries would be permanently altered.

In this paper, we investigate long-run effects of World War II on late-life economic and health outcomes in Western continental Europe (health, education, labor market outcomes and marriage). We explore several channels through which this war might have influenced individual lives, and document which groups of the population were most affected. Our research relies on retrospective life data from the European Survey of Health, Aging, and Retirement in Europe (SHARE) that have recently become available. SHARE covers representative samples of the population aged 50 and over in 13 European countries, with about 20,000 observations. We also collected external data on casualties, timing and location of combat action, yearly GDP by country, population movements, and male-female population ratios. To our individual-level analysis of the multidimensional effects of a major shock that affected life circumstances, we add new dimensions to a rapidly increasing literature that aims at explaining the causes of health and wealth gradients in labor and health economics (see Deaton (2007), Smith (2009a), Heckman (2012)).

SHARE not only measures major contemporaneous economic and health outcomes of adults over age 50 in these European countries, but includes retrospective modules meant to capture salient parts of early life experiences, including those related to the war. There simply are no micro economic panel

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* This chapter is based on joint work with Iris Kesternich, James Smith, and Joachim Winter

data in either the United States or in Europe that have prospectively tracked people for that long a time period[1]. The co-existence of current prospective data combined with retrospective data on key events that preceded the survey baseline opens up important new research opportunities not before possible, and not simply those associated with the World War II. Since the end of World War II, Western continental Europe has had a rich and sometime tumultuous economic and political history, the effects of which on its residents are not well documented.

There is legitimate concern about the quality of recall data, particularly for time periods decades in the past. But that concern has been lessened by a realization that recall of events during childhood is better than for other periods of life, particularly if events are salient as they certainly are in this application. Smith (2009b) investigated several quality markers and showed that his childhood health instrument was successful in matching known secular trends in childhood illnesses decades in the past[2]. Moreover, we will provide evidence in this paper that these recalled events in the SHARE retrospective about the war matched the historical record.

One aim of the paper is to illustrate how such retrospective life data can further our understanding of effects of early-life conditions as affected by large external shocks, such as a war. The existing literature measuring impacts of macro-events mostly used "natural experiments" such as wars or famines to study effects of early-life conditions at the aggregate level. Largely due to data reasons beyond their control, the studies of which we are aware could not use individual-level measures of whether a particular person was affected by the war and through which channel. Retrospective life data, such as those from SHARE, contain detailed information and provide the opportunity of studying that issue.

Analyzing different outcomes is a first step in understanding the channels and mechanisms by which wars affect people's lives. Another possibility is using different measures of war exposure such as the closeness of combat. We construct such measures from external data sources. In addition, SHARE data contain retrospective questions on several possible channels of war exposure: hunger, the absence of the father, dispossession, and persecution.

Given the scale of the war and number of ways it fundamentally changed the world, the existing economic literature using World War II as a natural experiment is surprisingly thin. Moreover, the literature that does exist using World War II is relatively recent and more American in context than European. This may reflect the fact that the popularity of 'natural experiments' framework in economics itself post-dated World War II by many decades. Still, it does suggest that excellent research opportunities remain, especially given the wide diversity of European experiences in World

[1] PSID, the longest micro-economic panel, began in 1968 more than 20 years after World War II. The longest running European micro-economic panel, GSEOP, began in 1984, almost 40 years after the war.

[2] There was also no evidence of backwards attribution of new episodes of adult health problems into a revaluation of childhood health. Adult respondents whose health deteriorated between PSID waves were no more likely than before to say their childhood health was not good or to cite additional childhood health problems (Smith (2009b)).

War II.

This paper is divided into five sections. The next highlights the main attributes of SHARE data and the additional data we collected for this research. Section 3 sets the stage for our analysis by presenting evidence of possible changes on which long-term effects of World War II may operate. Section 4 summarizes statistical models that capture impacts of the experience of World War II on individual adult labor market, demographic, and health outcomes. We also present our models of the influence of the war on some of the primary pathways through which it had long lasting impacts – hunger, dispossession, the absence of a father, and marriage. The final section 5 highlights our main conclusions.

## 3.2 SHARELIFE Data

### 3.2.1 SHARE and Retrospective Early-life Data from SHARELIFE

SHARE is a multidisciplinary cross-national panel interview survey on health, socio-economic status, and social and family networks of individuals aged 50 or over in continental Europe. The original 2004/2005 SHARE baseline included nationally representative samples in 11 European countries (Denmark, Sweden, Austria, France, Germany, Switzerland, Belgium, Netherlands, Spain, Italy, and Greece) drawn from population registries, or from multi-stage sampling (http://www.share-project.org/). For these countries, a second wave of data collection took place in 2006, and the third wave of data collection on this panel (SHARELIFE) was completed in 2008.

In addition to a standard set of demographic attributes (age, marital status, education), SHARE data include health variables (self-reported health, health conditions, health behaviors), psychological variables (e.g., depression and well-being) economic variables (current work activity, sources and composition of current income, and net wealth (including housing, cars, and all financial assets (stocks, bonds, and cash) minus all debts)).

SHARE's third wave of data collection, SHARELIFE, collected detailed retrospective life-histories in 13 countries (Poland and the Czech Republic were added in wave 2) in 2008-09. SHARELIFE was based on life history calendar (LHC) methods. The interview starts with the names and birth dates of the respondent's children (and other information about them including any deaths), which is followed by a full partner and residential history. This information is used to aid in dating of all other events.

The information in the life history includes family composition and type of home (number of rooms, running water, toilet, etc), number of books, and occupation of father. These measures were used to create an index of childhood SES at age 10. A childhood health history is also included based on the Smith module included in the PSID and HRS that queries individual specific childhood diseases and

an overall subjective evaluation of childhood health status (Smith (2009b)). In addition, respondents are asked about childhood immunizations and hunger during childhood. Adult health histories and job and income histories were also collected. Moreover, SHARELIFE provides detailed data on within country region of residence and housing during the full life of respondents (childhood and adulthood).

### 3.2.2 Other Data Sources

In addition to SHARE data, we also use external data sources to identify aggregate channels of war-affectedness. Since World War II affected not only countries differentially, but also regions within countries, we constructed data on combat operations using sources from military history (Ellis (1994)). Using maps of within country regions for each month during the war, we documented whether armies engaged in battle in that place at that time. We combined these data with information about the region in which respondents lived during each year of World War II and use it as one measure of individual war exposure.

Since we analyze data over a time period of 50 years, we also have to account for country-specific economic performance that may have affected childhood circumstances differently. We therefore use GDP data, which are available for each European country (Maddison (2008)). We also used external data on country specific civilian and military causalities associated with World War II, population movements, and the sex ratio. Table 3.1 contains definitions of variables derived from SHARE and SHARELIFE that will be used in our analysis in this paper. Appendix table A.1 provides a parallel list of variables constructed from external data sources with a documentation of the source that was used.

## 3.3 The Channels of Long-term Effects of World War II

This section presents descriptive data and reviews the current literature on possible major channels through which World War II might have affected people's lives well into their older years. The channels include future per capita income growth of countries affected, mortality, changing sex ratios, absence of a father, periods of hunger, migration, dispossession, and persecution. This section is used to motivate the rationale for analyses pursued in section four.

### 3.3.1 Per Capita Income Growth

If wars alter long-term economic growth, they would permanently depress economic prospects of future generations. Warfare reduces capital stock through the destruction of infrastructure, productive capacity, and housing through bombing and fighting, and results in a relocation of food and other

production into military production. It obviously destroys human capital-but the real question for our analysis is will there be catch-up growth, or will the destruction show up many decades later? Based on Harrison (1998), table 3.2 displays GPD per capita of some of the major countries involved in the war relative to that of the US at key illustrative dates. The immediate impact of World War II was apparently quite destructive for the countries involved, especially so for those on the losing side-Germany, Japan, and Italy-presumably reflecting their much larger losses in both physical and human capital during the war. However, by 1973 and certainly by 1987, the European 'losers' actually had higher per capita growth than European 'winners'. What appears to be essential in the long-term was not whether a country was on the winning or losing side, but whether or not they transited to democracy and open-market economies[3]. The poor performance of USSR countries illustrates that point[4].

### 3.3.2 Mortality

In 1939, there were about 2 billion people in the world. The best estimates indicate that between 62 and 78 million of them would die due to World War II-more than 3% of the world's population. While earlier wars also resulted in deaths of civilians[5], civilians were particularly heavily affected by World War II with about half of the World War II European casualties being civilians. Among civilian deaths, between 9.8 and 10.4 million civilians were murdered for political or racial reasons by the Nazi regime (Auerbach (1992)). Deaths due to the war were very unequally distributed across countries, whether they were military deaths due to combat, civilian deaths, or the holocaust. The upper figure 3.1 displays the fraction of the 1939 population who died in a large array of affected countries. Among European countries covered by our data, Germany and Poland bore the brunt of these casualties. In contrast and for comparative purposes only, American causalities in the European and Asian theatres combined were a bit over 400,000, the overwhelming majority of whom were soldiers. Similarly, total deaths in the UK are estimated to be about 450,000, 15% of whom were civilians.

[3] Waldinger (2012) demonstrated one micro channel on human capital, the loss of Jewish university professors in Germany due to World War II. He shows that the productivity of those departments in Germany which lost a relatively high share of their professors was permanently lowered, while shocks to physical capital due to Allied bombing had returned to their old growth path by the 1960s. More generally, to make up for investments in human capital takes years, while plants and factories can be repaired and replaced much more quickly.

[4] A related issue is the impact of World War II on population growth in countries and affected cites. In spite of deaths of large numbers of civilians in World War II, the existing evidence indicates that affected cities went back to old population growths paths in Western Germany and Japan (Brackman *et al.* (2004)), while city growth, but also economic growth, was permanently depressed in East Germany and the Soviet Union (Acemoglu *et al.* (2011), Brackman *et al.* (2004)).

[5] For example in WWI there were 16 million total deaths of which almost 10 million were military deaths. Most of the civilian deaths in WWI were due to famine and disease.

The lower graph of figure 3.1 displays total number of deaths by type in the same countries. Deaths were highly concentrated in Germany and Poland where deaths measured around 5 million in both countries. In Germany, there were almost as many civilian deaths as military ones, while in Poland civilian deaths including the holocaust are by far the dominant ones. In many of the remaining countries in our data, deaths due to World War II are measured instead in the hundreds of thousands, but still often amount to a large fraction of the pre-war populations in several other countries, particularly Austria and the Netherlands. The other European countries that stand out are those that would comprise most of the Soviet Union, where one in seven perished in the war with about 10 million military deaths and 13 million civilian deaths. Unfortunately, data on these countries are not part of the SHARE network of European countries[6].

### 3.3.3 Sex Ratios and Absence of Father

Mostly men died during the war, producing low male/female ratios in Europe after the war as well as absence of many fathers during respondents' childhood years. Since the male bias in deaths was concentrated among soldiers as civilian and holocaust fatalities were largely gender neutral, it is countries in figure 3.1 who experienced many military deaths that were most affected. With 3 million military deaths, the most affected country in our data was Germany.

The top left-hand side of table 3.3 shows one immediate demographic consequence of the war by listing by country and period when one was age 10 the fraction of individuals who had a biological father absent when they were ten years old. Once again, the largest effects took place in the war-ravaged countries of Austria, Germany, and Poland. In Austria and Germany, about one in four children lived without their biological fathers when they were ten during the war. The legacy persists into years after the war since many who were age ten during 1950-1955 had fathers who died during the war. In Germany, almost a third of those age ten in these years were not living with their biological father. Absent father rates fall sharply in the post-1955 years since these children were born after the war. We observe war spikes in other countries as well (Italy, France, Denmark, and Belgium), but the contrasts with the pre- and post-war years are not as dramatic.

Sex ratios before, during, and after the war are contained in the bottom right half of table 3.3. In Germany, the sex ratio dropped from 0.96 in 1939 to 0.72 men per women in the 15-45 age group after the war in 1946. Thus, many women did not marry, and many children grew up without a father. Even after the war, about 4 of the 11 million German prisoners of war remained in captivity, and

[6] While we concentrate for data reasons primarily on the effects of World War II in continental Western Europe, the war's impact was just as stark in the Asian theatre. The two countries most directly affected in terms of number of causalities were Japan and China. About 2 million Japanese soldiers died in the war alongside up to a million Japanese civilians-about 4% of the pre-war Japanese population. The total number of deaths in China is believed to range between 10 and 20 million, with more than 70% being civilians.

the last 35,000 German soldiers returned from the Soviet Union in 1955 which further compounded the problem of absent fathers (Wehler (2008)).

### 3.3.4 Hunger

One channel by which World War II might have affected long-run adult health and SES outcomes is hunger. World War II caused several severe hunger crises which led to many casualties, and may have had long-term effects on the health of survivors. For example, since the beginning of the German occupation in Poland, the nutritional situation of the non-German population was poor. The average caloric intake for the Polish population was about 930 calories in 1941. The situation was worst in the Warsaw Ghetto where average food rations were limited to about 186 calories per day in 1941. Similarly, in the fall and winter 1941/1942, Greece was struck by a severe famine with about 100,000 to 200,000 deaths (Hionidou, 2006). In World War II, Greece was under Bulgarian, German, and Italian occupation. The famine was mainly caused by three factors: (1) occupiers imposed a naval blockade; (2) prices to farmers were fixed at such low levels that they were not willing to market their products; (3) mobility between different regions of the country was reduced due to occupation. The nutritional situation returned to acceptable levels towards the end of 1942. Neelsen and Stratman (2011) use Cohort Data to show that undernourishment of children who were 1 or 2 years old at the time of the famine had a significantly lower probability of being literate or to complete upper secondary education.

A combination of a food blockade and a harsh winter led to a severe hunger crisis in winter 1944/1945 in the Netherlands. About 20,000 deaths, mainly among elderly men, are attributed to this famine. The famine ended with the end of the German occupation in May 1945. The Dutch famine has been extensively studied because it affected an otherwise well-nourished population at a very specific time and region. Individuals exposed to this famine in utero are shown to suffer from cognitive and mental problems and addiction (Neugebauer *et al.* (1999), De Rooij *et al.* (2010)), diabetes and coronary heart disease, and they also perform worse regarding anthropometric and socio-economic indicators (Almond and Currie (2011)).

Germany suffered from hunger between 1945 and 1948 when the food supply from occupied countries ceased. The Office of Military Government for Germany established a goal of 1550 calories per day in 1945, but in the first months of occupation, this goal often could not be met. There were regions where average calories per day were around 700 (Gimbel (1968)). Death rates raised by the factor 4 for adults and 10 for infants during this period. With a good harvest and currency reform in June 1948, nutritional shortages were overcome (Zink (1957)).

Figure 3.2 demonstrates that hunger episodes during the war were much more severe in war countries than in those countries that did not participate in the war. We also see that there was a great amount of diversity in periods of hunger within war countries. Hunger is more common in regions

where combat took place within war countries. Finally and not surprisingly, the experience of hunger was far more common among those of low socio-economic background as a child. With respect to hunger, our analysis shows that the individual-level reports in SHARELIFE match well historical information on the timing and location of hunger episodes we collected from historic sources. To illustrate, in figure 2 the Greek hunger spike occurred in 1941-1942, the Dutch in 1944-45, and the German in 1946-1947.

### 3.3.5 Dispossession, Persecution, and Migration

SHARELIFE documents the extent of the experience of dispossession of property linked to World War II and its aftermaths. Dispossession was often associated with persecution and resulted in geographic displacement of populations during and immediately after the war. A further advantage of SHARELIFE is that we can observe where and when individuals moved during their lifetimes, including the wartime period.

There were three main periods when people were forced to flee their homelands. During World War II, millions of Jews, but also opponents of the Nazi regime, were expropriated, and often sent to concentration camps and died there. Second, the end of World War II was associated with dramatic border changes in Eastern Europe. These border changes induced millions of individuals to leave their places of residence and flee to other parts of Europe. The Soviet Union annexed territory from some of its neighboring countries, inter alia from Czechoslovakia, Germany, and Poland. Poland in turn received one part of pre-war Germany in compensation. Those Poles having lost their homes in the part occupied by the Soviet Union were moved to the new part, so Poland and with it millions of people were moved westwards.

Figure 3.3 shows inflows and outflows of populations during and after the end of World War II into the new states in their new borders. Germany lost about one quarter of its territory. About 2 million people have been estimated to have died on the flight. After the war, the remaining territory of Germany was divided into four occupational zones. About 4 million people fled from the approaching Soviet armies to the British and US zone where the occupation was less severe. In Germany, destroyed cities had to accumulate millions of ethnic Germans from other parts of Europe. A further wave of dispossessions happened in Eastern countries after World War II when private property was nationalized in the socialist and communist economies. Even in France, there was a wave of nationalizations at the end of World War II. Mainly banks, energy, and transport firms were nationalized, but there were also some expropriations which happened as penalty for cooperation with the Nazi regime.

The bottom left-hand side of table 3.3 displays dispossession rates in our SHARE countries by time period with the final column indicating the percent ever dispossessed. Figure 3.4 complements the data in table 3.3 by showing the percentage of dispossessed individuals in SHARELIFE for the

foreign and native-born populations. In the Czech Republic, Germany, and Poland more than 5% of respondents experienced dispossession during their lifetime. For respondents living in Germany and Poland, dispossession happened more frequently during the war period, while they happened after the war in Czechoslovakia. Dispossessed individuals in our sample are over proportionally born outside of the current borders of their country. Analyzing countries of origin, many of them came from Eastern Europe, thus they most probably lost their property with the big wave of nationalizations after World War II. Not surprisingly, it is the foreign-born living in our SHARE countries that were most likely to be dispossessed.

## 3.4 World War II and Individual Outcomes: Analysis of SHARELIFE Data

Based on the descriptive data and review in the prior section, we find enormous variation even among war countries in the immediate impact of World War II. Long-term economic or population growth rates seem unlikely to be a primary pathway through which the war's influence took place. Instead, changing gender ratios induced by differential male mortality in the war appear to be a more plausible pathway operating both through absence of fathers and difficulties faced by women in marrying. Hunger and immediate and long-term stress created by battles, dispossession, and persecution would also appear to be plausible pathways that could impact adult health, both physical and mental, and our later life measures of adult SES.

### 3.4.1 Measures of War Exposure

To analyze long-term impacts of World War II on health and economic outcomes, we use the fact that different countries in Europe and different people in those counties were differentially affected by World War II at different points in time. To study effects on adult outcomes, we use two indicators of being affected by World War II: (a) that one lived in a war country during the war period, and (b) that one was exposed to combat in the area within a country in which one lived during the war. Our first measure essentially creates a war dummy equal to zero for everybody in a non-war country (Denmark, Switzerland, and Sweden), and for everybody born after the war period no matter what country they lived in. Alternatively, it is equal to one for everybody alive in a war country (Austria, Belgium, Czech Republic, France, Germany, Greece, Netherlands, and Poland) during the war period. The war period ends in 1945 for all war countries, while it includes 1946 to 1948 in Germany and Austria, when they were under allied occupation. For these countries, the war period ended with the currency reform in Germany in 1948. Individuals could certainly have been affected by the war even if they were born after the war, but the channels we emphasize in this paper-combat, hunger, dispossession, persecution, and the absence of a father-were more likely to have affected those who

lived during the war.

Our second war measure involves constructing a variable indicating whether there were combats and how many combats occurred in the region within the country in which the individual lived during World War II. Thus, in the war countries, we create two dummy variables based on the number of months of exposure the respondent had to combat in the place they lived during the war-0 to 2 months of exposure to combat and 3 or more months of exposure to combat. The purpose of this variable was to test whether actual exposure to combat was an important mechanism for the war effects that we estimate below.

Table 3.4 provides the list of SHARE countries that are part of our analysis with the sample sizes of those SHARELIFE respondents who experienced the war and those who had no direct experience of war. We did not include Spain in our analysis since Spain experienced a civil war in the late 1930s, so a distinction between whether Spain is a war country or not is very ambiguous. The results were not significantly different if Spain was included.

### 3.4.2 Micro-level Regressions of Adult Health and SES Outcomes

We next turn to our statistical modeling of whether individuals' experiences during World War II predict their health and socio-economic status in their later adult life. For all of our later-life health and SES outcomes and channel outcomes, our estimating equation takes the form

$$Y_{itc} = \beta_1 * war_{itc} + \beta_2 * male_i + \lambda_t + \eta_c + \epsilon_{itc} \quad (1)$$

where $Y_{itc}$ is the late-life outcome of respondent $i$ born in year $t$ and living in country $c$. Male indicates a respondent was male. War is one of our two measures of war exposure outlined above, which vary by country (or region within a country) and year of birth. Because there may be unmeasured country and year effects associated with these outcomes, $\lambda_t$ is a full set of year of birth dummies and $\eta_c$ is a full set of country dummies. $\epsilon_{itc}$ is a random error term. Since error terms within country and within year may be correlated, we used the cluster option in STATA.

Our principal interest is to obtain estimates of $\beta_1$–the "war" effect in addition to birth-year and country effects. We estimate reduced form models using our two war variables on latter adult life health and SES outcomes and the principal channels of war. We consider several adult dependent variables all measured in 2009, the year of SHARELIFE. Health outcomes include prevalence of diagnosed diabetes and heart disease, body height in centimeters (a summary measure of early-life health conditions), whether an individual is depressed using a dummy variable for presence of at least four symptoms on the EURO-D scale, and self-reported health status. Self-reported health status is recorded on a scale excellent, very good, good, fair, and poor which we have translated to a scale from one to five with five the best health status. Our adult SES and economic outcomes include log of household net worth, whether the individual was ever married, and life-satisfaction in 2009. SHARE respondents are asked *"On a scale from 0 to 10 where 0 means completely dissatisfied and*

*10 means completely satisfied, how satisfied are you with your life?"* which we model as a score from 0-10.

We have two education measures in SHARE. The first is obtained from baseline SHARE in 2004 and, in an attempt to make the education variable comparable between individuals in the same country, assigns a standardized year for each education value. For example, university graduates in a country would be assigned a 16. The second education variable is available in the second SHARE wave and is equivalent to the actual number of years spent in education. We use the second measure because Poland and the Czech Republic were not part of baseline SHARE and for those two countries the first measure is not available. However, we hypothesize that World War II may have disrupted education for many respondents and resulted in a longer time to complete a given level of education. To test that hypothesis for the sub-sample of respondents who have both measures of education from the second and first SHARE waves, we estimated a model that amounts to the difference between the two education measures (the second-wave education minus the first-wave education variable).

Figure 3.5 displays the association of three of our key outcomes-education, self-reported health, and depression-with time period of birth using three sub-sets of countries-Germany and Austria combined, other war countries, and the non-war countries. These outcomes are each expressed as the difference between each of the first two kinds of war countries minus the outcome in the non-war countries. For all three outcomes, the outcomes deteriorate relative to the non-war countries for those born at a time they would experience war.

Table 3.5 summarizes results obtained for adult health outcomes and table 6 for adult SES outcomes. We present regressions in the A panels that use only the aggregate war exposure measures and in panel B the measure that distinguishes between very limited exposure to combat (two months or less, including zero) or an more extensive combat exposure (three or more months) with the left-out category being not exposed to war at all. In terms of right-hand side variables, there are no missing values for gender. If the outcome in any particular model is missing, this observation was not included in that specific model. Missing values in our outcomes are relatively rare. In terms of main channels (dad absence, dispossession, hunger, and persecution), missing values are in the order of one in a thousand observations.

Consistent with the literature, men have higher levels of adult diabetes and heart disease, lower levels of depression, and report themselves in better subjective health than woman do (Banks *et al.* (2010), Smith (2007)). Our principal concern involves estimates for aggregate war and combat variables. Living in a war country during the period of World War II is consistently statistically significantly associated with higher levels of adult diabetes, being more depressed, and reporting one's subjective health as worse. Being in a war country during the war increased the probability of diabetes in later life by 2.6 percentage points and depression by 5.8 percentage points while decreasing self-reported health by 9.4 percentage points. These increases are all high relative to baseline rates (Appendix table A.2). Estimated effects on heart disease and height are not statistically significant.

The B panel of table 3.5 displays results for months of combat exposure variables-number of months of exposure respondents had to combat in the place they lived during the war in war countries using 0-2 months of exposure to combat and 3 or more months of exposure to combat. These results basically parallel those obtained for the war variable in both direction and magnitude-those with combat exposure were more likely to have diabetes as an adult, were in worse self-reported health, and were more likely to be depressed. The results are weaker for heart problem although 3 or more months of combat exposure increases the likelihood of heart disease as an adult and is statistically significant at the 10% level.

Table 3.6 repeats the same type of models for adult economic outcomes in 2009. Not surprisingly for these generations, compared to women men achieve more years of schooling, have higher net worth, are less likely to marry, and have higher levels of life satisfaction-common findings in the literature. Our measure of war exposure is strongly associated with all these SES outcomes, except log net worth. Those in a war country during the war achieved about three-tenths of a year less education[7] and achieved lower levels of life satisfaction (about a third of a point lower relative to a mean of 7.6) as older adults. The education difference model suggests that war makes respondents take longer (a third of a year) to reach a given level of education. Similarly, this exposure to war reduced the probability of women being ever married (about three percentage points) but not the marriage probability for men, consistent with the relative scarcity of men due to war. In contrast, the log of household net worth is not associated with the wartime experience suggesting that this outcome mainly depends on post-war savings behavior and trends in asset prices. The war combat models in the B panel of table 3.6 produce roughly similar results in direction and magnitude of these outcomes.

One purpose of our combat variables was to test whether the actual exposure to combat was an important mechanism for the war effects that we estimate above. With the sole exceptions of adult depression (table 3.5) and live satisfaction (table 3.6), the estimated magnitude of the worse adult

[7] Ichino and Winter-Ebmer (2004) compare educational outcomes from cohorts affected by the war in Austria and Germany to cohorts in Switzerland and Sweden, using the main economic datasets with information on education and earnings in the countries. They find that the loss of schooling is about a fifth of a year compared to the following cohort. They suggest that the mechanisms are closing down of religious schools, absence of teachers due to the war, absence of students due to escaping bombing, and actual destruction of schools. Akbulut-Yuksel (2009) uses GSOEP to identify effects of destruction of German cities through bombing on schooling. She finds that destruction caused children to attain 0.4 fewer years of schooling. Her estimates suggest that this schooling reduction is mainly due to physical destruction of schools and the absence of teachers. Juerges (2013) uses the Micro-Census to analyze impacts of nutritional shortages in Germany on educational outcomes. He estimates a drop in educational achievements (having more than basic education) of about 5 percentage points for a baseline risk of about 30%. His suggested pathway is nutritional deprivation in utero. Our result of about a third of a year of schooling lies between the estimates of Ichino and Winter-Ebmer (2004) and Akbulut-Yuksel (2009), but are lower than those of Juerges (2013). However, we include a different set of countries, and not all of them were equally strongly affected by World War II as Germany was.

SES and health outcomes appear to be about the same amongst those with large or small exposures to actual combat[8]. This suggests that experiencing combat and battles close by to where you lived during the war are not the primary mechanisms by which these war effects operate. The exceptions are of interest since it seems reasonable that frequent exposure to combat is associated with adult depression and lower levels of life satisfaction as the vivid memories of that experience persist into adulthood.

### 3.4.3 Selection Effects

As in any such analysis, there are issues of possible selection effects due to fertility, mortality, and migration that may have biased our estimates. The concern with selective fertility is that high-SES mothers reduced their fertility more during the war, which on average would lead to less healthy babies. SHARE does not contain variables on education of parents so we used instead our measure of childhood SES, acknowledging its possible endogeneity. We examined fertility in SHARE in three time periods by SES-pre-war (before 1939), during the war (1939-1945), and post-war (>1945). Table 3.7 displays the mean number of children per woman for the three time periods and by childhood SES which was split at the median.

In all three periods, fertility is highest in the low-SES groups. But differential changes by SES in fertility across these three time periods do not seem large enough to be producing our results. Comparing pre-war and during-war periods, there was about 0.14 increase in fertility for both low-SES and high-SES groups. Similarly, comparing post-war to during-war periods, average fertility rose by about one child in both SES groups. Moreover, when we added childhood SES measures to our models, which should partly control for any selective fertility associated with the war, our estimates of the long-term effects of war did not change much. Individuals in our analytical sample are those still alive in 2009 so they are a selected sample of the population that experienced World War II. To the extent that those more affected are less likely to have survived, our results should understate the full effects of war on long-term health and SES outcomes.

A more complicated issue concerns differential mortality by SES induced by the war. If mortality due to the war was much higher in low-SES groups (whose health would have been worse anyway), we would further understate health effects of war. We examined data on age of death of father by SES by whether one lived in a war or non-war countries, and by whether you experienced the war as a child (born before 1946). The results are displayed in table 3.8. Once again, dividing SES at the median we found the following for the mean age of death of father. Those born after 1946 who did die should be younger but the key comparison is differentials by SES. For non-war countries, we find that in comparing pre- and post-1945 that the age of death of father decreased by 0.8 of a year

[8] For the two exceptions-depression and life satisfaction-the effect of 3 or more months of combat greater than 0-2 months and is statistically significant at the 1% level.

in both low and high SES groups. Using the same comparison, the age of father fell by 0.4 of a year in the war countries, but this was approximately the same for the low and high SES groups. Once again, this degree of selection does not seem large enough to be driving our results.

Because of population shifts, especially inflows documented in figure 3.3, we confined our analysis to the native-born in each country. Among countries in our data, figure 3.3 shows that outflows were significant only in Czech Republic, Poland, and Germany. Since it was not encouraged by receiving countries, migration during and after the War was quite difficult in Europe. But there was some migration and one must allow for the possibility that selective migration may influence our estimates on war effects especially for these three countries. Of course, people could have temporarily left combat areas as combat was taking place but stayed inside the same country, which should lead to an understatement of combat effects.

### 3.4.4 Models of Channels of War

We next turn to our estimates of how the micro pathway channels we highlighted above-hunger, dispossession, persecution, and the absence of father-are related to the experience of World War II. SHARELIFE respondents were asked *"(Looking back at your life), was there a period when you suffered from hunger?"* If the answer was yes, they were asked when this occurred. Individuals in SHARE are also asked "whether they or their family were ever dispossessed of any property as a result of war or persecution", and if yes the date of that dispossession. They were also asked whether they had ever been victims of persecution because of their political beliefs, religion, nationality, ethnicity, sexual orientation, or their background. Unfortunately, no time period for that persecution was asked. Finally, the absence of the father is defined as the absence of the biological father at the age of 10. About 8% of our respondents experienced a period of hunger, 9% have lived without their father at age 10, and 5% suffered from persecution and dispossession, respectively. We also included in our models an additional possible pathway-whether a respondent received immunizations as a child.

Table 3.9.A shows how micro channels are related to the experience of war. Males are both more likely to suffer from hunger and to be persecuted. The latter is what we expected given that mainly men were politically active during this period of time. Having experienced World War II increases the likelihood of experiencing hunger by about eight percentage points, dispossession and persecution by one percentage point, the absence of a father by two percentage points[9]. These estimates are

[9] Regarding the influence of hunger on late-life outcomes, we do not present structural estimates of the influence on late-life outcomes as there are no suitable instruments for the whole of Europe. These types of estimations are possible for a smaller set of countries. Van den Berg *et al.* (2012) use hunger periods caused by World War II for Greece, Germany, and the Netherlands as instrument to establish causal effects of under-nutrition on hypertension and adult height. For Germany, we collected data on monthly caloric rations in regions where respondents live. We see large drops in calories towards the end of the war and in occupation zones with the French and Soviet zone hit

large relative increases given baseline risks. For example, the probability of experiencing hunger is doubled by war exposure and the probability of an absent dad is increased by 25% in relative terms. The experience of war was associated with a lower probability of immunization as a child which is unsurprising given that this was wartime. This immunization result may be a pathway through which adult health eventually suffers.

In table 3.9.B our interest lies in whether the experience of combat is the mechanism that leads to war effects. Once again, the size of these estimates is very similar to those obtained by the country wartime variable. The experience of hunger and absence of the father is somewhat stronger for our respondents who lived in a region strongly affected by combat (3-10 months of combat) than for those in regions with none or only mild experience of combat. However, differences are not large. In fact, we expect the death of men during wartime to not necessarily happen in their region of residence. Persecution is related to war per se, but not necessarily to an increased experience of combat. Thus, combat does come with an increased likelihood of hunger as, for example, was the case in the Dutch hunger winter. It can be due to other aspects of war, as was the case for the Greek and German experience with hunger during World War II. Also, combat led to local deaths of the civilian population, but military casualties and the deaths of fathers often occur far from the families affected by it.

### 3.4.5 The Uneven Consequences of War

In addition to models summarized above, we investigated whether consequences of experiencing World War II vary by respondents' socio-economic status (SES) as a child by estimating models that included interactions of War variables with childhood SES. Childhood SES is an index generated by factor analysis (Mazzonna (2011). SES unifies four measures for SES during childhood at age 10. The variables are: logged proportion of number of room in the household and persons living in that household; logged number of books in the household; features in the household, namely warm water, cold water, fixed bath, toilet inside, central heating; and occupation of main breadwinner. For our analysis, we divide childhood SES status into three terciles and label those terciles low, middle, and high.

We separate our analysis of distributional consequences of World War II from our main analysis above since we recognize that childhood SES may partly be endogenous to World War II. Given the

hardest. When we regress our health outcomes on average calories available between age 0 and 16 in respondents' region (again controlling for gender and year of birth), we find that an increase of 1000 kcal per day decreases the chance of suffering from diabetes by 14.3 percentage points, increases SRH by 0.7 points, and increases height by 3.3 centimeters. When we distinguish different age groups (0-4, 5-10, and 11-16), we see strongest results for 0-4 group and impacts on adult depression. This suggests that hunger analysis should not only be seen as operating through nutrition-related outcomes such as adult height, but also and equally through adult outcomes such as depression. Our effects on height are similar to Van den Berg *et al.* (2012) who find an effect of between 3 and 6 centimeters.

destructive scale of World War II that included bombing that sometimes destroyed civilian homes and movement of men into the military, the possibility of such endogeneity is clearly an important caveat to keep in mind. We did re-estimate all models in tables 5-7 with these dummy variables for childhood SES terciles included and our estimates of the war barely changed.

Our distributional results are contained in table 3.10. All models continue to contain country and year of birth dummies and a dummy for male. We include both main effects for experiencing war and for childhood SES being either low or middle class. To identify distributional effects of war, we include a full set of interactions of the war with childhood SES. Once again, the results obtained are very similar whether we use the war country variable or our combat variable so table 3.10 only displays the results for the war variable. The outcomes modeled are the same as those in tables 3.5, 3.6, and 3.9-adult health, adult SES, and channels of war.

We first discuss main effects of childhood SES. Compared to those in the high childhood SES group, those in the lowest one have higher levels of adult diabetes (3.2 percentage points), are smaller in stature as adults (1.8 centimeters), experience higher levels of adult depression (2.5 percentage points), and self-rate themselves in worse adult subjective health. The middle childhood SES group consistently lies between the bottom and top in terms of these adult health outcomes. These results conform to the general finding in the literature that childhood economic circumstances are very predictive of later-life adult economic and health outcomes (Currie (2009), Case *et al.* (2002), and Smith (2009b)). Similarly, in accordance with the literature, higher childhood SES is associated with much higher levels of adult education, net worth, and life satisfaction, another indication of the strong economic transmission across generations in these outcomes. The more novel results are in the third panel of table 3.10 which deals with the channels of war. The probability of being dispossessed was highest in the high childhood SES group, not surprising as there was more to capture. Persecution was also highest in the high SES category, while obtaining childhood immunizations was highest in the lowest SES category. Absent fathers were not strongly differentiated across SES categories.

Finally, we examine differences in associations with war by childhood SES categories. For childhood SES by World War II interactions among the health variables, we find the negative health effects to be either neutral by SES categories or that the negative health effects are concentrated on the middle class as in the summary measure of self-reported health or concentrated in the middle and lower class as with heart disease, possibly reflecting the role of lifetime stress with that disease.

In contrast, we find very strong interactions of a negative middle class war interaction for many of our adult SES outcomes-education, and log net worth. Life satisfaction decrements associated with the war were concentrated on the lower and middle class. In terms of being ever married, the negative effects of the war were highest on the highest SES women and the lowest SES men. A summary of health and SES outcomes does suggest that the middle class suffered more due to the war with the lower class next in line. Finally, the length of time it takes to achieve a given level of education due to war expands the most for the low and middle class compared to the upper class.

The bottom panel of table 3.10 shows that some pathways through which war operates are concentrated among the poorest households (hunger and immunizations (present for the middle class), some are concentrated among middle class (dad absent), or the highest SES households (dispossession). Persecution was focused on the middle and upper classes.

## 3.5 Conclusion

In this paper, we present a micro analysis of effects of World War II on some key SES and health outcomes of those experiencing the war. To conduct this analysis we use new data-SHARELIFE-that records not only adult outcomes in 2009, but also contain retrospective data for salient aspects of the wartime experiences of respondents. We augment these data with historical information on how World War II affected individuals differently over time and across regions. Our data allow us to analyze which type of individuals were most affected, and by which channels.

Our analysis shows that experiencing war increased the probability of suffering from diabetes, depression, and with less certainty heart disease so that those experiencing war or combat have significantly lower self-rated health as adults. Experiencing war is also associated with less education and life satisfaction, and decreases the probability of ever being married for women, while increasing it for men. We also analyze pathways through which these wartime effects took place and found strong effects for hunger, dispossession, persecution, childhood immunizations, and having an absent father. While a war of the magnitude of World War II affected all social classes to some degree, our evidence does suggest that the more severe effects were on the middle class with the lower class right below them in size of impact.

This paper highlights advantages of having life-histories in prospective studies such as SHARE. Population-based economic panels are relatively recent, but combining them with life-histories covering salient past personal and macro events opens up many new research opportunities of which World War II is only one illustration. This is especially so in Western Europe where the political and economic history of the past four decades is particularly rich and varied.

## Tables and Figures

Table 3.1: Variable definition

| variable name | definition |
|---|---|
| **background information** | |
| year of birth | year of birth |
| male | dummy = 1 if respondent is male |
| childhood SES | unifies four measures for SES at age 10: logged number of books in household; logged number of rooms and persons in household; features in household; occupation of main breadwinner |
| **outcome measures** | |
| childhood immunizations | dummy = 1 if respondent got any vaccinations during childhood |
| depression | dummy = 1 if respondent suffers from more than three depression symptoms in EURO-D scale |
| diabetes | dummy = 1 if respondents has diabetes or high blood sugar |
| ever married | dummy = 1 if respondents was ever married |
| heart disease | dummy = 1 if respondent has heart problems (including heart attack) |
| height | height in cm |
| life satisfaction | life satisfaction on a scale from 0-10 with 0 very unsatisfied and 10 very satisfied |
| log(net worth) | logged household net worth as the sum of values from bank accounts, bonds, stocks, mutual funds, retirement accounts, contractual savings and life insurances minus liabilities |
| self-rated-health | categorical variable for self-rated health with excellent health = 5 |
| years of education | years of education |
| **channels of war exposure** | |
| dad absent | dummy = 1 if biological father was absent at the age of 10 |
| dispossession | dummy = 1 if respondent reports ever being dispossessed |
| hunger | dummy = 1 if respondent ever suffered hunger and when |
| persecution | dummy = 1 if respondent reports ever being persecuted |
| **war variables** | |
| war | dummy = 1 if respondent was living in a war country during the war period |
| war combat 0-2 months | respondent was living in a war country during the war period in a region within the country that experienced 0-2 months of combat |
| war combat 3-10 months | respondent was living in a war country during the war period in a region within the country that experienced 3-10 months of combat |

Table 3.2: GDP per head relative to US GDP per head

| **country** | **1938** | **1950** | **1973** | **1987** |
|---|---|---|---|---|
| UK | .98 | .72 | .72 | .73 |
| Germany | .84 | .45 | .79 | .82 |
| France | .72 | .55 | .78 | .78 |
| Italy | .53 | .36 | .63 | .70 |
| Japan | .38 | .20 | .66 | .77 |
| USSR | .35 | .30 | .36 | .33 |

Source: Harrison (1998), Table 1-10.

Table 3.3: Percentage of SHARELIFE respondents with father absent at age 10; by time period

| **country** | **<1939** | **1939-45** | **1945-4** | **1950-55** | **>1955** | **<1939** | **1939-45** | **1945-49** | **1950-55** | **>1955** |
|---|---|---|---|---|---|---|---|---|---|---|
| | **% father absent** | | | | | **% hunger** | | | | |
| Austria | 0.195 | 0.256 | 0.274 | 0.221 | 0.152 | 0.054 | 0.116 | 0.078 | 0.026 | 0.015 |
| Germany | 0.113 | 0.191 | 0.223 | 0.297 | 0.099 | 0.006 | 0.211 | 0.162 | 0.029 | 0.016 |
| Sweden | 0.113 | 0.126 | 0.092 | 0.140 | 0.123 | 0.006 | 0.008 | 0.005 | 0.005 | 0.018 |
| Netherlands | 0.074 | 0.083 | 0.102 | 0.073 | 0.044 | 0.005 | 0.116 | 0.008 | 0.001 | 0.003 |
| Italy | 0.044 | 0.103 | 0.068 | 0.069 | 0.046 | 0.032 | 0.123 | 0.053 | 0.027 | 0.020 |
| France | 0.106 | 0.149 | 0.158 | 0.128 | 0.056 | 0.008 | 0.117 | 0.026 | 0.014 | 0.026 |
| Denmark | 0.073 | 0.092 | 0.118 | 0.103 | 0.076 | 0.000 | 0.004 | 0.001 | 0.001 | 0.007 |
| Greece | 0.019 | 0.024 | 0.042 | 0.049 | 0.013 | 0.032 | 0.123 | 0.046 | 0.027 | 0.014 |
| Switzerland | 0.067 | 0.054 | 0.085 | 0.030 | 0.046 | 0.014 | 0.013 | 0.008 | 0.007 | 0.013 |
| Belgium | 0.046 | 0.083 | 0.065 | 0.086 | 0.055 | 0.004 | 0.070 | 0.012 | 0.006 | 0.014 |
| Czechia | 0.080 | 0.063 | 0.134 | 0.095 | 0.081 | 0.012 | 0.038 | 0.021 | 0.006 | 0.004 |
| Poland | 0.078 | 0.115 | 0.173 | 0.154 | 0.082 | 0.015 | 0.123 | 0.054 | 0.018 | 0.033 |

| | **% dispossession** | | | | | **% ever dispossessed** | **sex ratio** | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | <1939 | >1945 | 1945-48 |
| Austria | 0.004 | 0.015 | 0.003 | 0.000 | 0.000 | 0.029 | 0.95 | 0.86 | - |
| Germany | 0.004 | 0.039 | 0.011 | 0.007 | 0.006 | 0.064 | 0.96 | 0.81 | 0.72 |
| Sweden | 0.000 | 0.002 | 0.000 | 0.000 | 0.001 | 0.003 | 0.99 | 1.02 | 1.02 |
| Netherlands | 0.002 | 0.019 | 0.000 | 0.000 | 0.000 | 0.015 | 0.99 | 0.97 | - |
| Italy | 0.001 | 0.004 | 0.000 | 0.000 | 0.000 | 0.004 | 0.94 | 0.95 | 0.94 |
| France | 0.003 | 0.024 | 0.001 | 0.001 | 0.006 | 0.032 | 0.96 | 1.02 | - |
| Denmark | 0.000 | 0.003 | 0.000 | 0.000 | 0.001 | 0.004 | 0.95 | 0.99 | - |
| Greece | 0.014 | 0.011 | 0.001 | 0.000 | 0.001 | 0.014 | 0.92 | 0.99 | - |
| Switzerland | 0.006 | 0.002 | 0.000 | 0.001 | 0.003 | 0.011 | 0.96 | 0.99 | 0.98 |
| Belgium | 0.007 | 0.035 | 0.003 | 0.000 | 0.002 | 0.036 | 0.99 | 1.02 | - |
| Czechia | 0.018 | 0.012 | 0.020 | 0.078 | 0.023 | 0.136 | 0.95 | 0.97 | 0.97 |
| Poland | 0.002 | 0.058 | 0.008 | 0.001 | 0.003 | 0.065 | 0.89 | - | - |

Note: the countries are defined as of 2009 when SHARELIFE data were collected. 1-year refers to year age 10; source: SHARELIFE; calculations by authors. For sex ratios, see Appendix table A.

Table 3.4: Number of observations available in SHARELIFE; by country

| | observations | | |
|---|---|---|---|
| **country** | war = 0 | war = 1 | total |
| Austria | 146 | 565 | 711 |
| Germany | 450 | 1,001 | 1,451 |
| Italy | 863 | 1,470 | 2,333 |
| Czechia | 723 | 925 | 1,648 |
| Greece | 1,149 | 1,482 | 2,631 |
| Poland | 819 | 758 | 1,577 |
| Belgium | 1,026 | 1,480 | 2,506 |
| France | 793 | 1,105 | 1,898 |
| Netherlands | 883 | 1,069 | 1,952 |
| Denmark | 1,927 | 0 | 1,927 |
| Sweden | 1,639 | 0 | 1,639 |
| Switzerland | 993 | 0 | 993 |
| Total | 11,411 | 9,855 | 21,266 |

Note: The countries are defined as of 2009 when the SHARELIFE data were collected. Native born only; war = 1 means that the respondent was living in a war country sometime during World War II; source: SHARELIFE; calculations by authors.

Table 3.5: Adult health outcomes associated with World War II

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| **VARIABLES** | **diabetes** | **heart** | **height** | **depression** | **self-reported health** |
| **A. war variable** | | | | | |
| war | 0.026*** | 0.014 | 0.196 | 0.058*** | -0.094*** |
| | [0.009] | [0.010] | [0.179] | [0.014] | [0.034] |
| male | 0.010** | 0.045*** | 11.579*** | -0.170*** | 0.115*** |
| | [0.004] | [0.004] | [0.106] | [0.006] | [0.015] |
| observations | 21,228 | 21,228 | 21,115 | 21,266 | 21,254 |
| R-squared | 0.022 | 0.061 | 0.510 | 0.073 | 0.148 |
| **B. combat variable** | | | | | |
| war combat 0-2 months | 0.030*** | 0.011 | 0.138 | 0.047*** | -0.091*** |
| | [0.009] | [0.010] | [0.205] | [0.015] | [0.036] |
| war combat 3 or more months | 0.018* | 0.018* | 0.308 | 0.078*** | -0.098*** |
| | [0.010] | [0.012] | [0.221] | [0.016] | [0.037] |
| male | 0.010** | 0.045*** | 11.576*** | -0.169*** | 0.114*** |
| | [0.004] | [0.005] | [0.086] | [0.006] | [0.015] |
| observations | 21,212 | 21,212 | 21,099 | 21,250 | 21,238 |
| R-squared | 0.022 | 0.061 | 0.510 | 0.073 | 0.148 |

Robust standard errors in brackets allow for correlation year/birth level; ***p<0.01, **p<0.05, *p<0.1; OLS regressions include both country dummies and birth year dummies.

Table 3.6: Adult SES outcomes associated with World War II

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| **VARIABLES** | **education** | **ed Diff** | **ln net worth** | **ever married** | **life satisfaction** |
| **A. war variable** | | | | | |
| war | -0.274** | 0.362*** | 0.076 | -0.032*** | -0.306*** |
| | [0.127] | [0.126] | [0.057] | [0.008] | [0.051] |
| male | 0.964*** | -0.084 | 0.224*** | -0.017*** | 0.212*** |
| | [0.060] | [0.056] | [0.022] | [0.005] | [0.025] |
| currently married | | | 0.859*** | | |
| | | | [0.045] | | |
| war×male | | | | 0.024*** | |
| | | | | [0.007] | |
| observations | 19,572 | 11,355 | 20,341 | 21,266 | 19,549 |
| R-squared | 0.276 | 0.145 | 0.356 | 0.011 | 0.128 |
| **B. combat variable** | | | | | |
| war combat 0-2 months | -0.215 | 0.406*** | 0.097* | -0.030*** | -0.242*** |
| | [0.134] | [0.129] | [0.058] | [0.008] | [0.054] |
| war combat 3 or more months | -0.398*** | 0.264** | 0.029 | -0.036*** | -0.436*** |
| | [0.134] | [0.154] | [0.065] | [0.009] | [0.058] |
| war combat 0-2 months×male | | | | 0.029*** | |
| | | | | [0.008] | |
| war combat 3 or more months×male | | | | 0.020*** | |
| | | | | [0.007] | |
| currently married | | | 0.885*** | | |
| | | | [0.041] | | |
| male | 0.965*** | -0.085 | 0.223*** | -0.017*** | 0.211*** |
| | [0.060] | [0.056] | [0.022] | [0.005] | [0.025] |
| observations | 19,556 | 11,347 | 20,327 | 21,250 | 19,533 |
| R-squared | 0.276 | 0.145 | 0.333 | 0.011 | 0.129 |

Robust standard errors in brackets allow for correlation year/birth level; ***p<0.01, **p<0.05, *p<0.1; OLS regressions include both country dummies and birth year dummies.

Table 3.7: Selective fertility by socioeconomic status during childhood

| **mean number of children per women** | | | |
|---|---|---|---|
| Median SES split | pre-war | during war | post-war |
| low SES | 1.28 | 1.43 | 2.47 |
| high SES | 1.11 | 1.251 | 2.25 |

Table 3.8: Differential mortality by socioeconomic status during childhood, before/after World War II

| **age of death of fathers** | | | | |
|---|---|---|---|---|
| Country | low SES | | high SES | |
| | before 1946 | after 1945 | before 1946 | after 1946 |
| non-war | 73.76 | 72.89 | 73.73 | 72.95 |
| war | 71.22 | 70.87 | 70.67 | 70.36 |

Table 3.9: Channels of war associated with World War II

| VARIABLES | (1) hunger | (2) dispossession | (3) persecution | (4) dad absent | (5) childhood immunizations |
|---|---|---|---|---|---|
| **A. war variable** | | | | | |
| war | 0.077*** | 0.013*** | 0.014** | 0.022** | -0.044*** |
| | [0.011] | [0.005] | [0.005] | [0.009] | [0.007] |
| male | 0.014*** | 0.004 | 0.010*** | -0.003 | 0.001 |
| | [0.003] | [0.003] | [0.003] | [0.004] | [0.003] |
| observations | 21,240 | 21,239 | 21,240 | 20,906 | 21,005 |
| R-squared | 0.077 | 0.047 | 0.020 | 0.032 | 0.079 |
| **B. combat variable** | | | | | |
| war combat 0-2 months | 0.071*** | 0.013*** | 0.017*** | 0.017* | -0.044*** |
| | [0.012] | [0.005] | [0.005] | [0.009] | [0.006] |
| war combat 3 or more months | 0.091*** | 0.012** | 0.008 | 0.033*** | -0.043*** |
| | [0.012] | [0.005] | [0.006] | [0.010] | [0.006] |
| male | 0.013*** | 0.004 | 0.010*** | -0.003 | 0.001 |
| | [0.003] | [0.003] | [0.003] | [0.004] | [0.003] |
| observations | 21,225 | 21,225 | 21,225 | 20,892 | 20,993 |
| R-squared | 0.077 | 0.047 | 0.020 | 0.033 | 0.079 |

Robust standard errors in brackets allow for correlation year/birth level; ***p<0.01, **p<0.05, *p<0.1.; OLS regressions include both country dummies and birth year dummies.

Table 3.10: War interaction with childhood SES models

| | **health outcomes** | | | | |
|---|---|---|---|---|---|
| | diabetes | heart | height | depression | self-reported health |
| war | 0.021** | -0.006 | 0.263 | 0.045** | -0.057 |
| SES low | 0.032*** | 0.007 | -1.835*** | 0.025** | -0.342*** |
| SES middle | 0.014*** | 0.005 | -0.852*** | 0.002 | -0.137*** |
| war×SES low | 0.003 | 0.028** | 0.123 | 0.014 | 0.032 |
| war×SES middle | 0.010 | 0.024** | -0.118 | 0.017 | -0.098*** |

| | **SES outcomes** | | | | |
|---|---|---|---|---|---|
| | education | ed diff | ln net worth | ever married | life satisfaction |
| war | -0.044 | 0.021 | 0.082 | -0.056*** | -0.171*** |
| SES low | -3.438*** | -0.017 | -0.684*** | 0.008 | -0.333*** |
| SES middle | -2.107*** | -0.086 | -0.265*** | 0.003 | -0.094*** |
| war×SES low | -0.156 | 0.572*** | 0.078 | 0.038*** | -0.229*** |
| war×SES middle | -0.390*** | 0.316** | -0.094* | 0.027** | -0.168*** |
| male | | | | -0.013 | |
| war×male | | | | 0.055*** | |
| war×SES low×male | | | | -0.046** | |
| war×SES mid×male | | | | -0.032* | |
| SES low×male | | | | -0.012 | |
| SES Mid×male | | | | -0.003 | |

| | **channels** | | | | |
|---|---|---|---|---|---|
| | hunger | dispossession | persecution | dad absent | childhood immunizations |
| war | 0.072*** | 0.023*** | 0.022*** | 0.020* | -0.024*** |
| SES low | 0.002 | -0.012*** | -0.011*** | 0.007 | -0.011*** |
| SES middle | -0.002 | -0.011*** | -0.010** | -0.002 | -0.006* |
| war × SES low | 0.020* | -0.009 | -0.013* | -0.016 | -0.039*** |
| war × SES middle | -0.008 | -0.015** | -0.008 | 0.017* | -0.018** |

Figure 3.1: World War II casualties as percentage of the population and total

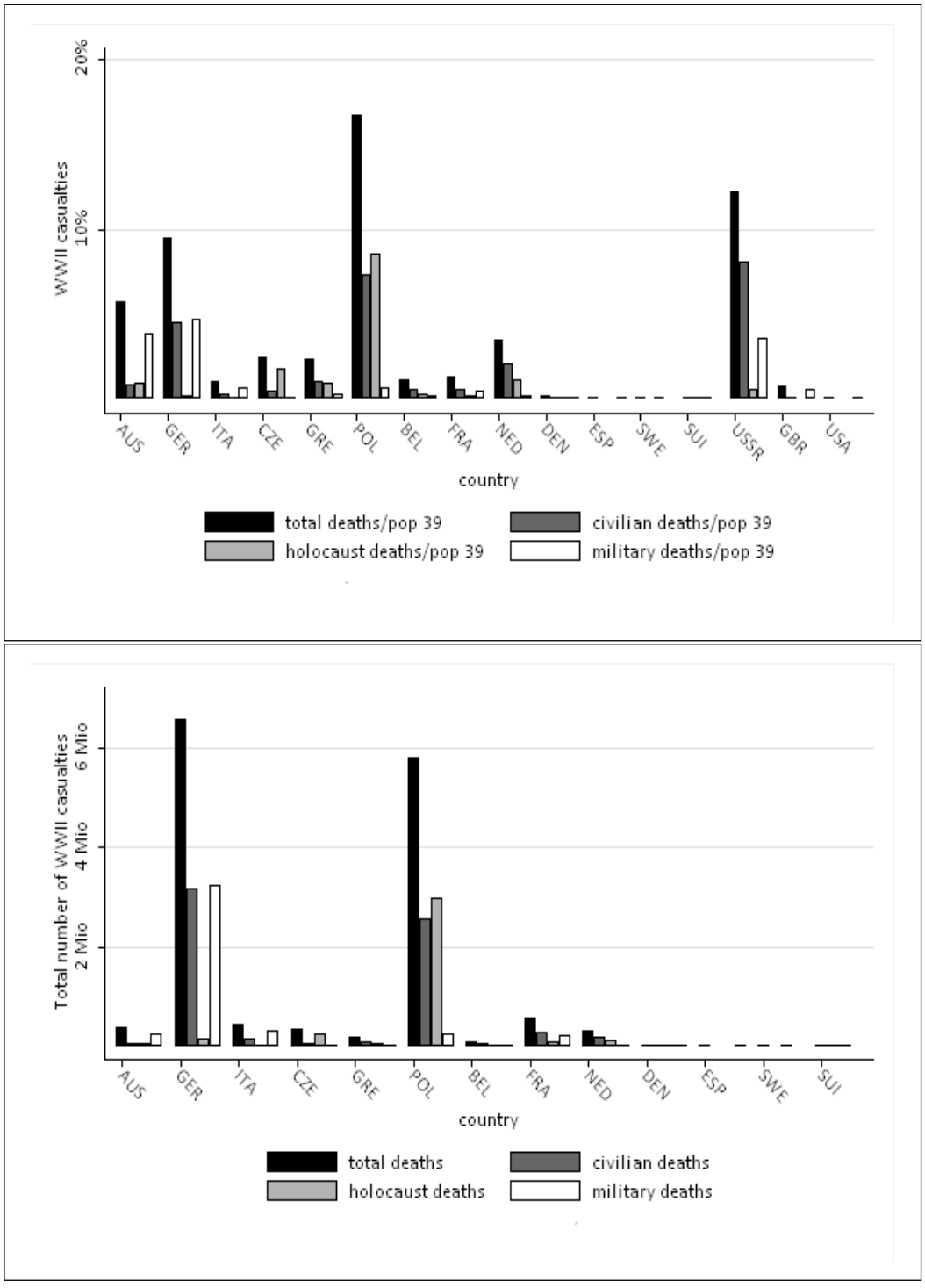


Figure 3.2: Percentage of SHARE respondents suffering from hunger: war versus nonwar countries

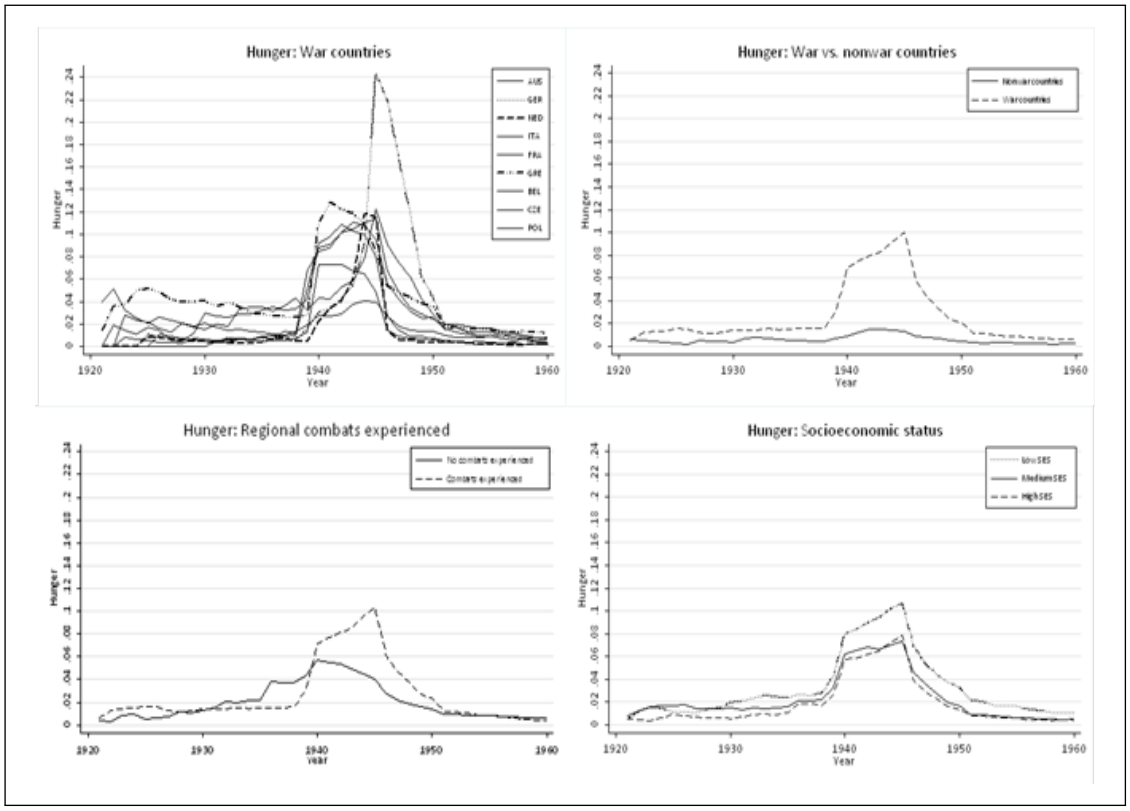


Figure 3.3: Total inflow and outflow of population, 1939-1947

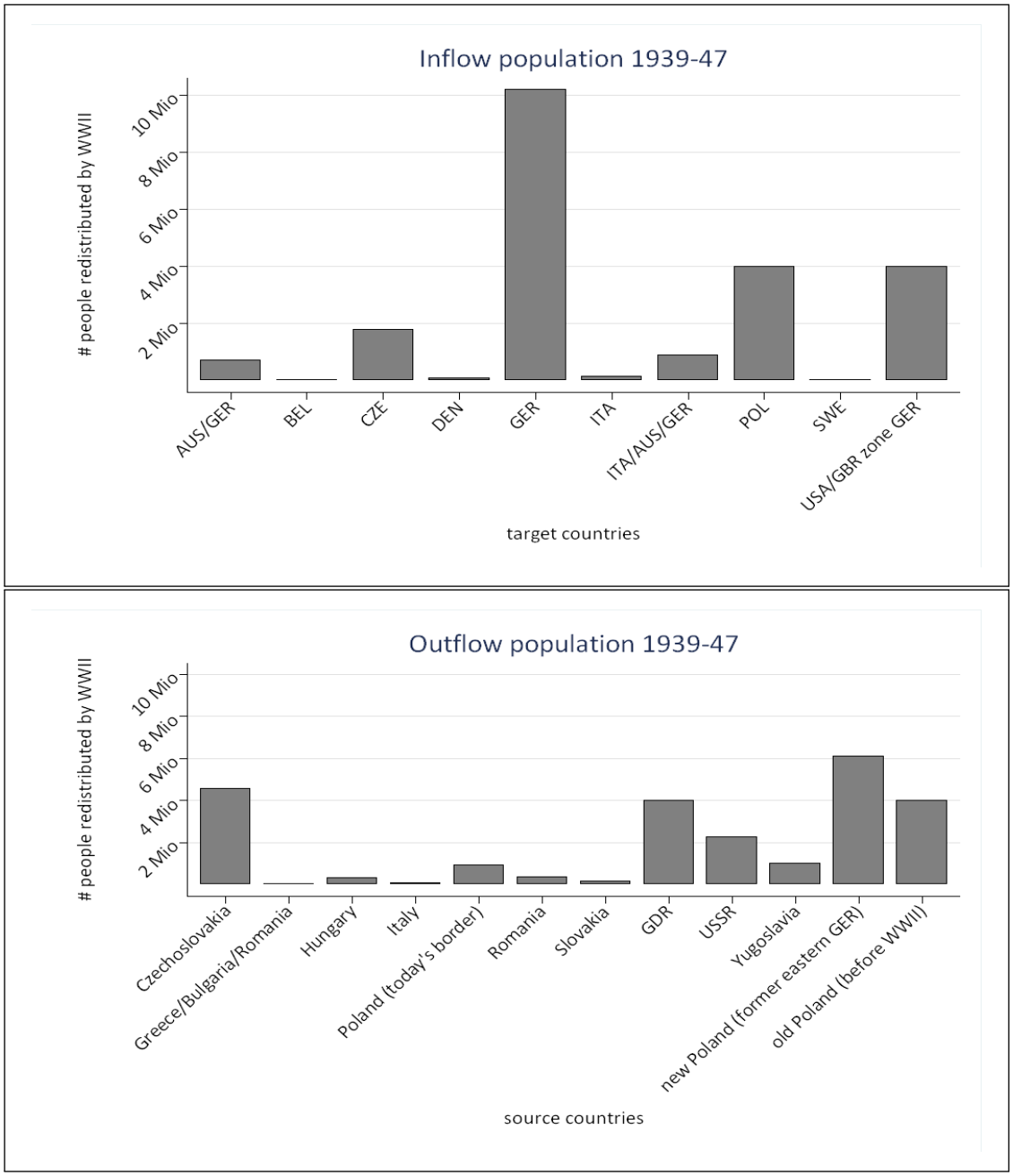


Figure 3.4: Dispossession of population in World War II

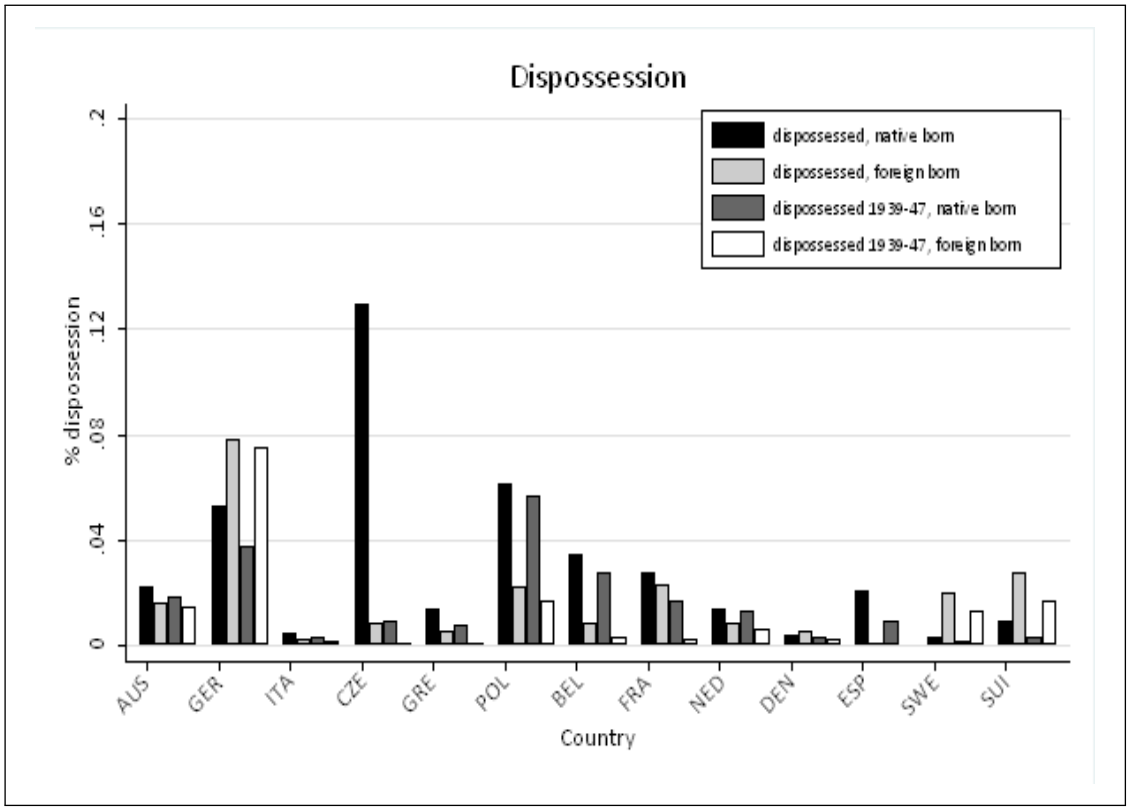
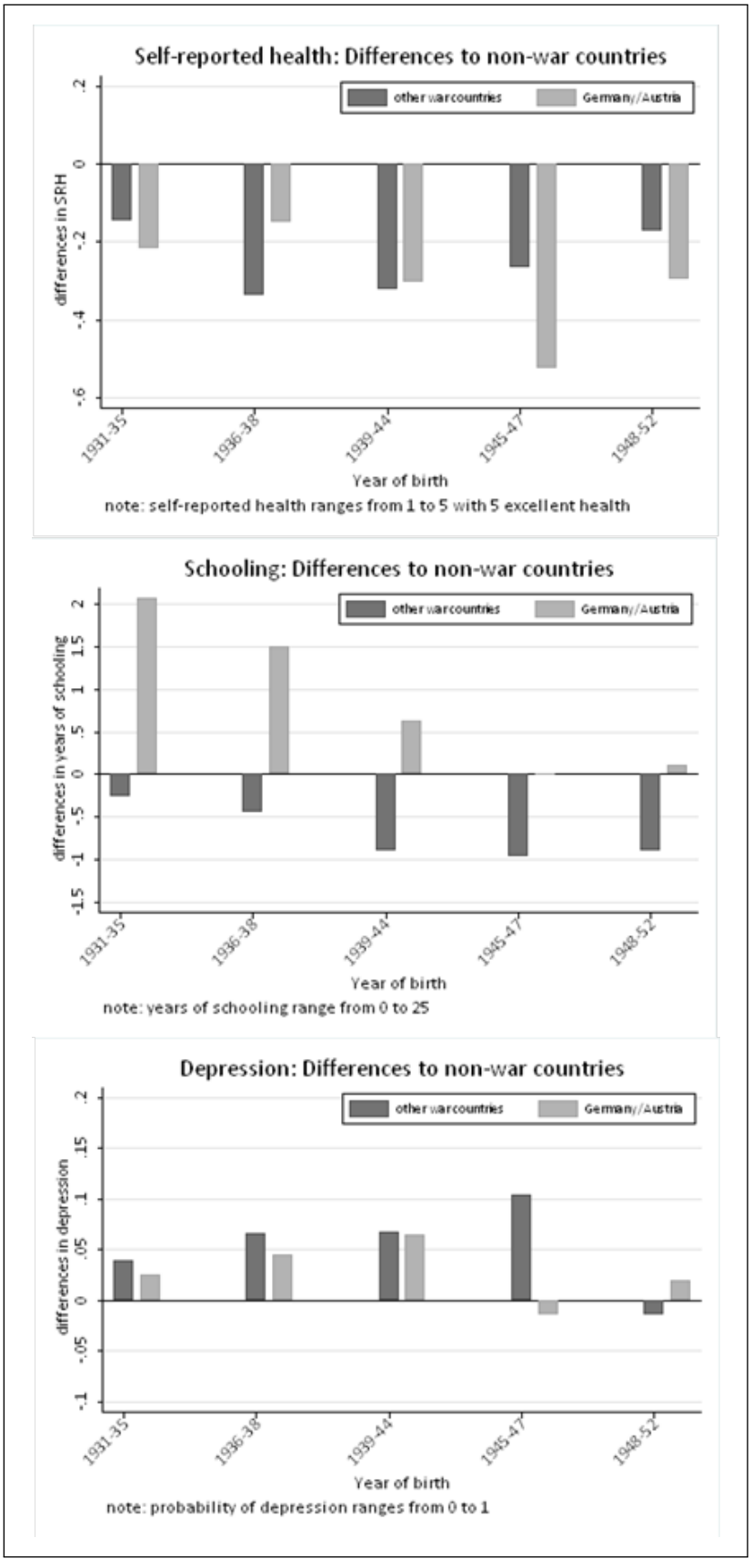


Figure 3.5: Later life outcome differences between war and nonwar countries by period of birth



# Appendix

Table A.1: External data variable definitions

| variable name | definition | sources |
|---|---|---|
| **casualties** | | |
| total deaths,<br>total deaths/ pop 39 | total casualties of WWII,<br>percentage of casualties in population | The data has been collected from three sources:<br>1. van Mourik, W. (1978): Bilanz des Krieges. Lekturama Rotterdam.<br>2. Putzger, F. W. (1963): Historischer Weltatlas. Velhagen & Klasing.<br>3. Overman, R. (1999): Deutsche Militaerische Verluste im 2. Weltkrieg. Oldenburg Verlag.<br>4. Statistical Yearbook for the German Reich 1939. |
| military deaths,<br>military deaths/ pop 39 | military deaths in WWII,<br>percentage of military deaths in population | |
| civilian deaths,<br>civilian deaths/ pop 39 | civilian deaths of WWII,<br>fraction civilian deaths/ total population | |
| holocaust deaths,<br>holocaust deaths/ pop 39 | holocaust deaths of WWII,<br>fraction holocaust deaths/total population | |
| **population movement** | | |
| source country | country from which people left | Kulischer, E. M. (1948): Europe On the Move. War and Population Changes, 1917-47. Columbia University Press. |
| target country | country to which people moved | |
| time period | year in which people moved to target countries | |
| **number of combat operations** | | |
| war combat 0-2 months | respondent was living in a war country during the war period in a region within the country that experienced 0-2 months of combat | Ellis, J. (1994): World War II – a Statistical Survey. Aurum Press. |
| war combat 3 or more months | respondent was living in a war country during the war period in a region within the country that experienced 3-10 months of combat | |
| **gross domestic product** | | |
| log(GDP) | country specific GDP | Maddison, A. (2011): Historical Statistics: Statistics on World Population, http://www.ggdc.net/MADDISON/oriindex.html (accessed June, 2011). |
| **sex ratio** | | |
| sex ratio | ratio of men to women for time periods before and after war | Statistical Yearbooks for Germany, chapter "Bewegung und Bevoelkerung", and "Internationale Uebersichten", 1909-1939 and 1945-53. |

Table A.2: Descriptive statistics

| | **percentage** | **all** | | **war = 0** | | **war = 1** | |
|---|---|---|---|---|---|---|---|
| | missing | **N = 21,266** | | **N = 11,411** | | **N = 9,855** | |
| | observations | mean | s.d. | mean | s.d. | mean | s.d. |
| **background information** | | | | | | | |
| year of birth | 0.000 | 1942 | 9.52 | 1947 | 8.07 | 1936 | 7.13 |
| male | 0.000 | 0.45 | 0.50 | 0.45 | 0.50 | 0.46 | 0.50 |
| childhood SES | 0.034 | 0.02 | 1.00 | 0.22 | 0.99 | -0.21 | 0.96 |
| **outcome measures** | | | | | | | |
| depression | 0.000 | 0.36 | 0.48 | 0.34 | 0.47 | 0.38 | 0.48 |
| diabetes | 0.002 | 0.10 | 0.30 | 0.07 | 0.25 | 0.13 | 0.34 |
| ever married | 0.000 | 0.94 | 0.23 | 0.93 | 0.25 | 0.95 | 0.22 |
| heart disease | 0.002 | 0.12 | 0.32 | 0.08 | 0.27 | 0.16 | 0.37 |
| height | 0.007 | 168.31 | 8.85 | 169.47 | 8.92 | 166.97 | 8.59 |
| life satisfaction | 0.081 | 7.60 | 1.72 | 7.87 | 1.62 | 7.29 | 1.79 |
| log(net worth) | 0.040 | 12.45 | 1.87 | 12.94 | 1.83 | 11.88 | 1.75 |
| self-rated health | 0.001 | 2.80 | 1.07 | 3.03 | 1.07 | 2.53 | 1.01 |
| years of education | 0.080 | 10.72 | 4.16 | 11.71 | 3.88 | 9.56 | 4.18 |
| education diff in years | 0.466 | 0.42 | 2.91 | 0.51 | 2.78 | 0.34 | 3.03 |
| **channels of war exposure** | | | | | | | |
| dad absent | 0.017 | 0.09 | 0.28 | 0.07 | 0.25 | 0.10 | 0.31 |
| dispossession | 0.001 | 0.03 | 0.18 | 0.02 | 0.15 | 0.04 | 0.21 |
| hunger | 0.001 | 0.07 | 0.25 | 0.02 | 0.14 | 0.13 | 0.33 |
| persecution | 0.001 | 0.04 | 0.19 | 0.03 | 0.18 | 0.04 | 0.21 |
| childhood immunizations | 0.012 | 0.95 | 0.23 | 0.98 | 0.15 | 0.91 | 0.29 |

See table 1 for definitions of variables in first column and Appendix table A for definition of war combat variable.

# Chapter 4

## Individual Behavior as Pathway between Early Life Shocks and Adult Health
## Evidence from Hunger Episodes in Post World War II Germany *

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### 4.1 Introduction

The fetal origins hypothesis establishes a biological link between health shocks experienced in utero and adult health (Smith (1999) and Almond and Currie (2011) provide excellent overviews). Through adverse events, in particular undernutrition, the fetus is programmed to have certain metabolic characteristics which then impact health in later life. There is also evidence that hunger episodes experienced later in life have long-lasting health effects (Van den Berg *et al.* (2012)). A well-established biological channel explains these effects, at least in part: Hunger has long-run effects if the undernutrition occurs in certain sensitive periods of growth or fat storage for later growth (Barker (2004)).

In this paper, we study a separate pathway between early-life shocks and adult health that has received less attention in the literature (Heckman (2012)). We argue that an early-life event – in our case a prolonged experience of severe hunger during childhood – is not only an immediate negative biological shock to the stock of health but also affects subsequent behaviors that will impact health at older ages. A one-time shock changes the entire future path of health investments over time and thus levels of health in later life. Such a mechanism would easily provide an additional rationale for the (causal) effect of early life shocks on late-life health outcomes (and also on SES outcomes which in turn are a function of the time path of health). We illustrate this channel using retrospective data on long-run effects of a major health shock in Germany: the hunger periods experience throughout the country in the immediate years after the second World War (World War II).

To test the hypothesis that there is a behavioral channel linking early-life events and adult outcomes, one would like to use a panel dataset that contains information on health, health shocks and health-related behaviors over long periods of time, ideally starting at birth or even before and extending through adult life. Such datasets unfortunately do not exist, particularly for countries most affected by World War II. Our research relies instead on retrospective life data from the European Survey of

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Health, Aging, and Retirement in Europe (SHARE) that have recently become available. SHARE covers representative samples of the population aged 50 and over in 13 European countries, with about 20,000 observations. In this analysis, we concentrate on Germany that was among those countries most affected by World War II, but also by hunger (Kesternich *et al.* (2012), forthcoming). There are 1,852 observations in Germany in SHARELIFE. In addition, we make extensive use of external data on the amount of calories available in different regions of Germany in the postwar period.

The shock we exploit is hunger in post-World War II Germany, and the subsequent behavior we consider is food consumption. Our hypothesis is that individuals who suffered from hunger in early life "eat more" in later life as a precaution against the risk of new hunger episodes. They are therefore at a higher risk to be overweight and diabetic [1].

We will reveal a behavioral mechanism of how a childhood shock translates into adult health by showing that early-life hunger experiences also predict food consumption late in life. In particular, Engel curves for food consumption are significantly different for individuals who suffered from hunger and those who did not. We proceed to showing that health outcomes follow the same pattern. To establish the link between behavior and outcomes more rigorously, we then estimate a system of equations, linking the experience of hunger, the behavioral path and health outcomes.

We find that the effect of hunger on food shares is strongest for low-income individuals. This confirms our assumption that poorer individuals are more at risk of ever becoming hungry again and thus eat more as a type of precautionary measure. Some support for our hypothesis is provided through the influence of hunger on measures of risk-aversion and trust.

An important methodological contribution is that we do not only rely on self-reported measures of hunger, but also on data on food supply (measured in calories available per capita per month, which varied in postwar Germany not only over time but also by occupation zone)[2]. Our data on caloric rations suggest that self-reports are indeed a reliable measure for the experience of hunger episodes as these self-reports accurately match both regional and time variation of the hunger episodes.

This paper is divided into five sections. The next section documents the main episodes of hunger in pre- and postwar Germany and the principal economic reasons for those hunger episodes. Section 3 highlights the central elements of the main data on which we rely in this research. It shows the

[1] Several papers use the combination of retrospective data on childhood circumstances and adult outcomes available in SHARELIFE to study the long-run effects of early-life shocks across Europe (Halmdienst and Winter-Ebmer (2012); Havari and Peracchi (2011); Van den Berg *et al.* (2012)). Juerges (2013) studies the long-run effects of post-war shocks on adult education and labor-market outcomes in Germany using data from the Mikrozensus. Neither these papers nor our own earlier research (Kesternich *et al.* (2012)) consider the behavioral channel we analyze in the present paper.

[2] Similar data on caloric rations have also been collected by Juerges (2013). In a study of the long-run effects of the post-war food crisis on educational attainment and labor market outcomes, he uses these food supply data for illustrative purposes but he does not link them to the individual-level outcome data.

correspondence between retrospective self-reported episodes of hunger and our objective contemporaneously reported calorie data. In the following section 4, we model the relationship between childhood hunger and caloric restriction on later life food consumption, obesity, and diabetes. We summarize our main conclusions in section 5.

### 4.2 The Nutritional Situation in Germany during and after World War II

Rationing of food in the form of food stamps in Germany starts with the beginning of the war on August 28, 1939. From this date until the end of the war, Germany's food supply and distribution was organized centrally (Schlange-Schoeningen (1955)). There was a forced cartel of all trade organizations dealing with the production and processing of food (Rothenberger (1980)). Food and many other objects could only be bought in exchange for food stamps. There was some black market, in which stamps were traded for alcohol, cigarettes, coffee or clothes against food stamps.

Before World War II, Germany had been able to cover about 83 percent of its food demand through domestic production within its borders at that time (Farquharson (1985)). At the beginning of World War II, daily rations of food were quite high with just below 2,500 kcal until the end of 1941 (Eitner (1991)). Thus, cuts were small for those who had not lived in luxury before the war. Starting at the end of 1941, food rations were lowered to about 2,000 kcal/day. In addition, the increased work load, night shifts, and stress of bombing decreased the time spent sleeping and increased the caloric needs of the population (Eitner (1991)). Some of the decrease was due to productive capacity being channeled into war production and to men being absent (basically this work was done often by elderly women and prisoners of war or forced laborers). However, actual famine conditions started only towards the end of World War II and the allied occupation in 1945, and lasted until the winter of 1947/48[3].

There were several reasons for this famine. First, with the end of the war, the prior central organization of food distribution and production in Germany completely collapsed. It was replaced by an organization imposed by the Allies in each of the four occupation zones. The administration of each occupation zone set and enforced caloric goals for each occupied region. The official rations lay between 1200 and 1400 kcal per day in 1946 and 47 (Schlange-Schoeningen (1955)). Since the German currency was basically valueless, trade took place against food stamps or other products.

Second, in Germany (in today's frontiers), agricultural productivity per hectare dropped to about 2/3 to 3/4 of the pre-war levels (Liebe (1947)). This drop was caused by a lack of fertilizer, large-scale war destruction of farm buildings and machinery, as well as death and displacement of workers. In addition, land mines were an additional hazard to agriculture (Farquharson (1985), Liebe (1947)). In total, the harvest of 1946 was at about 67 percent, the one of 1947 at about 50-55 percent of a

[3] The first hunger edemas were reported in the summer of 1945 (Farquharson (1985)).

normal harvest (Haeusser and Maugg (2011)). Besides the drop in agricultural output in its territory, Germany lost its pre-war Eastern parts (East Pomerania, Silesia, and West Prussia) to Poland, the Czech Republic and the Soviet Union.

The pre-World War II Eastern parts of the German Reich were a vital part of the German agricultural system. Until the end of the war, about 30 percent of the agricultural products and the inputs of agricultural production, e.g. seeds, had been produced in the Eastern part of the German Reich (Rothenberger (1980)). Germans living in these regions, who were mainly farmers, were forced to leave and move westwards. They arrived as refugees in the remaining parts of Germany. A large share of these refugees was hosted by the British occupation zone. The large-scale migration also posed problems for the Eastern parts. Even though inhabitants of other parts of Poland and the Czech Republic were moved there and took over the farms, production after the war was only at about 50 percent of what had been produced before (Rothenberger (1980)).

Right after the war, the nutritional situation was bad not only in Germany, but also in Austria, Finland, Hungary, Yugoslavia, Southern France and Romania. But with the exception of Romania, the situation was most severe in Germany (Rothenberger (1980)). In addition, harvests had been bad in the traditional grain producing countries such as Canada, Australia and Argentina (Haeusser and Maugg (2011)). In addition, there were further episodes of hunger in India and the rest of Asia which had to be alleviated by food imports from abroad.

Agricultural output across the four occupation zones varied to a large extent. The Russian and French zones did worse than the British and US zones, because parts of the local production (fertilizer, meat, milk) were confiscated and distributed among the military personnel or brought back to the occupying countries (Schlange-Schoeningen (1955)). French occupiers saw Germany as a "compensation object" after having suffered from German occupation during the war. Even entire factories were deconstructed and brought to France. Until the French zone was united with the English and US zone, its inhabitants faced a worse nutritional situation than their counterparts in the Anglo-Saxon zones (about 200-300 kcal per day less). As the war had been especially atrocious for Russia (it had lost more than twenty million people), the attitude towards the occupied German country was even harsher. Thus, the nutritional situation was also worse in the Soviet zone.

Third, the severe problems in food production were exacerbated by the breakdown of trade and transport within Germany. Before World War II, agricultural production in Germany was fairly specialized due to differences in geographic location and there was ample trade of food products between the different parts of Germany. For example, seeds had mostly come from the Eastern parts of the German Reich. In addition, industries producing agricultural inputs, such as the chemical industry, relied on suppliers from all over the German Reich (Rothenberger (1980)). Trade between the different occupation zones basically stopped, which amplified the problems in food supply due to a shortage in inputs such as seeds and fertilizers.

A third reason for the German famine was the destruction of transport infrastructure (roads, rail

tracks and bridges) which caused a stop in trade. Both shipping and transportation via rail was nearly impossible after the war, because rails were destroyed and the debris of destroyed bridges blocked major rivers. In the British occupation zone, for example, only 1,000 out of 13,000 km of railway tracks were operational in spring 1945 (Farquharson (1985)). In addition, roads were in bad shape and only a small part of the trucks were still able to function due to shortness in repair material and fuel (Rothenberger (1980)). Often, individuals had to resort to basic means of transport such as horse-drawn transport, or even handcarts (Farquharson (1985)).

Fourth, borders between the occupation zones, which were drawn without food supplies in mind which hampered trade even more. This led to zones of different nutritional potential, each of which had to meet its own demand completely from local production (Rothenberger (1980) and Schlange-Schoeningen (1955)). Trade was made impossible by the fact that the command of the allied governments ended at the zone frontiers, so all measures steering the economy ended there as well (Rothenberger (1980)). The three Western zones were completely blocked off from the Eastern zone that had a higher nutritional potential. There were some attempts of the British zone to trade steel against food produced in the Soviet zone, but largely these attempts failed (Farquharson (1985)). The French also saw their occupation zone as a separate entity so that the economic relationships with the other zones were cut to a large extent. The British government tried to approach the French government several times about selling some fertilizer from the French to the British zone, but the French preferred to use this fertilizer in France (Rothenberger (1980)). In addition to these supply side problems, there was an increase in demand, at least in the Western regions. About 10 million refugees from the formerly Eastern parts of the German Reich flooded into the remaining parts (Kulischer (1948)).

Famine was somewhat relieved through food relief shipments from the United States. These food shipments were allowed from December 1945 onwards. Food relief shipments to Germany in the form of CARE Package shipments to individuals started to be allowed from June 1946. Slowly, trade and agricultural production resumed. The occupation governments understood how big the impediment of the transport sector was, and efforts to restore this sector had been successful from 1947 onwards (Haeusser and Maugg (2011)).

Right after war, the food administration of the forces occupying the Western part of Germany allocated few resources in the right way. After a re-organization of the food administration, which included German citizens, this improved[4]. Still, the supply situation did not pick up immediately, because there was some hoarding by farmers (Haeusser and Maugg (2011)). In the Western occupation zones, markets started to function much better with the currency reform of 1948. Even though food prices were very high right after the currency reform, supply picked up considerably (Haeusser

[4] The harvest of 1947 was good, not only in Germany, but also in the US and Canada leading to a more relaxed nutritional situation.

and Maugg (2011)). In the Eastern part, there was a land- and currency reform, and the economy was transformed into a socialist system.

## 4.3 Data

In this section, we present our data. We first describe the source of the individual-level survey data that provide both retrospective information on early-life circumstances and adult outcomes (subsection 3.1). We then discuss in subsection 3.2 the sources of administrative data caloric rations and how we constructed the monthly and regional food supply variables. Finally, we describe how the analytic sample was constructed from these two main sources of data (subsection 3.3).

### 4.3.1 SHARE and Retrospective Early-life Data from SHARELIFE

SHARE is a multidisciplinary cross-national panel interview survey on health, socio-economic status, and social and family networks of individuals aged 50 or over in continental Europe. The original 2004/2005 SHARE baseline included nationally representative samples in 11 European countries (Denmark, Sweden, Austria, France, Germany, Switzerland, Belgium, Netherlands, Spain, Italy, and Greece) drawn from population registries, or from multi-stage sampling (http://www.share-project.org/). For these countries, a second wave of data collection took place in 2006, and the third wave of data collection on this panel (SHARELIFE) was completed in 2008. In addition to a standard set of demographic attributes, SHARE data include health variables (self-reported health, health conditions, and health behaviors), psychological variables (e.g., depression and well-being), economic variables including gross family income and education, and expenditures on food both inside and outside the home.

SHARE's third wave of data collection, SHARELIFE, collected detailed retrospective life-histories in 2008-09. SHARELIFE was based on life history calendar (LHC) methods. This data include region of residence, childhood SES, and the experience of hunger. The information in the life history comprises family composition and type of home (number of rooms, running water, toilet, etc), number of books, and occupation of father. We use the number of rooms and the facilities in the accommodation, the number of books, and the occupation of the main breadwinner to construct a measure of household SES at age 10[5]. We follow Mazzonna (2011) and apply a principal component analysis. Each principal component refers to a weighted average of the underlying four indicators[6]. We retain the

[5] The two items on the state of the accommodation are considered as asset indicators and long-run household wealth (McKenzie (2005)). The number of books represents the household's cultural background, and the occupation of the main breadwinner is a general indicator about the household's financial endowment.

[6] Weights are chosen such that the principal components accounts for the maximum amount of the variance in our four single items.

first component, since it explains about 42% of total variation, and is the only one with an eigenvalue above average. We moreover split this index up into three groups, high SES, medium SES and low SES during childhood, using the quantiles of the linear SES index as threshold values.
Moreover, SHARELIFE provides detailed data on within country region of residence and housing during the full life of respondents (childhood and adulthood). Respondents were asked about the region and the type of accommodation when they were born. Then they were asked if they ever moved and, if yes, when, where and why. These questions were asked in a loop for each subsequent move.

### 4.3.2 Caloric Rations and Self-reported Hunger

In SHARELIFE, respondents were asked

> ***"(Looking back at your life), was there a period when you suffered from hunger?"***

If respondents answered "yes" to this question, they were then asked when the period started and when the period ended. Thus, we get information on the starting date and the end date of hunger, as well as on the duration of this hunger episode.
Figure 4.1 shows the percentage of respondents suffering from hunger at a given point in time in all countries in SHARE that participated in World War II. We see that periods of hunger respondents reported coincide closely with the historical facts: The prevalence of hunger picks up with the end of the war and after the war for Austria and Germany and during the war for the other countries. Germany is the country with by far the most respondents reporting suffering from hunger. This might be surprising at first, considered the hunger episodes in the Netherlands and Greece. However, the famines in the Netherlands and Greece were due to transport blockades in otherwise hardly destroyed countries. In the Netherlands, the famine affected only the Western parts of the country, and lasted for a relatively short time period (winter 1944/45 in the Netherlands). The famine in Germany took place in a country where the destructions of capital stock, both human and physical, and infrastructure were huge.
The main reason for choosing Germany as our case study is that in Germany, during and after World War II, food production and supply was under state-control, and organized centrally. Thus, in addition to individuals' self-reports on periods of hunger, we were able to collect data on official caloric rations in the German Reich, the occupation zones, the Federal Republic of Germany and the German Democratic Republic. Our data include caloric measures from January 1929 to December 1971.
Food supply was not restricted in Germany before 1939. We therefore use the average caloric intake per day recommended by the "Völkerbund" for the German population (Liebe (1947)). From 1939 till

the end of World War II the rationing period was one year, and new caloric rations were determined in the mid or end of September (see Schlange-Schoeningen (1955), Farquharson (1985) for ration periods). Since the residential history of respondents is available on a yearly basis, we computed a weighted average of the official caloric rations provided from 1939 to 1945. The official caloric ration for a given year consists of 10/12 of the ration period for the corresponding year and 2/12 of the caloric ration of the next years rationing period.

In April/May 1945 the Allies divided the remaining German territory into four occupation zones independently administered by the USA, Great Britain, the Soviet Union, and France. From this time on, official caloric rations provided to the German population varied by occupation zones. Moreover, the rationing periods changed from one year to monthly updates in the US, the UK, and the French zone. There was a considerable variation in the official caloric rations between occupation zones and over time. The three western occupation zones updated the official caloric ration every four weeks. Changes in the official rations were less regular in the Soviet zone (see Schwarzer (1995)). In May 1947, the UK and the US zone merged into a united economic territory, thus providing an identical amount of calories per day. In June 1948 the currency reform took place in the Western occupation zones leading to a sudden improvement in the food supply. In 1949 the UK zone, the US zone and the French zone merged into the Trizone which then became the Federal Republic of Germany whose constitution was adapted on May 24th 1949. From 1950, any food rationing was completely eliminated and the recommended level of kcal/day was again achieved. With the founding of the Federal Republic of Germany, the former German territory was divided into two separate states. In the German Democratic Republic the situation of food supply was still problematic and food rationing took place until 1956 (Schwarzer (1995)).

We merge monthly official caloric rations to respondents that participated in both the first and the third wave of SHARE by using information on the respondent's region of residency during each year of their life's, and birth dates. With exception of Baden-Wuerttemberg each German state completely belonged to one of the occupation zones (see Table 4.1). Therefore we can identify the occupation zone in which a respondent lived in a particular year[7].

We generate age-specific caloric intakes at age 0-4, 5-10, and 11-16. Starting from the month of birth, we sum up 48 months of official daily caloric rations for age 0-4. For age 5-10 and 11-16 we take the sum of 72 months of caloric rations starting at the first month after the respondent's fifth and eleventh birthday, respectively. This yields three age-specific variables containing the sum of daily official caloric intake. We then divide these sums by the number of months in order to get a

[7] Baden-Wuerttemberg was the only state that did not uniquely belong to one occupation zone, but was split up between the French zone and the US zone. We use information on the area of the respondents' residence in order to assign as many respondents from Baden-Wuerttemberg to any occupation zone. We can identify 21 respondents that lived in Stuttgart, because it was only big city in Baden-Wuerttemberg in 1945-48. Since Stuttgart was part of the US occupation zone, we can attach the official caloric ration of this zone to respondents from Stuttgart (see for instance Cornides (1948)).

measure for the average daily caloric intake by ages 0-4, 5-10- and 11-16. With these caloric rations we can now construct a measure of hunger intensity. In the literature, the caloric thresholds used to construct poverty lines lie between 2,100 and 2,400 kcal (see Deaton (1997)). However, we are interested in the effect that a famine with a clearly defined period has on the respondents' later life outcomes, not in periods of malnutrition. Thus, we use the metabolic rate at rest, which was estimated to lie between 1,500 kcal and 1,637 kcal (Boldorf (1998), Silbernagl and Despopoulos (1988)). We construct a variable that measures the number of months an individual was below this threshold of 1,500 kcal. We supplement hunger self-reports with official caloric rations as a measure of hunger. These administrative data are not afflicted with misreports due to memory errors, and they are exogenous to the individual. Even though the official caloric rations do not vary by individual, they vary by occupation zone and change monthly rather than yearly, while individual retrospective hunger episodes are reported on a yearly basis.

Figure 4.2 illustrates the relation between self-reported hunger and the official caloric rations. The upper line presents the variation in the official caloric ration from January 1930 to January 1970. The lower line illustrates the hunger self-reports. The two measures similarly vary quite closely over time. When the official caloric rations start to decrease in 1939, the fraction of respondents reporting hunger increases at the same time. The official caloric intake achieves its minimum in the summer months of the years 1946/47. The rations are higher in the winter than in the summer where the people might have gotten some additional calories from self-grown fruits or vegetables.

If self-reports are reliable measures for actual experienced individual hunger periods, then we should find a significant correlation between the official caloric rations and self-reported hunger. Hunger reports are binary variables which take on the value one if a respondent reported a period of hunger. The caloric intake is measured by the amount of official calories that were provided in each month. Each specification includes dummy variables for the occupation zone. This allows us to control if respondents from a specific occupation zone report systematically more hunger than others. In a similar way we control for whether the respondent lived in an urban area (at birth). The overall pattern of the first stage regressions provided in table 4.2 suggests that there is a strong association between the official caloric intake and hunger self-reports, and that this association gets stronger the older the respondents were at the time of the hunger incidence. While the official rations are exogenous to the individual and not affected by possible recall biases, they do not reflect the inter-individual variation, which was quite strong in this hunger-episode: Those consumers who or whose families worked in agriculture, were somewhat better off, because they could put away some of the production aside for home consumption. For those consumers who were depending on the official supply, the official rations could often not be met (Schlange-Schoeningen (1955))[8].

It is further not surprising that the association between the official rations and the hunger self-reports

[8] As mentioned above, there was also some black market trading, which grew in importance with time and with an increase in output. This black market trading was more common in the Western occupying zones where it reached

is weaker at younger ages: The very young were often breastfed, and, in the bi-zone (British and American) there were additional allowances for pregnant women and nursing mothers (Farquharson (1985)). Starting in February 1946, there were additional daily meals for school children with portions of 235 additional kcal for those under twelve, and 500 additional kcal for those aged twelve to fourteen in the bi-zone. Coverage of these school feedings increased over time and reached about 2/3 of those children in the bi-zone aged 6-18 by 1947 (Farquharson (1985)).

To get a visual understanding of how hunger self-reports vary with time and age, consider Figure 4.3 which shows when at which age our respondents report to have suffered from hunger. While there are basically no reports of hunger before the beginning of World War II, there is already a small fraction of respondents who reports to have suffered from hunger towards the end of World War II. However, most respondents report hunger between 1945 and 1950, and we find a higher incidence for those aged 5-10 and 11-15 than for the very young (aged 0-4).

Finally, we want to analyze whether our hunger measures only reflects low SES status, because individuals with higher SES were better off. During World War II, those in the upper part of the hierarchy of the Nazi party, but also economic leaders, were still able to consume much more than the official rations (Eitner (1991)). After the war, however, suffering from hunger or not was more a question of living in the country-side or in a city. About 30.5 percent of Germans lived in the major cities and about 13.1 percent in minor cities. Here, the nutritional situation was much worse. In figure 4.4, we show self-reported hunger by SES, and we see that, although low SES individuals do a little worse, hunger was a universal phenomenon across all SES groups in Germany.

### 4.3.3 Construction of the Analytic Dataset

For our analysis, we use a sample that originally consisted of 1,763 respondents from Germany. We restrict our sample to only those respondents who were born within the borders (before 1945) of the German Reich. 207 respondents were not native-born according to this definition, leaving a sample of 1,555 respondents[9].

In wave 1 and wave 2, SHARE asks respondents about the average monthly amount the household spent on food inside and outside the home in the 12 months prior to the interview. We observe 985 non-missing observations for food inside and 1,030 for food outside the home. 1,084 respondents reported a yearly income in the sample. We use this information to infer the average monthly income. We construct a variable measuring the monthly food share of a household as the monthly amount spent on food inside and outside the home over monthly gross income. Dropping observations with a

an extend of up to 1/6 of industrial output in the bi-zone in 1948, while it was less possible in the Soviet zone where production was already heavily controlled (Farquharson (1985))

[9] We lose one observation when we constructed the monthly caloric intake by age for each respondent due to lack on information about the residence.

valid food share only for the amount spend on food outside, but not at home, we get 938 respondents with valid food shares[10].

Since we do not want to lose more observations than necessary, we conducted a simple imputation procedure using food reports of wave 2. For those respondents who participated in wave 1 and wave 2 and do not report a valid food share in wave 1 we construct a food share variable based on the reported amounts spent on food in wave 2 and the reported gross income in wave 1[11]. This allows us to impute 90 missing observations, and 3 food shares greater than one with positive food share values using the food amount variable from wave 2 (0 observations with zero food shares,)[12]. After the imputation, we obtain 1,028 observations for food share. Next, we eliminate outliers in the variable measuring the food share by dropping observations below the 1% and above the 99% percentile of the food share distribution. This reduces the number of respondents by 22, leaving 1,005 observations in the sample.

We are interested in the consequences of hunger as an adverse health event for later life health outcomes. Our identifying variation comes from the period between 1945 and 1948 when Germany suffered from extreme food shortages as a direct consequence of having lost World War II. Accordingly, respondents born before 1929 are out of the range of age that is interesting for us. Moreover, individuals born before this time might also be affected by the consequences of the Great War, or the world economic crisis. We thus restrict our sample to only respondents born in 1929 or later. Our final sample consists of 923 respondents. Table 4.3 gives an overview over the different steps of sample construction and corresponding numbers.

### 4.4 Empirical Strategy and Results

The objective of this study is to reveal individual behavior as pathway between an early life shock - hunger - and later life health outcomes. Our behavioral dependent variable is the food share, defined as the share of household food expenditure (at home and outside the house) in total household gross income[13]. Our health outcomes are the body mass index, a binary indicator for being overweight and for having diagnosed with diabetes. Our measures for experiencing a period of undernutrition

[10] A considerable fraction of respondents (553 observations) do not provide a value for the amount spent on food at all.

[11] SHARE collected the household net income rather than the gross income in wave 2. Thus, we could not construct an appropriate and comparable food share measure for wave 2 respondents.

[12] 882 Respondents participated in wave 1 and wave 2 and gave a valid answer to questions on gross income and to the amount spend on food at home/outside in wave 2. The correlation coefficient between the food amount variables of wave 1 and wave 2 is about 0.54, The correlation for food shares in wave 1 and wave 2 is even higher (0,71).

[13] Similar to De Luca and Peracchi (2011), we define the food share as the ratio of expenditures for food at home and foot outside home over total gross household income.

is a binary indicator variable, based on the survey self-report, for whether the respondent suffered from hunger, and measures of calorie supply that vary across occupation zones and by months. In the main analysis, we use the number of months that an individual lived in a region during a time period with the official caloric rations below a threshold of 1500 kcal per day. The main explanatory variables are the natural logarithm of total household gross income (in the following: log income), and interactions with log income and the undernutrition measures. Additional covariates, included in all regressions, are age and squared age, gender, urban area (at birth), occupation zone (at birth), years of education, and being in a partnership.

### 4.4.1 Engel Curves

We start by specifying the link between hunger episodes during childhood and food shares as a prominent measure for consumption behavior. To model food shares, we use a variant of the well-known parametric Working-Leser specification of an Engel curve and estimate the model by OLS (Lewbel (2008)).

The Engel curve states that the fraction of income spent on food decreases as income increases, for a given set of tastes and preferences. In a linear model this relationship is characterized by a downward line in a food share-log (income) graph. The Engel curve is assumed to the shape-invariant, suggesting that changes in policies and demographic variables shift and scale the function without altering the overall shape (Blundell *et al.* (2007)). We assume that experiencing hunger during childhood represents such a type of Engel curve shifter[14]. More specifically, individuals might change their spending on food in response to the experience of hunger leading to higher food shares in adult life. We analyze this assumption by interacting hunger measures with the household log income. In order to add flexibility to the Engel curve, we use median split log income splines rather than a linear specification for the household gross income (Lewbel (2010)). The Engel curve equation then is

$$FS_i = \beta_0 + \beta_1 H_i + \beta_2 I_i + \beta_3 S(I < p50)_i \times H_i + \beta_4 S(I \geq p50)_i \times H_i + \mathbf{x'}_{it}\boldsymbol{\gamma} + e_i \quad (1)$$

where $I_i$ refers to the log household income, $H_i$ is self-reported hunger, $S(I < p50)_i$ and $S(I \geq p50)_i$ are interactions of hunger and log gross income below/above the median, $\mathbf{x}_{it}$ is a vector of covariates, and $e_i$ is the error term.

Table 4.4 presents the results from an estimation of food shares on hunger, log gross income, interactions of hunger with median income splines, and a number of covariates. Income has a strong negative impact on food shares reflecting the Engel curve relation quite well as established in the literature (see Blundell *et al.* (2007), Lewbel (2010)). Moreover, we find a strong positive impact of the hunger main effect on food shares for ages 0-4 and 11-16: Having suffered from hunger at

[14] There is some evidence that families suffering from food insecurity have a different behavior towards food (Hamelin *et al.* (1999)).

these ages leads to a significantly higher food share later in life. This relationship seems to particularly driven by those whose log gross household income is far below the sample-median. As income increases towards the median income in this group, the differences between those who experienced hunger at ages 0-4 and 11-16, and the other respondents becomes smaller. For log gross incomes above the median this relation is not significant at age 0-4. For ages 11-16 those who suffered from hunger also show higher food shares as along the income dimension.

In order to check the robustness of the results found for hunger self-reports, we now replace $H_i$ in equation (1) by the months below a threshold value of 1500 kcal/day. In contrast to self-reported hunger, the caloric measure is not a self-report, but was fixed by the administrations of the occupation zones. Thus, while hunger self-reports might be correlated with unobserved effects such as growing up in a family of farmers, the caloric rations are exogenous to the individual. Table 4.5 reports the results. The impact of log gross household income on food share is similar in size as in the regressions for hunger self-reports. The main effect of the months below 1500 kcal/day now is significant for each age specification. The more months the individuals were exposed to a nutritional situation of below 1500 kcal/day, the higher the amount spent on food relative to their income. Moreover, the interaction terms of the caloric measure and income are significantly different from zero. These results confirm the findings from the hunger regressions in table 4.4. They suggest that individuals who suffered from hunger have a different food consumption behavior as adults than those who did not experience hunger, or had hunger later in life. These differences are stronger for those who have low gross household incomes and are amplified for a larger number of months below the threshold of 1500 kcal/day.

We illustrate the relation found in tables 4.4 and 4.5 by plotting the food share predictions for hunger and our measure of caloric rations at age 0-16 against log gross household income. The upper graph in Figure 4.5 represents Engel curves stratified by whether the individual suffered from hunger or not. For both groups we find the assumed relationship of an increasing food share as income decreases. Group-specific differences in the slope of the income coefficient become significant for very low incomes. Those with a log income below the median spend a considerably higher fraction of their income on food when they experienced hunger than those who did not suffer from hunger at this age. With increasing income, the food share of the hunger and the no hunger group converge for low incomes. For respondents with an income above the median, we do not find significant differences in the hunger and the non-hunger group. The findings from this upper figure 4.5 underline our results of behavioral differences in food consumption due to having suffered from hunger (table 4.4).

In the lower graph of figure 4.5 we replicate the upper one, but now stratify by whether the respondent ever experienced a month at age 0-16 at which the official caloric ration was below 1500 kcal. The differences between groups are even larger for the low income group, but also disappear as income increases. Both graphics in figure 4.5 confirm our hypothesis that those who have suffered from hunger behave differently in terms of food consumption if they hold low incomes. The more

income the respondents earn, the more the consumption differences decrease. Thus, having suffered from hunger might change individuals' behavior which in turn might have consequences on later life health outcomes.

One tentative explanation for why we only see significant changes in food consumption for low-income individuals is that these individuals precaution against the risk of new hunger episodes. To explore this line of argument further, we present some suggestive evidence that the experience of hunger makes individuals more risk averse and less trusting (see Appendix table A.1). Individuals are significantly more risk averse if they suffered from hunger at age 0-16. Most of this effect is generated by the group of respondents who suffered from hunger between 0-4. Moreover, this group of respondents also is significantly less trusty. Significant evidence is restricted to only this age group, but the sign of the coefficients all show into the right direction.

Given the evidence of a behavioral effect of hunger, we will show in the next section that food consumption is not only the outcome, but serves as a channel for hunger periods as an adverse childhood shock. Our hypothesis is that having experienced hunger has an effect on late life outcomes that are transferred by food consumption as a behavioral component. If this is correct, we would expect that food share has an effect on health outcomes, once we have controlled for hunger and income.

### 4.4.2 Early-life Hunger Experiences and Adult Health Outcomes

We start off modeling the relationship between late life health outcomes and hunger by specifying the following latent variable equation.

$$Y_i^* = \alpha_0 + \alpha_1 H_i + \alpha_2 I_i + \alpha_3 S(I < p50)_i \times H_i + \alpha_4 S(I \geq p50)_i \times H_i + \mathbf{x}'_{it}\boldsymbol{\gamma} + u_i \tag{2}$$

$Y_i^*$ refers to the health outcomes of interest that are either linear measures of binary indicators for health. The error term is defined as $u_i$, $I_i$ is the log gross household income, and $\mathbf{x}_{it}$ other exogenous covariates. The main and interaction terms for hunger $H_i$ and income $I_i$ are defined as in equation (1). We can interpret the coefficients $\alpha_1$, $\alpha_2$ and $\alpha_3$ as the general effect of having experienced hunger on late life health outcomes, capturing all possible channels including behavioral and physiological impacts.

Table 4.6 presents the results from an OLS estimation of BMI. Individuals who suffered from hunger at ages 11-16 and 0-16 have a significantly higher BMI than others. This link is even stronger for individuals at the lower tail of the income distribution. The higher the household's income, the smaller becomes the effect of the hunger experience on BMI.

This pattern is replicated when estimating a probit model on the probability of having overweight (table 4.7). There are significantly positive effects of having experienced hunger at ages 5-10 and 11-16, and also for the whole range of age 0-16. Again the difference between hunger and no hunger experience on the probability of having overweight decreases for the low income group as income increases. Finally, we estimate a probit regression on the probability of having diabetes and find

significantly positive effects of hunger at age 11-16 (table 4.8). Consistent with the findings from the previous regressions, the effect of hunger on the probability of having diabetes is significantly stronger for individuals with low household incomes.

The results provide an important implication. An adverse hunger shock seems not to only affect health outcomes when the individual is still in utero (see Barker (1992), Lumey and Stein (1997)). Instead, the hunger experiences made during the whole age of childhood seems to have negative consequences on late life health. In particular, individuals who have suffered from hunger during adolescence show worse health outcomes later in life[15].

The hunger effects presented in tables 4.6-4.8 are not restricted to a specific channel. Rather they bundle all possible pathways through which hunger affects late life health outcomes. This includes biological links associated with the development of organs or cognition (see Cameron and Demerath (2002)), but also non-biological mechanisms, such as changes in food consumption behavior (see Hamelin *et al.* (1999)). The aim of this study is to identify a link between hunger episodes and later life health outcomes which solely works through behavioral changes in food consumption. In the next step of our analysis we therefore isolate the behavioral link from other biological and physiological mechanisms affecting adult late life health.

### 4.4.3 Hunger, Engel Curves and Health Outcomes

In the previous estimates, the food share was an omitted variable such that the estimated hunger coefficients represent both the influence of hunger through various pathways. To distinguish between different biological and behavioral channels we estimate a system of equations in which food share is included in the second stage as an explanatory variable. Our model is fully recursive consisting of two equations.

$$\begin{bmatrix} FS \\ Y^* \end{bmatrix} = \begin{bmatrix} 0 & 0 \\ \theta_{21} & 0 \end{bmatrix} \begin{bmatrix} FS \\ Y^* \end{bmatrix} + \begin{bmatrix} \delta_{11} & \delta_{12} & \delta_{13} & \delta_{14} & \delta_{15} \\ \delta_{21} & \delta_{22} & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} H \\ I \\ S(I<50)\times H \\ S(I\geq 50)\times H \\ HHsize \end{bmatrix} + \mathbf{\Gamma} \boldsymbol{X} + \begin{bmatrix} \epsilon_1 \\ \epsilon_2 \end{bmatrix} \quad (3)$$

The system contains two endogenous variables, food share and health outcomes. $\theta_{21}$ is the coefficient of the endogenous variable in the second stage and measures the effect of food consumption on later life outcomes. The matrix $\boldsymbol{D}$ comprises the hunger and income effects for the equations, the median splines income-hunger interactions, and the exclusion restriction needed for identification. The $\mathbf{\Gamma}$ matrix contains coefficients for the effects of the exogenous variables. The two error terms

[15] The results from the estimation of equation (2) where self-reported hunger is replaced with the exogenous hunger measure of months below 1500 kcal/day can be found in the Appendix (tables A.3-A.5).

are written in the vector $E$.

Since our model has a recursive structure it is sufficient that there exists a variable which affects the outcome in the first stage equation, but can be excluded from the second stage. In equation (3), three variables are excluded from the second stage: $S(I < p50)_i$ and $S(I \geq p50)_i$ are interactions between hunger and income which both occur in the second stage. Thus they are unlikely to work as exclusion restrictions. The variable $HHsize$ measures the number of people currently living in the household. In each equation we control for whether the respondent lives with a partner or not, because it has an impact on the food share as well as on health outcomes (see literature on marriage selection and marriage protection, for instance, Hu and Goldman (1990) or Waite (1995)). After having controlled for partnership, the measure of household size still helps to predict the food share, but should not have any predictive power for late life health outcomes. Thus, household size is used as exclusion restriction in the second stage providing just identification.

For BMI as health outcome, we can estimate two linear equations. Having overweight and diabetes are binary outcome variables and require the estimation of a probit model on the second stage. For these two outcomes we therefore set up a mixed process model as suggested by Roodman (2009). For the estimation of both models we apply a limited maximum likelihood approach (LIML).

Table 4.9 displays the results from the estimation of equation (3) for BMI. We find a significantly higher BMI for respondents who have high food shares. The results suggest that, once we control for a direct hunger and income effect, the coefficient for food share represents a behavioral effect of hunger expressed by changed food consumption behavior. The coefficients measuring the impact of the hunger experience are not significantly different from zero. This indicates that there are no direct biological or other behavioral effects of hunger on BMI at any ages during childhood.

The patterns found for the estimation of BMI are broadly reflected in the estimation of the other two health outcomes (tables 4.10-4.11). Higher food share significantly increases the probability of having overweight if controlled for direct hunger and income effects, for age 11-16 and 0-16. A significant direct hunger effect is found for age 0-4 suggesting that at this age also other (presumably biological) mechanisms are responsible for a higher probability of having overweight. In the probit regression on diabetes, the effects of food share are not significantly different from zero. Moreover, the effect of hunger on diabetes is positive and significantly different from zero if the respondents suffered from hunger at age 5-10. This effect can again be considered as a direct effect of hunger through biological or other behavioral mechanisms.

The results from the simultaneous equation estimation suggest that late life outcomes are not solely affected by the experience of hunger during childhood through any biological or not further identified mechanisms, but also through a change in food consumption behavior. This confirms our hypothesis that individuals who suffer from hunger in early life spend more on food as a precaution against the risk of new hunger episodes. In particular, this link seems to be significant for the group aged 11-16. One explanation for this might be that those individuals are just old enough to being fully exposed

to the hunger episode after World War II[16]. As a consequence, these individuals changed their food spending behavior and thus hold worse levels of health later in life.

### 4.5 Conclusion

In this study, we show that an adverse life event during childhood affects not only adult health outcomes but also individuals' behavior at older ages (and thus presumably over the whole life-cycle). While the health effects of shocks in utero and at various stages of childhood on adult health are well established, most of the existing literature focused on biological channels. Our results suggest another causal link: Retrospective survey data on hunger experiences in post-war Germany, combined with administrative data on food supply (caloric rations) during those times and detailed data on adult outcomes, show that relatively short periods of severe hunger affect not only health outcomes of older individuals (higher body weight and higher rates of obesity) but also food consumption patterns as well as measures related to preferences (trust and risk aversion).

Our results further show that the behavioral effects vary with the age at which children were affected. In particular, ages 11-16 seem to be a sensitive period during childhood. This suggests that policy interventions focusing on this group of children might be particularly useful and complements existing research that stressed biological channels for which the most sensitive periods of childhood are much earlier. If the pathways between early-life shocks and late-life outcomes are not only biological but also behavioral, then there are ampler possibilities to counteract these pathways.

We also contribute to the literature on endogenous preference formation. Our finding that preferences are not only influenced by preferences of parents or role models as in Dohmen *et al.* (2011), but also by large shocks experienced during childhood and youth, provides a complementary explanation for a correlation of preferences between generations. Perhaps more importantly, this result further strengthens the case for interventions in early childhood and adolescence, as summarized by Heckman (2012).

[16] Individuals that have been in their adolescence after World War II might have had younger siblings to which parents gave their food rations. At the same time they might have taken care for the family.

## Tables and Figures

Table 4.1: German states by occupation zones

| **occupation zones** | **states** |
|---|---|
| French | Rhineland-Palatinate, southern part of Baden Wuerttemberg |
| US | Bavaria, northern part of Baden-Wuerttemberg, Hesse, Bremen |
| UK | North Rhine-Westphalia, Lower Saxony, Hamburg, Schleswig-Holstein |
| Soviet | Thuringia, Saxony-Anhalt, Saxony, Brandenburg, Mecklenburg-Vorpommern |

Table 4.2: OLS regressions on months below 1500 kcal/day, ages 0-16, 0-4, 5-10, 11-16

| **VARIABLES** | (1)<br>**hunger**<br>**age 0-16** | (2)<br>**hunger**<br>**age 0-4** | (3)<br>**hunger**<br>**age 5-10** | (4)<br>**hunger**<br>**age 11-16** |
|---|---|---|---|---|
| cal months below 1500 age 00-04 | | 0.002** | | |
| | | [0.001] | | |
| cal months below 1500 age 05-10 | | | 0.007*** | |
| | | | [0.001] | |
| cal months below 1500 age 11-16 | | | | 0.009*** |
| | | | | [0.001] |
| cal months below 1500 age 00-16 | 0.006*** | | | |
| | [0.001] | | | |
| urban (at birth) | 0.041 | 0.035** | 0.033 | 0.010 |
| | [0.026] | [0.016] | [0.022] | [0.018] |
| US zone (at birth) | -0.009 | -0.012 | -0.002 | -0.014 |
| | [0.048] | [0.034] | [0.041] | [0.037] |
| UK zone (at birth) | 0.039 | -0.026 | 0.016 | 0.022 |
| | [0.047] | [0.033] | [0.040] | [0.037] |
| Soviet zone (at birth) | 0.038 | -0.042 | 0.028 | 0.034 |
| | [0.048] | [0.032] | [0.042] | [0.037] |
| constant | 0.001 | 0.044 | 0.017 | 0.011 |
| | [0.045] | [0.030] | [0.038] | [0.035] |
| observations | 878 | 878 | 878 | 878 |
| R-squared | 0.075 | 0.023 | 0.092 | 0.140 |

Robust tandard errors in brackets, *** p<0.01 ** p<0.05 * p<0.1;

Table 4.3: Construction of food share sample

| **sample description** | **N** |
|---|---|
| respondents that participated in wave 1 and wave 3 of SHARE | 1,763 |
| food share | 1,071 |
| only native born respondents | 1,555 |
| food share | 938 |
| with wave 2 information | |
| food share | 1,028 |
| without outliers | |
| food share | 1,005 |
| without respondents born before 1929 | |
| food share | 923 |

Table 4.4: Engel curve estimation on self-reported hunger, median-income splines and interactions

| **VARIABLES** | (1)<br>**food share** | (2)<br>**food share** | (3)<br>**food share** | (4)<br>**food share** |
|---|---|---|---|---|
| log gross income | -0.105*** | -0.104*** | -0.105*** | -0.106*** |
| | [0.005] | [0.005] | [0.005] | [0.006] |
| hunger age 00-16 | 0.366 | | | |
| | [0.259] | | | |
| hunger age 00-16×loginc(lo) | -0.048 | | | |
| | [0.033] | | | |
| hunger age 00-16×loginc(hi) | 0.003 | | | |
| | [0.016] | | | |
| hunger age 00-04 | | 1.132** | | |
| | | [0.474] | | |
| hunger age 00-04×loginc(lo) | | -0.142** | | |
| | | [0.061] | | |
| hunger age 00-04×loginc(hi) | | -0.020 | | |
| | | [0.038] | | |
| hunger age 05-10 | | | 0.292 | |
| | | | [0.326] | |
| hunger age 05-10×loginc(lo) | | | -0.036 | |
| | | | [0.042] | |
| hunger age 05-10×loginc(hi) | | | -0.007 | |
| | | | [0.019] | |
| hunger age 11-16 | | | | 0.580** |
| | | | | [0.268] |
| hunger age 11-16×loginc(lo) | | | | -0.078** |
| | | | | [0.035] |
| hunger age 11-16×loginc(hi) | | | | 0.035** |
| | | | | [0.017] |
| constant | 0.492* | 0.639** | 0.593** | 0.554** |
| | [0.251] | [0.249] | [0.250] | [0.244] |
| observations | 878 | 878 | 878 | 878 |
| R-squared | 0.518 | 0.526 | 0.517 | 0.520 |

Standard errors in brackets, clustered on the household level; *** p<0.01 ** p<0.05 * p<0.1; food share is defined as the sum of the monthly amount spent on food at home and outside divided by the monthly gross income; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and household size.

Table 4.5: Engel curve estimation on months below 1500 kcal/day, median-income splines and interactions)

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **food share** | **food share** | **food share** | **food share** |
| log gross income | -0.093*** | -0.104*** | -0.102*** | -0.105*** |
| | [0.006] | [0.006] | [0.006] | [0.006] |
| cal months below 1500 age 00-16 | 0.016*** | | | |
| | [0.004] | | | |
| months below 1500 age 00-16×loginc(lo) | -0.002*** | | | |
| | [0.000] | | | |
| months below 1500 age 00-16×loginc(hi) | 0.001* | | | |
| | [0.000] | | | |
| cal months below 1500 age 00-04 | | 0.018** | | |
| | | [0.008] | | |
| months below 1500 age 00-04×loginc(lo) | | -0.002** | | |
| | | [0.001] | | |
| months below 1500 age 00-04×loginc(hi) | | 0.001** | | |
| | | [0.001] | | |
| cal months below 1500 age 05-10 | | | 0.018*** | |
| | | | [0.006] | |
| months below 1500 age 05-10×loginc(lo) | | | -0.002*** | |
| | | | [0.001] | |
| months below 1500 age 05-10×loginc(hi) | | | 0.001*** | |
| | | | [0.000] | |
| cal months below 1500 age 11-16 | | | | 0.015* |
| | | | | [0.008] |
| months below 1500 age 11-16×loginc(lo) | | | | -0.002* |
| | | | | [0.001] |
| months below 1500 age 11-16×loginc(hi) | | | | 0.001 |
| | | | | [0.001] |
| constant | 0.380 | 0.631** | 0.454 | 0.558** |
| | [0.329] | [0.286] | [0.282] | [0.255] |
| observations | 878 | 878 | 878 | 878 |
| R-squared | 0.538 | 0.523 | 0.526 | 0.520 |

Standard errors in brackets, clustered on the household level; *** p<0.01 ** p<0.05 * p<0.1; food share is defined as the sum of the monthly amount spent on food at home and outside divided by the monthly gross income; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and household size.

Table 4.6: OLS regression BMI on self-reported hunger, median-income spines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **BMI** | **BMI** | **BMI** | **BMI** |
| log gross income | -0.695*** | -0.680*** | -0.697*** | -0.709*** |
| | [0.227] | [0.215] | [0.226] | [0.232] |
| hunger age 00-16 | 24.305*** | | | |
| | [9.414] | | | |
| hunger age 00-16×loginc(lo) | -3.129** | | | |
| | [1.229] | | | |
| hunger age 00-16×loginc(hi) | 1.856 | | | |
| | [1.313] | | | |
| hunger age 00-04 | | 61.070 | | |
| | | [37.112] | | |
| hunger age 00-04×loginc(lo) | | -7.789 | | |
| | | [4.825] | | |
| hunger age 00-04×loginc(hi) | | 4.168 | | |
| | | [3.305] | | |
| hunger age 05-10 | | | 18.967 | |
| | | | [13.057] | |
| hunger age 05-10×loginc(lo) | | | -2.366 | |
| | | | [1.707] | |
| hunger age 05-10×loginc(hi) | | | 1.047 | |
| | | | [1.575] | |
| hunger age 11-16 | | | | 25.255*** |
| | | | | [7.405] |
| hunger age 11-16×loginc(lo) | | | | -3.351*** |
| | | | | [0.987] |
| hunger age 11-16×loginc(hi) | | | | 1.565 |
| | | | | [1.171] |
| constant | 32.016*** | 40.538*** | 38.370*** | 32.092*** |
| | [11.949] | [11.608] | [11.815] | [11.914] |
| observations | 876 | 876 | 876 | 876 |
| R-squared | 0.074 | 0.080 | 0.069 | 0.067 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table 4.7: Probit regression overweight on self-reported hunger, median-income spines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **overweight** | **overweight** | **overweight** | **overweight** |
| loginc | -0.275*** | -0.294*** | -0.267*** | -0.261*** |
| | [0.074] | [0.071] | [0.073] | [0.072] |
| hunger age 00-16 | 8.796*** | | | |
| | [3.182] | | | |
| hunger age 00-16×loginc(lo) | -1.154*** | | | |
| | [0.415] | | | |
| hunger age 00-16×loginc(hi) | 0.709* | | | |
| | [0.389] | | | |
| hunger age 00-04 | | 2.286 | | |
| | | [6.912] | | |
| hunger age 00-04×loginc(lo) | | -0.286 | | |
| | | [0.903] | | |
| hunger age 00-04×loginc(hi) | | 1.199 | | |
| | | [0.839] | | |
| hunger age 05-10 | | | 7.298* | |
| | | | [3.923] | |
| hunger age 05-10×loginc(lo) | | | -0.936* | |
| | | | [0.511] | |
| hunger age 05-10×loginc(hi) | | | 0.401 | |
| | | | [0.417] | |
| hunger age 11-16 | | | | 15.094*** |
| | | | | [4.405] |
| hunger age 11-16×loginc(lo) | | | | -2.000*** |
| | | | | [0.578] |
| hunger age 11-16×loginc(hi) | | | | 0.607 |
| | | | | [0.465] |
| constant | 7.059* | 8.770** | 7.757** | 7.922** |
| | [3.853] | [3.878] | [3.889] | [3.896] |
| observations | 876 | 876 | 876 | 876 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table 4.8: Probit regression diabetes on self-reported hunger, median-income spines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **diabetes** | **diabetes** | **diabetes** | **diabetes** |
| log gross income | -0.142 | -0.179* | -0.163 | -0.162* |
| | [0.099] | [0.096] | [0.099] | [0.095] |
| hunger age 00-16 | 3.575 | | | |
| | [2.502] | | | |
| hunger age 00-16×loginc(lo) | -0.434 | | | |
| | [0.330] | | | |
| hunger age 00-16×loginc(hi) | -0.521 | | | |
| | [0.519] | | | |
| hunger age 00-04 | | -0.387 | | |
| | | [5.596] | | |
| hunger age 00-04×loginc(lo) | | 0.139 | | |
| | | [0.739] | | |
| hunger age 00-04×loginc(hi) | | -1.086 | | |
| | | [0.951] | | |
| hunger age 05-10 | | | 1.424 | |
| | | | [2.970] | |
| hunger age 05-10×loginc(lo) | | | -0.132 | |
| | | | [0.393] | |
| hunger age 05-10×loginc(hi) | | | -0.691 | |
| | | | [0.623] | |
| hunger age 11-16 | | | | 7.107** |
| | | | | [3.413] |
| hunger age 11-16×loginc(lo) | | | | -0.911** |
| | | | | [0.452] |
| hunger age 11-16×loginc(hi) | | | | -0.445 |
| | | | | [0.786] |
| constant | -4.699 | -2.076 | -1.897 | -5.690 |
| | [5.114] | [5.091] | [5.148] | [5.300] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table 4.9: 2.stage results of simultaneous equation estimation: BMI on foodshare

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **BMI** | **BMI** | **BMI** | **BMI** |
| food share×100 | 0.349** | 0.275** | 0.229 | 0.256** |
| | [0.150] | [0.124] | [0.158] | [0.121] |
| log gross income | 3.140* | 2.327* | 1.810 | 2.105 |
| | [1.695] | [1.402] | [1.776] | [1.370] |
| hunger age 00-16 | 0.600 | | | |
| | [0.496] | | | |
| hunger age 00-04 | | 1.366 | | |
| | | [0.866] | | |
| hunger age 05-10 | | | 0.761 | |
| | | | [0.555] | |
| hunger age 11-16 | | | | 0.204 |
| | | | | [0.632] |
| constant | 10.864 | 19.646 | 22.755 | 16.317 |
| | [17.824] | [16.704] | [18.093] | [16.186] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education, whether being partnered; household size is excluded from this 2.stage equation; food share was multiplied by factor 100.

Table 4.10: 2.stage results of simultaneous equation estimation: overweight on foodshare

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **overweight** | **overweight** | **overweight** | **overweight** |
| food share×100 | 0.084*** | -0.015 | 0.059 | 0.080*** |
| | [0.025] | [0.036] | [0.040] | [0.026] |
| log gross income | 0.726** | -0.439 | 0.412 | 0.673* |
| | [0.332] | [0.391] | [0.492] | [0.344] |
| hunger age 00-16 | 0.029 | | | |
| | [0.124] | | | |
| hunger age 00-04 | | 0.455* | | |
| | | [0.252] | | |
| hunger age 05-10 | | | 0.073 | |
| | | | [0.164] | |
| hunger age 11-16 | | | | -0.057 |
| | | | | [0.197] |
| constant | -0.273 | 9.694** | 2.536 | 0.820 |
| | [4.579] | [4.692] | [5.783] | [4.869] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education, whether being partnered; household size is excluded from this 2.stage equation; food share was multiplied by factor 100.

Table 4.11: 2.stage results of simultaneous equation estimation: diabetes on foodshare

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **diabetes** | **diabetes** | **diabetes** | **diabetes** |
| food share×100 | 0.028 | 0.005 | -0.021 | 0.049 |
| | [0.048] | [0.042] | [0.061] | [0.036] |
| log gross income | 0.127 | -0.139 | -0.430 | 0.367 |
| | [0.565] | [0.478] | [0.674] | [0.435] |
| hunger age 00-16 | 0.195 | | | |
| | [0.162] | | | |
| hunger age 00-04 | | 0.438 | | |
| | | [0.293] | | |
| hunger age 05-10 | | | 0.344* | |
| | | | [0.180] | |
| hunger age 11-16 | | | | 0.241 |
| | | | | [0.201] |
| constant | -5.251 | -2.426 | 0.287 | -8.110 |
| | [6.266] | [6.286] | [7.285] | [5.667] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; We control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education, whether being partnered; household size is excluded from this 2.stage equation; food share was multiplied by factor 100.

Figure 4.1: Self-reported hunger by SHARE countries, 1920-1960

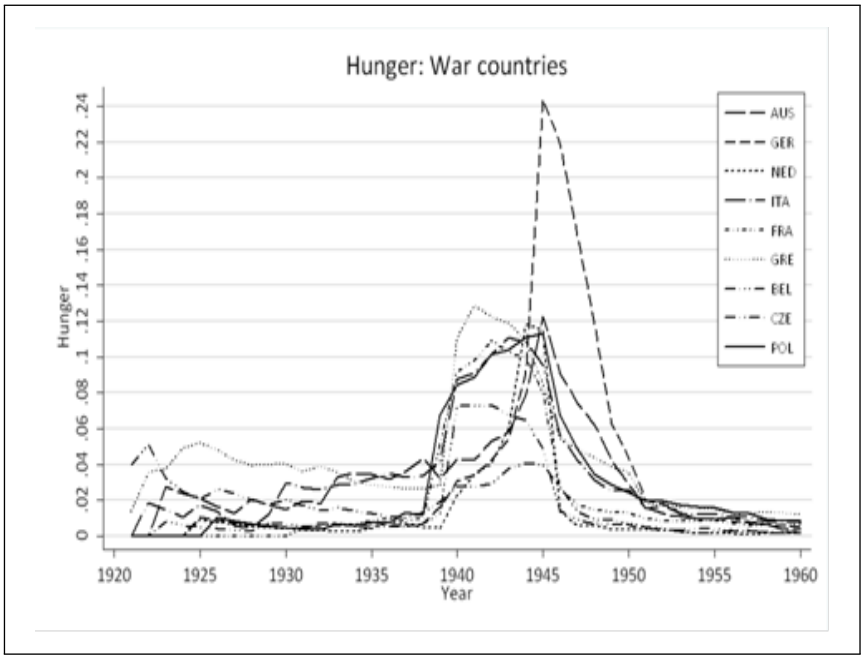



Figure 4.2: Amount of official caloric intake per day and fraction of self-reported hunger, January 1930-January 1970

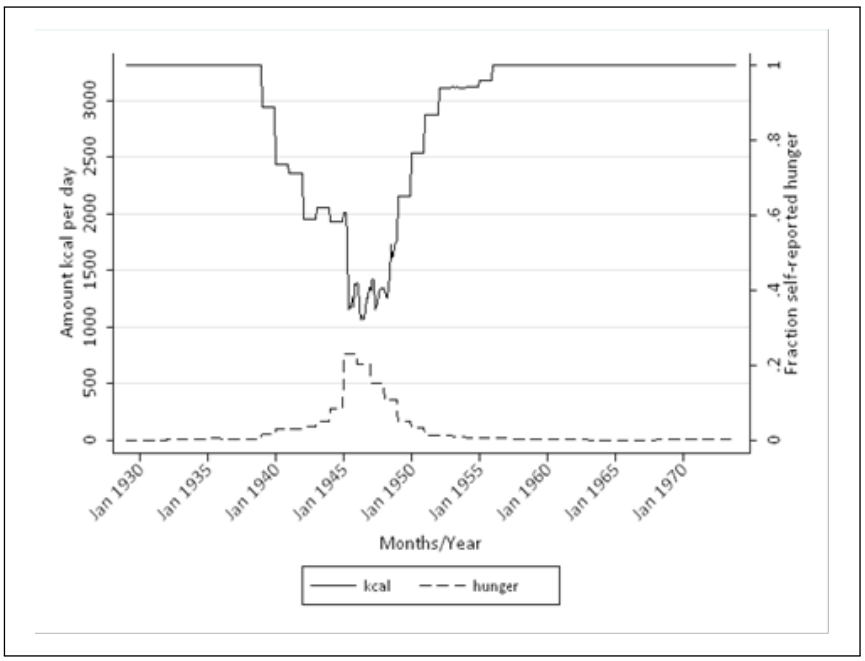


Figure 4.3: Concentration exposure to hunger at age 0-16, the straight lines refer to birth cohorts 1939, 1945, and 1949

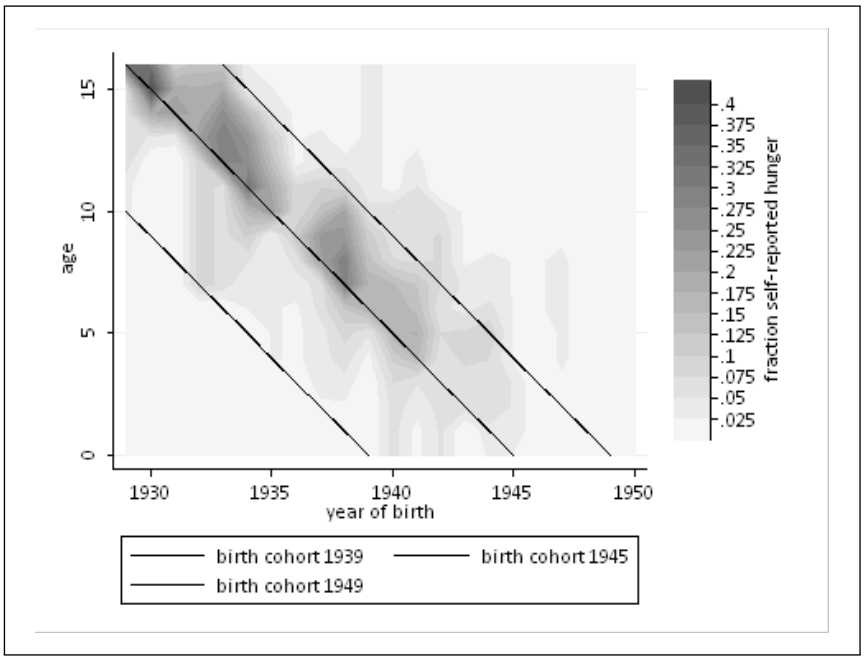



Figure 4.4: Fraction of self-reported hunger by socioeconomic status, 1930-1960

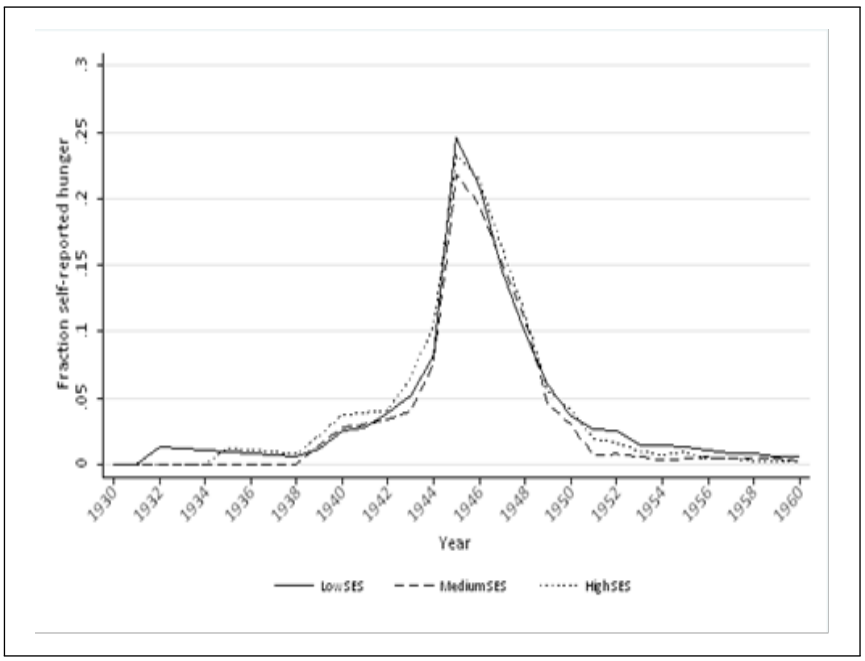
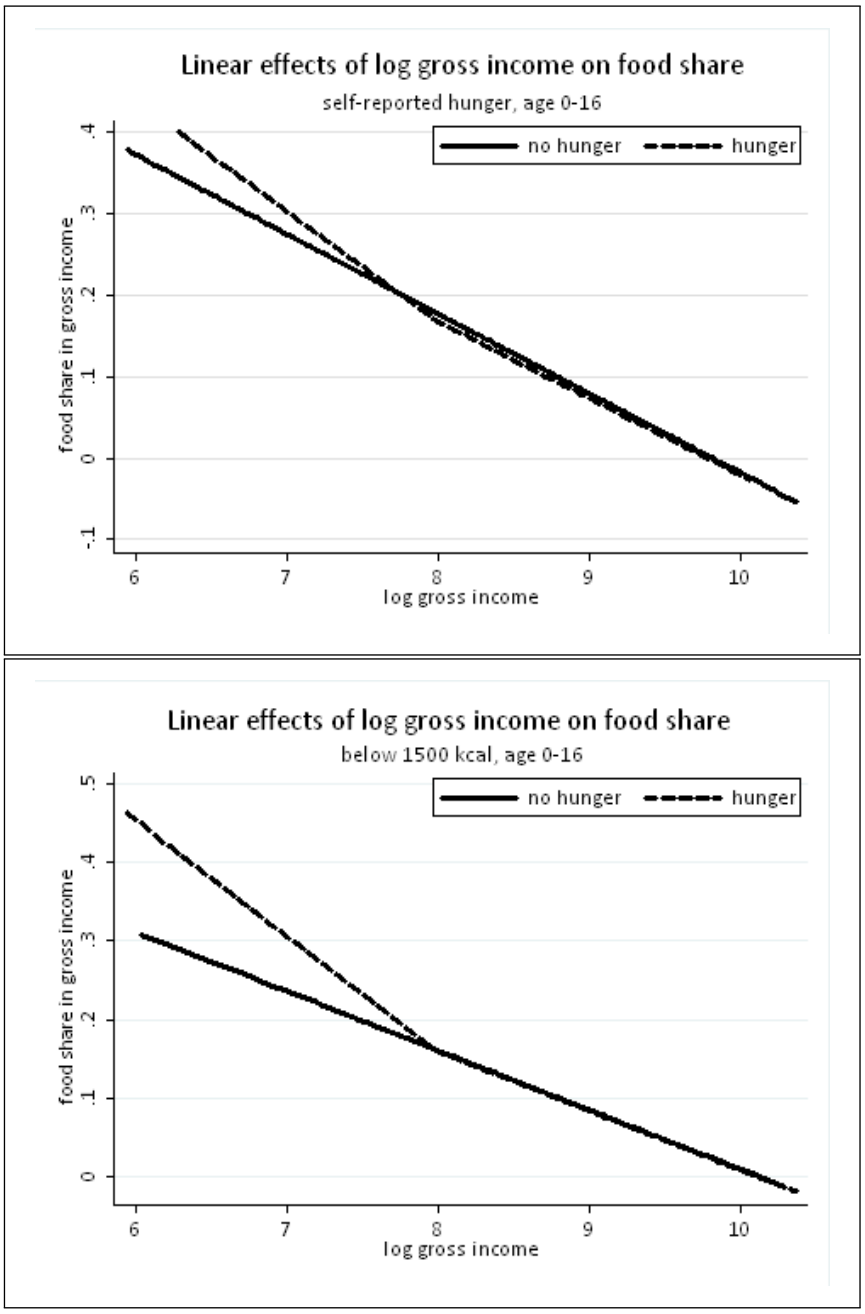


Figure 4.5: Engel curve relationship by self-reported hunger and caloric threshold of 1500 kcal, age 0-16



# Appendix

Table A.1: Regression of risk aversion on self-reported hunger

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **risk aversion** | **risk aversion** | **risk aversion** | **risk aversion** |
| log gross income | -0.112*** | -0.112*** | -0.112*** | -0.114*** |
| | [0.033] | [0.033] | [0.033] | [0.033] |
| hunger age 00-16 | 0.107* | | | |
| | [0.060] | | | |
| hunger age 00-04 | | 0.258*** | | |
| | | [0.078] | | |
| hunger age 05-10 | | | 0.108 | |
| | | | [0.068] | |
| hunger age 11-16 | | | | -0.008 |
| | | | | [0.089] |
| constant | 6.558*** | 7.177*** | 6.908*** | 6.451*** |
| | [1.801] | [1.836] | [1.825] | [1.849] |
| observations | 544 | 544 | 544 | 544 |
| R-squared | 0.111 | 0.115 | 0.110 | 0.107 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and household size.

Table A.2: Regression of trust on self-reported hunger

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **trust** | **trust** | **trust** | **trust** |
| log gross income | 0.088 | 0.084 | 0.085 | 0.087 |
| | [0.130] | [0.129] | [0.130] | [0.130] |
| hunger age 00-16 | -0.041 | | | |
| | [0.282] | | | |
| hunger age 00-04 | | -0.836* | | |
| | | [0.497] | | |
| hunger age 05-10 | | | -0.205 | |
| | | | [0.330] | |
| hunger age 11-16 | | | | -0.139 |
| | | | | [0.413] |
| constant | -5.623 | -7.964 | -6.642 | -4.979 |
| | [7.565] | [7.607] | [7.656] | [7.847] |
| observations | 801 | 801 | 801 | 801 |
| R-squared | 0.045 | 0.049 | 0.046 | 0.045 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and household size.

Table A.3: OLS regression BMI on months below 1500 kcal/day, median-income splines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **BMI** | **BMI** | **BMI** | **BMI** |
| log gross income | -0.632* | -0.596** | -0.939*** | -0.693*** |
| | [0.358] | [0.257] | [0.273] | [0.246] |
| cal months below 1500 age 00-16 | 0.111 | | | |
| | [0.136] | | | |
| months below 1500 age 00-16×loginc(lo) | -0.017 | | | |
| | [0.018] | | | |
| months below 1500 age 00-16×loginc(hi) | 0.001 | | | |
| | [0.018] | | | |
| cal months below 1500 age 00-04 | | 0.14 | | |
| | | [0.311] | | |
| months below 1500 age 00-04×loginc(lo) | | -0.017 | | |
| | | [0.040] | | |
| months below 1500 age 00-04×loginc(hi) | | -0.029 | | |
| | | [0.025] | | |
| cal months below 1500 age 05-10 | | | -0.197 | |
| | | | [0.170] | |
| months below 1500 age 05-10×loginc(lo) | | | 0.022 | |
| | | | [0.022] | |
| months below 1500 age 05-10×loginc(hi) | | | 0.019 | |
| | | | [0.024] | |
| cal months below 1500 age 11-16 | | | | 0.402** |
| | | | | [0.196] |
| months below 1500 age 11-16×loginc(lo) | | | | -0.051** |
| | | | | [0.026] |
| months below 1500 age 11-16 × loginc(hi) | | | | 0.031 |
| | | | | [0.043] |
| constant | 18.52 | 31.306** | 24.766* | 31.763*** |
| | [18.922] | [12.982] | [14.286] | [11.778] |
| observations | 876 | 876 | 876 | 876 |
| R-squared | 0.062 | 0.062 | 0.064 | 0.064 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table A.4: Probit regression overweight on months below 1500 kcal/day, median-income splines, and interactions

| **VARIABLES** | (1) **overweight** | (2) **overweight** | (3) **overweight** | (4) **overweight** |
|---|---|---|---|---|
| log gross income | -0.216** | -0.248*** | -0.332*** | -0.225*** |
| | [0.099] | [0.079] | [0.083] | [0.074] |
| cal months below 1500 age 00-16 | 0.006 | | | |
| | [0.044] | | | |
| months below 1500 age 00-16×loginc(lo) | 0 | | | |
| | [0.006] | | | |
| months below 1500 age 00-16×loginc(hi) | -0.006 | | | |
| | [0.005] | | | |
| cal months below 1500 age 00-04 | | -0.139* | | |
| | | [0.082] | | |
| months below 1500 age 00-04×loginc(lo) | | 0.020* | | |
| | | [0.011] | | |
| months below 1500 age 00-04×loginc(hi) | | -0.026** | | |
| | | [0.011] | | |
| cal months below 1500 age 05-10 | | | -0.05 | |
| | | | [0.070] | |
| months below 1500 age 05-10×loginc(lo) | | | 0.005 | |
| | | | [0.009] | |
| months below 1500 age 05-10×loginc(hi) | | | 0.005 | |
| | | | [0.007] | |
| cal months below 1500 age 11-16 | | | | 0.223** |
| | | | | [0.099] |
| months below 1500 age 11-16×loginc(lo) | | | | -0.028** |
| | | | | [0.013] |
| months below 1500 age 11-16×loginc(hi) | | | | -0.008 |
| | | | | [0.014] |
| constant | 6.915 | 8.530** | 3.306 | 5.519 |
| | [5.973] | [4.275] | [4.674] | [3.820] |
| observations | 876 | 876 | 876 | 876 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table A.5: Probit regression diabetes on months below 1500 kcal/day, median-income splines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **diabetes** | **diabetes** | **diabetes** | **diabetes** |
| log gross income | -0.074 | -0.212** | -0.167 | -0.183* |
| | [0.148] | [0.106] | [0.118] | [0.098] |
| cal months below 1500 age 00-16 | 0.042 | | | |
| | [0.055] | | | |
| months below 1500 age 00-16×loginc(lo) | -0.006 | | | |
| | [0.007] | | | |
| months below 1500 age 00-16×loginc(hi) | -0.007 | | | |
| | [0.008] | | | |
| cal months below 1500 age 00-04 | | 0.058 | | |
| | | [0.098] | | |
| months below 1500 age 00-04×loginc(lo) | | -0.01 | | |
| | | [0.013] | | |
| months below 1500 age 00-04×loginc(hi) | | 0.012 | | |
| | | [0.013] | | |
| cal months below 1500 age 05-10 | | | -0.013 | |
| | | | [0.081] | |
| months below 1500 age 05-10×loginc(lo) | | | 0.002 | |
| | | | [0.011] | |
| months below 1500 age 05-10×loginc(hi) | | | -0.012 | |
| | | | [0.011] | |
| cal months below 1500 age 11-16 | | | | 0.079 |
| | | | | [0.083] |
| months below 1500 age 11-16×loginc(lo) | | | | -0.01 |
| | | | | [0.011] |
| months below 1500 age 11-16×loginc(hi) | | | | 0.005 |
| | | | | [0.016] |
| constant | -13.308 | -6.092 | -3.079 | -3.775 |
| | [9.131] | [5.506] | [6.333] | [5.160] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered.

Table A.6: 1.stage results of simultaneous equation estimation with BMI as 2.stage outcome: food-share on self-reported hunger, median-income splines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **fs×100** | **fs×100** | **fs×100** | **fs×100** |
| log gross income | -11.013*** | -10.920*** | -11.040*** | -11.133*** |
| | [0.420] | [0.409] | [0.421] | [0.410] |
| hunger age 00-16 | 52.374*** | | | |
| | [12.975] | | | |
| hunger age 00-16×loginc(lo) | -6.872*** | | | |
| | [1.708] | | | |
| hunger age 00-16×loginc(hi) | 2.875* | | | |
| | [1.723] | | | |
| hunger age 00-04 | | 135.009*** | | |
| | | [31.449] | | |
| hunger age 00-04×loginc(lo) | | -17.162*** | | |
| | | [4.136] | | |
| hunger age 00-04×loginc(hi) | | 1.862 | | |
| | | [3.866] | | |
| hunger age 05-10 | | | 44.574*** | |
| | | | [16.190] | |
| hunger age 05-10×loginc(lo) | | | -5.686*** | |
| | | | [2.136] | |
| hunger age 05-10×loginc(hi) | | | 1.362 | |
| | | | [2.201] | |
| hunger age 11-16 | | | | 73.121*** |
| | | | | [17.456] |
| hunger age 11-16×loginc(lo) | | | | -9.846*** |
| | | | | [2.313] |
| hunger age 11-16×loginc(hi) | | | | 6.176** |
| | | | | [2.746] |
| constant | 59.644*** | 73.144*** | 69.379*** | 64.057*** |
| | [22.738] | [22.667] | [23.015] | [22.919] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered; food share was multiplied with 100.

Table A.7: 1.stage results of simultaneous equation estimation with overweight as 2.stage outcome: foodshare on self-reported hunger, median-income splines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **fs×100** | **fs×100** | **fs×100** | **fs×100** |
| log gross income | -11.027*** | -10.899*** | -11.043*** | -11.101*** |
| | [0.420] | [0.410] | [0.421] | [0.411] |
| hunger age 00-16 | 52.779*** | | | |
| | [13.118] | | | |
| hunger age 00-16×loginc(lo) | -6.933*** | | | |
| | [1.726] | | | |
| hunger age 00-16×loginc(hi) | 3.200* | | | |
| | [1.675] | | | |
| hunger age 00-04 | | 112.138*** | | |
| | | [35.140] | | |
| hunger age 00-04×loginc(lo) | | -14.041*** | | |
| | | [4.637] | | |
| hunger age 00-04×loginc(hi) | | -2.204 | | |
| | | [4.305] | | |
| hunger age 05-10 | | | 45.037*** | |
| | | | [16.567] | |
| hunger age 05-10×loginc(lo) | | | -5.750*** | |
| | | | [2.185] | |
| hunger age 05-10×loginc(hi) | | | 1.484 | |
| | | | [2.212] | |
| hunger age 11-16 | | | | 76.492*** |
| | | | | [17.297] |
| hunger age 11-16×loginc(lo) | | | | -10.280*** |
| | | | | [2.291] |
| hunger age 11-16×loginc(hi) | | | | 5.718** |
| | | | | [2.667] |
| constant | 60.189*** | 72.213*** | 69.445*** | 64.098*** |
| | [22.751] | [22.647] | [23.018] | [22.928] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered; food share was multiplied with 100.

Table A.8: 1.stage results of simultaneous equation estimation with diabetes as 2.stage outcome: foodshare on self-reported hunger, median-income splines, and interactions

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **VARIABLES** | **fs×100** | **fs×100** | **fs×100** | **fs×100** |
| log gross income | -10.993*** | -10.910*** | -11.079*** | -11.131*** |
| | [0.428] | [0.411] | [0.427] | [0.412] |
| hunger age 00-16 | 47.372*** | | | |
| | [14.384] | | | |
| hunger age 00-16×loginc(lo) | -6.188*** | | | |
| | [1.894] | | | |
| hunger age 00-16×loginc(hi) | 1.743 | | | |
| | [2.058] | | | |
| hunger age 00-04 | | 114.124*** | | |
| | | [34.702] | | |
| hunger age 00-04×loginc(lo) | | -14.323*** | | |
| | | [4.574] | | |
| hunger age 00-04×loginc(hi) | | -1.557 | | |
| | | [4.208] | | |
| hunger age 05-10 | | | 36.506** | |
| | | | [18.383] | |
| hunger age 05-10×loginc(lo) | | | -4.625* | |
| | | | [2.418] | |
| hunger age 05-10×loginc(hi) | | | 1.062 | |
| | | | [2.382] | |
| hunger age 11-16 | | | | 72.819*** |
| | | | | [18.074] |
| hunger age 11-16×loginc(lo) | | | | -9.804*** |
| | | | | [2.393] |
| hunger age 11-16×loginc(hi) | | | | 6.026** |
| | | | | [2.929] |
| constant | 58.399** | 72.191*** | 69.414*** | 63.980*** |
| | [22.730] | [22.647] | [23.020] | [22.922] |
| observations | 878 | 878 | 878 | 878 |

Robust standard errors in brackets; *** p<0.01 ** p<0.05 * p<0.1; we control for age, age squared, gender, born in a rural/urban region, occupation zone at birth, years of education and whether being partnered; food share was multiplied with 100.

# Tables

# Figures

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