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## Does stress shorten your life? Evidence from parental bereavement

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Bernhard Schmidpeter

## **Does Stress Shorten Your Life? Evidence from Parental Bereavement**

## Imprint

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**Does Stress Shorten Your Life?  
Evidence from Parental Bereavement**

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Bernhard Schmidtpeter<sup>1</sup>

# Does Stress Shorten Your Life? Evidence from Parental Bereavement

## Abstract

*This paper studies how stress affects the mortality risk. Using a flexible approach and allowing for time-varying treatment effects, I find no impact of stress on the short-run mortality risk but a substantially increase in the long-run. The effects are especially pronounced for men. I provide evidence that this is likely caused by changes towards adverse health behaviours as a reaction to stress. Investigating the possible protective effects of mental health support, I find that it can substantially lower the mortality risk for women. The results for men point towards lower effectiveness likely due to stigma effects associated with mental health care. Finally, I show that my results are robust to specific departure of my identifying assumptions.*

*JEL-Code: I12, I11, C31, C41*

*Keywords: Stress; mortality; mental health; bereavement; propensity score weighting; adjusted Kaplan-Meier Estimator; direct effects; indirect effects*

*December 2019*

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# 1 Introduction

High levels of stress are ubiquitous in everyday life. An increasing number of adults in the US report levels of extreme stress caused by work and financial pressure ([American Psychological Association, 2015](#)). Similarly, every fifth worker in Europe states that s/he has constant stress at work or suffers from other stress-related health issues ([European Agency for Safety and Health at Work, 2009](#)). Amid the recent economic crisis and fears of job insecurity this number is likely to increase in the future.

High and persistent levels of stress have been associated with various adverse health outcomes. They have been linked to general mental health problems, depression, and physical problems (see, for example, [Mohr, Hart, Julian, Cox and Pelletier \(2004\)](#), [Hammen \(2005\)](#), [van den Berg, Lundborg and Vikstrom \(2017\)](#), and the articles in [Conrad \(2011\)](#) among others). There is evidence that alleviated stress levels during pregnancy can affect the health and educational attainment of the offspring ([Aizer, Stroud and Buka, 2015](#); [Persson and Rossin-Slater, 2018](#)). In the worst case, stress can increase mortality. Despite the interest in the negative consequences of stress, not much is known about its dynamics and the interaction with health care utilization, in particular mental health support. Obtaining a better understanding about the consequences of stress is, however, important for implementations of effective public health policies.

In this paper, I examine the dynamic effects of a sudden rise in stress on the mortality risk using administrative data for Austria. To identify changes to stress levels, I use the unexpected death of a child. A similar strategy has been used in various public health and epidemiology studies (e.g. [Levav, Friedlander, Kark and Peritz, 1988](#); [Li, Precht, Mortensen and Olsen, 2003](#); [Rostila, Saarela and Kawachi, 2012](#)), and economic research (e.g. [Black, Devereux and Salvanes, 2016](#); [van den Berg et al., 2017](#); [Persson and Rossin-Slater, 2018](#)). Importantly, in my empirical application I neither restrict the dynamics of how stress evolves over time nor the underlying heterogeneity of individuals. This is important as any ad-hoc modeling assumptions can lead to biased results.

Then, I go beyond establishing the dynamic effects of stress on mortality and investigate if provided mental health support can alleviate its negative consequences. To do so, I decompose my estimates into an (indirect) effect which affects the mortality risk through the utilization of mental health support shortly after the stressful event and an (direct) effect which accounts for all other channels.

I find that a sudden increase in stress levels has a non-monotonic impact on the mortality risk over time. For women, I find that the mortality risk remains relatively flat during the first years after the stressful event. Afterwards, I estimate a continuous increase until its peak in year ten which is followed by a reversal towards zero. In general, the estimates for this group are largely insignificant and with a maximum increase by 1 percentage point also rather moderate. For men, I estimate a similar flat impact during

the first years. In contrast to my results for women, I estimate a sudden and strong increase in the mortality risk during the second half of my observation period. At its high 13 years after the stressful event, affected men have a 6 percentage points higher mortality risk with no sign of reversal thereafter. I provide evidence that one of the main drivers behind these results is the change towards adverse health behaviour, such as excessive drinking and smoking. In my analysis, I show that the results are robust to specific failures of my identifying assumption.

To gain a better understanding what drives the pronounced gender differences in the mortality risk, I apply a decomposition analysis to my dynamic effects. I find that mental health support shortly after the stressful event can lower the overall mortality risk for women by up to 10%. While I find in general protective effects of mental health support for men, my estimates for this group are substantially lower. I also provide evidence that men, contrary to women, sort themselves into mental health support in the long-run, likely as consequences of stigma effects and deterioration in general health. Taken together, my results highlight the necessity to provide long-term preventive care and the need to de-stigmatize mental health support.

My work is closely related to the literature on parental bereavement and mortality ([Li et al., 2003](#); [Rostila et al., 2012](#); [Russ, Stamatakis, Hamer, Starr, Kivimäki and Batty, 2012](#); [Espinosa and Evans, 2013](#)). These studies find in general a negative impact of bereavement on the mortality risk but do not provide robust estimates of the dynamics of the effect. This is, as they use a proportional hazard model and fix the time intervals over which they hold the impact of stress constant. It is, however, a priori not clear how to choose these time intervals and why stress should shift the mortality risk in proportional ways within the chosen intervals. In addition, previous work on this topic is silent about possible alleviating effects of health care utilization, in particular mental health support.

The results presented in my analysis do not restrict the underlying process or the dynamics of how stress evolves over time. Thus, my estimates provide a clear picture of how stress is affecting mortality and allow to identify crucial points in time when health interventions might be most promising. In addition, my decomposition analysis enables me to investigate mental health support as an underlying possible channel which might protect from the negative consequences of stress. In this sense, my work also extends the findings of [van den Berg et al. \(2017\)](#). By linking the impact of mental health support back to my estimates, I show that it can have long-run benefits but also that stigma effect can lower the effectiveness of mental health support, especially for men.

Taken together, my results have important implications for public health specialists and policy makers and show that in times of economic uncertainty and increasing stress health reforms should consider the positive long-term effects of mental health support.



They also imply that even today stigma effects associated with mental health are a main obstacle in providing effective care.

## 2 Data

My analysis is based on three different data sets: the Austrian Social Security Database (ASSD), the Austrian Death Register, and the Register for Family Allowances. The ASSD is a matched firm-worker database and contains detailed information on demographic characteristics, daily labour market states, and yearly earnings. [Zweimüller, Winter-Ebmer, Lalive, Kuhn, Wuellrich, Ruf and Büchi \(2009\)](#) provide an extensive description of the ASSD. I use the information provided in this data set to obtain pre-treatment confounders.

The Register for Family Allowances contains information on child benefits, the parental benefit recipient, and the eligible child. Furthermore, in case a spouse is indicated during the application process, information on her/him is supplied. This enables me to link parents to children. The unique person identifier also allows me to link parents to their work history and demographic characteristics.

The Austrian Death Register contains the exact death date and detailed information on the underlying causes of death of an individual through the *International Classification of Diseases* (ICD9/ ICD10). It covers the time span from 1970 until 2009, and the exact cause of death is available for around 82% of all entries. Most of the missing entries occur at the beginning of the time period. By concentrating on recent deaths and applying the sample restriction rule discussed below, I obtain the exact cause for more than 95% of all the observations in my sample. The Death Register can be linked to the ASSD and the Register for Family Allowances using a unique person identifier available in all three data sets.

One drawback of my combined data is that although I can identify all main parents-children combinations, I have problems linking very young children to the ASSD and the Death Register. I concentrate therefore on parents with at least one child aged 10 to 24. While not being able to include younger children in my analysis is certainly a caveat, previous research has not found that parental bereavement effects differ substantially by the age of the deceased child ([Li et al., 2003](#); [van den Berg et al., 2017](#)).

### 2.1 Treatment and Control Group

For each quarter during my observation period between the beginning of 1993 and the second quarter of 2005, I first identify those parents aged 30-60 who lost a child during

the respective quarter and the child was between the age of 10 and 24 at the time of death. These parents constitute my treatment group.

From this sample, I then disregard 36 individuals who suffered from more than one child loss. Excluding parents suffering from multiple bereavements ensures that I capture the effect of an abrupt increase in stress on mortality. I also exclude 25 individuals who died during the same quarter as the child. In total, this gives me 3,269 parents who suffered from bereavement. Out of this sample, 1,500 are men (46%) and 1,769 are women (54%).

Parents who can anticipate the death of their child might adjust their behaviour well before the actual death date. As this adjustment is unobserved, including those individuals in my estimation leads to biased results. Following the discussion in [van den Berg et al. \(2017\)](#), from my treatment sample I only include individuals in my analysis who lost their child due to death by external causes (ICD-9:800-999; ICD-10: V01-Y98), but I exclude those where the death cause is due to self-harm and assault (ICD-9: 950-969; ICD-10: X60-X84, X85-Y09). Among death by external causes non-intentional accidents account for 73%, and suicides and homicides for 27% of my sample. Applying all my restrictions, I end up with 1,737 observations in total. 804 individuals are men (46%) and 934 individuals are women (54%).

Table 1 gives an overview over the five most common death causes as a share within different age groups in my sample. As one can see from the table, most of my cases result from fatal motor vehicle accidents. This cause is most important at the age 16 to 18. Around this time most children obtain their driving license and are inexperienced drivers. As a result, they are more prone to fatal traffic accidents. For younger children, death due to other traffic accidents, other accidents (falling objects, electric current) and accidents due to submersion, suffocating, and foreign bodies play also important roles. In general, most of the variation in the analysis stems from older children.

[Table 1]

To obtain the control group, I randomly draw for each quarter I observe a child's death (base quarter) 1,500 individuals with the same baseline characteristics as parents in my treatment group but who have never suffered from child bereavement. In total, I obtain 78,000 observations; 35,320 are men (47%) and 41,685 (53%) are women.

From the final data set, I discard all those persons from the analysis who have at least one disabled child in the base quarter or were younger than 16 at the birth of the first child. Having a disabled child in the family is itself a source of stress, and hence, the effect of interest might be contaminated. Parenthood before the age of 16 is very rare in my sample and is likely driven by unobserved individual traits. After all these

adjustments, my final sample comprises of 76,456 observations in the control group and 1,722 observations in the treatment group.

Table 2 provides summary statistics of my estimation sample. As one can see, bereaved parents tend to have lower educational attainment and are less likely to live in Linz, Graz, or Vienna, the three major cities of Austria. They also tend to have slightly more and older children on average and have also more boys. In terms of previous labour market characteristics, I find that bereaved parents have lower income and slightly more unemployment days. I do not find large differences in terms of long-term sickness absence from work or health related retirement.

[Table 2]

## 2.2 Outcome Variables

The data sets in hand provide information on individuals until the end of 2009, so I can follow each individual for at least 4 years. For every quarter during the follow-up period, I determine whether the individual died during the quarter. If the person died, I calculate the duration as the difference between the death and the base quarter. If an individual has not died until the end of 2009, I regard this observation as censored. This implies, for example, that some individuals are censored after 4 years while others are censored after 10 years.

I consider various death causes to obtain a clear picture of how stress affects mortality. Stress can affect the circulatory system but might also lead to a change in lifestyle and health behaviour. Those suffering from alleviated stress levels might increase smoking or drinking in order to cope with the stress. Thus, besides all-cause mortality, I investigate these specific causes. In particular, I study death due to circulatory diseases (ICD-9:390-459; ICD-10: I00-I99) and smoking- & alcohol related death causes. For smoking-related deaths, I use the same definition as Malarcher, Schulman, Epstein, Thun, Mowery, Pierce, Escobedo and Giovino (2000). For alcohol-related death causes, I use the definitions provided by the Center for Disease Control and Prevention ([http://nccd.cdc.gov/DPH\\_ARDI/Info/ICDCodes.aspx](http://nccd.cdc.gov/DPH_ARDI/Info/ICDCodes.aspx)). From these definitions, I include all direct and indirect chronic causes. For acute causes, I only use those which are 100% attributable to alcohol. When considering specific outcomes, I censor all persons at their death date as to whether the person died, and the reason of death does not belong to the cause studied.

To investigate the potential beneficial effect of mental health support, I use information provided by the *Gebietskrankenkasse* in Upper Austria. The *Gebietskrankenkasse* insures the majority of all employees in the private economy. For individuals, who held

insurance provided by the Upper Austrian *Gebietskrankenkasse*, I also observe the utilization of psychological or neurological treatments from the end of 1998 until 2009. I use this utilization later in my analysis to investigate how mental health support might lower the mortality risk associated with stress. For this sample, the number of individuals in the treatment group reduces to 258 (120 men and 138 women) and the number of individuals in the control group is 7,771 (3,574 men and 4,197 women). In addition, the maximum time I can follow an individual is 10 years.

### 3 Empirical Approach

In this Section, I describe my empirical approach in more detail. First, I discuss how I obtain my dynamic effects using a Weighted Kaplan Meier Estimator (WKM). Then, I show how one can adjust the WKM in order to analyse the indirect effect of mental health support on the mortality risk.

#### 3.1 Weighted Kaplan Meier Estimator

For at least two reasons, standard approaches, such as using the Cox model, are not appropriate when investigating the effect of stress on the mortality risk. First, there is no a priori knowledge about how stress should affect mortality. For example, in the context of the Cox model, it is not obvious that the mortality risk for bereaved parents shifts proportional to the motility risk of non-bereaved parents. Indeed, the results presented in Section 5 imply that the proportionality assumption is violated in my setting. Thus, imposing likely unjustified assumptions will lead to biased estimates. Second, there is no reason to assume that the effect of stress remains constant after the treatment or to impose arbitrary restrictions on how it evolves over time. Knowing the dynamics is, however, crucial for designing effective policies. In my work, I use a flexible approach which allows me to investigate the treatment effect dynamics without relying on arbitrary restrictions on the underlying process. It is based on a combination of propensity score weighting and nonparametric estimation of the mortality risk ([Anstrom and Tsiatis, 2001](#); [Xie and Liu, 2005](#); [Browning and Heinesen, 2012](#); [Sant’Anna, 2016](#)).

I am interested in estimating the Average Treatment Effect on the Treated at each time  $t$ ,  $\Delta(t)$ . Let  $Y_i$  be the observed time until death of an individual  $i$  and  $B$  the treatment indicator with  $B = 1$  if the parent has suffered from a child loss. Denote by  $\mathbb{1}\{A\}$  the indicator function which takes a value of 1 if the argument  $A$  is true. Then,  $\Delta(t)$  can be formally defined as following

$$\Delta(t) = E[\mathbb{1}\{Y_{\tau \leq t}(1)\} - \mathbb{1}\{Y_{\tau \leq t}(0)\} | T = 1] \quad (1)$$

In Equation (1),  $Y_{\tau \leq t}(1)$  is the potential outcome under treatment and  $Y_{\tau \leq t}(0)$  is the potential outcome under control at time  $t$ ; see, for example, [Imbens and Wooldridge \(2009\)](#) for an overview over treatment effects and the potential outcome notation. A challenge in my setting is that  $Y$ , is not observed for all individuals in my sample and thus subjected to censoring. What is observed in the data is a variable  $D = \min\{Y, C\}$ , where  $C$  is the censoring time, together with a censoring indicator  $\xi = \mathbf{1}\{Y \leq C\}$ . To obtain an unbiased estimate of Equation (1), I need to take this censoring problem into account while controlling for various background characteristics which can affect an individual's mortality risk.

I can estimate my counterfactual outcomes by applying a two-step approach. In a first step, I estimate the propensity score  $\pi(X) = P(B = 1|X)$  by means of a logistic regression using a rich set of covariates  $X$ . I use these estimates  $\hat{\pi}(X)$  to construct weights of the form  $\hat{w} = \frac{B}{\pi} + \frac{(1-B)}{1-\pi} \frac{\hat{\pi}(X)}{(1-\hat{\pi}(X))}$ , where  $\pi = P(B = 1)$ .

In a second step, I use the weights  $\hat{w}$  in a Kaplan-Meier estimation to obtain my counterfactual outcomes. Denote by  $D_i$  the time of death (or censoring) of an individual  $i$ . The censoring indicator  $\xi_i$  takes the value one if I observe an exit and zero if the individual is censored. The counterfactual numbers of incidents for group  $B=b$ , with  $b \in 0, 1$ , at time  $t_j$  is given by

$$\widehat{d_{w,t_j}^{B=b}} = \sum_{i:D_i=t_j} \hat{w}_i \xi_i Y_i(b)$$

Likewise, the counterfactual number at risk in this particular group at time  $t_j$  can be estimated by

$$\widehat{R_{w,t_j}^{B=b}} = \sum_{i:D_i \geq t_j} \hat{w}_i Y_i(b)$$

The weight-adjusted Kaplan-Meier Estimator can then be defined analogously to the standard Kaplan-Meier Estimator as

$$\widehat{S_t(B)} = \Pi_{t_j \leq t} \left[ 1 - \frac{\widehat{d_{w,t_j}^k}}{\widehat{R_{w,t_j}^b}} \right] \quad (2)$$

By weighting my Kaplan-Meister estimator with the estimated propensity score, I am able to account for differences in observable characteristics without imposing restrictive assumptions on the distribution or dynamics of my effects.

Once I have obtained the estimates for the treatment and control group, I can calculate my parameter of interest. I estimate the ATET as following

$$\widehat{\Delta}(t) = \hat{E}[\mathbb{1}\{Y_{\tau \leq t}(1)\} - \mathbb{1}\{Y_{\tau \leq t}(0)\} | T = 1] \cdot 100$$

$$= \left( \hat{S}_t(0) - \hat{S}_t(1) \right) \cdot 100 \quad (3)$$

where  $\hat{S}_t(1)$  and  $\hat{S}_t(0)$  are the weighted Kaplan-Meier estimates at time  $t$  for the treatment and control group respectively from Equation (2). Equation (3) expresses the treatment effect as percentage point (pp) differences in the mortality risk. I base inference on the bootstrap using 999 replications.

$\hat{\Delta}(t)$  gives me the causal effect of (bereavement) stress on the mortality risk if 1) unconfoundedness (or conditional independence), 2) overlap, and 3) random censoring holds. The unconfoundedness assumption  $Y(0) \perp\!\!\!\perp B|X$  requires that there are no unobservables which affect jointly the potential mortality risk and treatment once I control for all my covariates. The assumption would be violated, for example, if more careful parents might have a lower mortality risk themselves but also have more careful children. In my empirical specification, I include a wide range of characteristics to control for this potential confounding such as educational attainment, place of residence, and previous labour market attachment as well as concentrating on unanticipated death causes of the child which makes sorting less likely. Later in this paper, I also explore the robustness of my results to specific failures of my unconfoundedness assumption.

The overlap assumption  $P(B = 1|X) < 1$  can be assessed graphically and requires that I find suitable control observations for my treatment group. While I do not find strong evidence that this assumption is violated, the precision of my estimates are lower when considering observations in areas with thin overlap. I account for this by applying the trimming approach suggested in Heckman, Ichimura and Todd (1997) and Heckman, Ichimura, Smith and Todd (1998). This guarantees at least a minimum overlap between my treatment and control and greatly improves precision while leaving the estimated effects virtually unchanged. A detailed discussion on how I implement the trimming procedure as well as untrimmed results can be found in Appendix B.

The assumption of random censoring requires that there is no selective drop out of individuals from my data. It would be violated, for example, if individuals with higher mortality risk also had a higher attrition rate. As my analysis is based on administrative data which follows individuals over their entire lifetime, this assumption is not restrictive in my setting.

### 3.2 Decomposition of the Treatment Effect

The second goal of my paper is to see if health care provision, in particular for mental health, can lower the impact of stress on mortality. This indirect effect can be obtained in a similar way as my WKM with a slight adjustment to the weighting functions using results from mediation analysis (Imai, Keele and Yamamoto, 2010); see also Bijwaard and Jones (2019). To see how my decomposition works, rewrite the potential outcome

defined in the previous section  $Y(B)$  as a function of mental health care utilization  $M$  (mediator)  $Y(B) = Y(B, M(B))$ , for  $B = b$  and  $b \in \{0, 1\}$ . Then, I can decompose the treatment effect from Equation (1) as follows

$$\begin{aligned}
\Delta(t) &= E[\mathbb{1}\{Y_{\tau \leq t}(1)\} - \mathbb{1}\{Y_{\tau \leq t}(0)\} | T = 1] \\
&= E[\mathbb{1}\{Y_{\tau \leq t}(1, M(1))\} - \mathbb{1}\{Y_{\tau \leq t}(0, M(1))\} | T = 1] \\
&\quad + E[\mathbb{1}\{Y_{\tau \leq t}(1, M(1))\} - \mathbb{1}\{Y_{\tau \leq t}(0, M(0))\} | T = 1] \\
&= (S_t(0, M(1)) - S_t(1, M(1))) \\
&\quad + (S_t(0, M(0)) - S_t(0, M(1))) \\
&= \theta(t) + \delta(t)
\end{aligned} \tag{4}$$

In Equation (4),  $\delta(t)$  measures the change in the mortality risk due to the utilization of mental health care  $M$  by keeping the treatment status fixed and varying the mediator over different treatment states.  $\theta$  accounts for all other channels through which the treatment can affect mortality and which are not attributable to  $M$ . In the decomposition, I follow [Vansteelandt and VanderWeele \(2012\)](#) and use the potential mediator under treatment. This constitutes a natural reference level for treated subjects and corresponds to their actual choice.

I can estimate  $\theta$  and  $\delta$  in a similar fashion as my WKM with a slight change in the weighting function; see also [Huber \(2014\)](#). Notice that  $S(0, M(0))$  is identified using the weighting approach outlined in the previous section. To estimate  $S(0, M(1))$  via re-weighting I need in addition a mediator score  $\pi(X, M) = Pr(B = 1 | X, M)$ .  $\pi(X, M)$  is obtained via a logistic regression on the treatment indicator, but in addition to my covariates  $X$ , I also include  $M$  in the estimation. The predicted values are then used to construct weights of the form  $w^M = \frac{(1-B)}{\pi} \frac{\pi(X, M)}{(1-\pi(X, M))}$ . Using  $\hat{w}_i^M$  instead of  $\hat{w}_i$  allows me to estimate  $S(0, M(1))$  from the data (see also [Hong \(2012\)](#), and [Schmidpeter \(2018\)](#) for weighting approaches to recover mediation effects).

To make the mediating effect of mental health utilization comparable between mothers and fathers, I report the relative impact of the mediator on the overall mortality risk instead of an absolute effect as defined in Equation (4). More precisely, I will measure the indirect effect as the percentage reduction (or increase) in the overall mortality risk  $\delta^M(t) = \frac{\Delta(t)}{\theta(t)} - 1$ . Notice that  $\Delta(t)$  measures the overall mortality risk while  $\theta(t)$  measures the mortality risk holding  $M$  constant. Hence, the fraction of both parameters gives me the percentage effect of varying  $M$ .

To give  $\delta(t)$  a causal interpretation, I need to impose slightly stronger assumptions than before. Besides common support  $P(B = 1 | X, M) < 1$  and random censoring, I also need a sequential conditional independence assumption (SCIA) to hold. The SCIA requires not only that the treatment is (conditionally) independent of the potential out-



comes and the treatment but also that there is no selection into mental health care utilization with respect to the outcome, once I control for treatment status and my observed covariates. [Imai et al. \(2010\)](#) provides an extensive discussion. This assumption would fail, for example, if more health-conscious parents have lower mortality risk but are also more likely to ask for psychological help after bereavement. But even when the SCIA is violated does the decomposition provide interesting insights into how mental health support can change the mortality risk after a stressful even.

Before presenting my estimation results, two additional points warrant discussion. First, my decomposition approach allows for one mediator at a time only. For example, my approach does not allow to separately disentangle the impact of health, divorce, and changes in labour market status from the direct effect of losing a child, although all these factors are likely important contributors (e.g. [Sbarra, Law and Portley, 2011](#); [Fox, Cacciatore and Lacasse, 2014](#); [van den Berg et al., 2017](#)). The estimates of  $\delta$  capture only the effect of mental health support while all the other effects are included in the estimates of  $\theta(t)$ . Accounting for multiple mediators would require a clear causal ordering of the underlying mechanisms ([Daniel, Stavola, Cousens and Vansteelandt, 2015](#)). For example, in order to disentangle the separate impact of divorce and health on mortality, one would need to specify that either health is affecting the divorce risk or vice versa, but there is no obvious choice how to order the mediators here. As there is no justification which ordering is the right one, I will not use multiple mediators in my analysis.

Second, my approach cannot account for dynamic mediation effects and therefore is silent if the mechanism amplifies over time. Under the imposed assumption it is not possible to identify a dynamic effect, and additional stronger restrictions would be required. [Robins \(2003\)](#) and [Huber \(2014\)](#) provide an extensive discussion.

## 4 Balancing Properties

In this section, I briefly discuss the balancing properties of my propensity score. Detailed results can be found in [Appendix A](#).

I assess the balancing properties of my estimated propensity score using the standardized-difference-in means (SDM) ([Rosenbaum and Rubin, 1985](#)) and the t-test for differences in means. [Figure 1](#) shows box-plots for the SDM and the t-test before and after weighting the data separately for mothers and fathers. The vertical line in the middle of the box corresponds to the median. The lower and upper ends of the box represent the 25th and 75th percentile. The vertical lines (whiskers) extend to the 95th and 5th percentile respectively. The crosses represent extreme observations.

[[Figure 1](#)]



As one can see from the left panel in the figure, the unadjusted differences exhibit imbalances both when considering the SDM and the t-statistic as relevant measure. I find the largest difference in the unadjusted sample in the number of boys for both men and women with a SDM of above 50. After applying my estimated propensity score weights to the sample, the test statistics fall considerably. The estimated SDMs are now well below the threshold of 20 recommended in [Rosenbaum and Rubin \(1985\)](#). The statistics are also well below the maximum SDM of 10 as recommended in [Normand, Landrum, Guadagnoli, Ayanian, Ryan, Cleary and McNeil \(2001\)](#). I come to a similar conclusion when looking at the test statistic derived from the t-test.

## 5 Results

In this section, I first present the main estimation results for overall mortality and death due to cardiovascular diseases as well as alcohol & smoking related death causes. Then, I present heterogeneous effects for certain subgroups. In addition, I provide a sensitivity analysis for my results for certain failures of my identification assumptions. Before discussing my findings, it should be noted that I use the unexpected death of a child as a trigger for a sudden rise of stress levels. In the long term, the consequences are likely better summarized by psychological distress or grief. In [Section 6](#), I relate my results to other forms of stress.

### 5.1 Main Results

[Figure 2](#) presents the estimates of my treatment effect of  $\Delta(t)$  for all-cause mortality, death due to circulatory diseases, and alcohol & smoking related death causes. In each panel, the solid line presents the dynamic estimates of the mortality risk for women and the dashed line for men. Marker indicate the significance level of my estimate; a triangle indicates significance on a 10% level, a circle on a 5% level, and a square on a 1% level.

[[Figure 2](#)]

Panel a shows the estimated treatment effect for all-cause mortality. The estimates exhibit clear temporal variation for both genders. For women, the mortality risk remains relatively flat during the first four years after bereavement. The treatment effect increases afterwards and reaches a high of 1 pp at year 10. After this point, the effect converges back towards zeros. The results are largely imprecisely estimated. This shows that the gain in flexibility of applying the weighted Kaplan-Meier estimator comes at a loss of efficiency compared to specifying a parametric model.

My estimates for men strongly contrast the ones obtained for women. Although, I estimate a similar flat pattern during the first years there is a long run positive and significant impact of stress on the mortality risk for this group. One striking feature is that the mortality risk is steadily increasing over the second half of my follow-up period, with a sudden and sharp upturn seven years after the stressful event. At its high at year 13, bereaved men have a more than 6 pp higher mortality risk. These estimates are quite sizable and highly significant. But even after this point, my estimates do not show any sign of reversal but a long-term increase in the mortality risk associated with stress.

It is clear from my results that there are substantial dynamics in the effect of stress on mortality. These patterns would not have been captured in this detail when applying standard duration methods with arbitrary restrictions. [Espinosa and Evans \(2013\)](#) report the highest effect during the first two years after the treatment which is almost 3.5 times higher than the effect from year 3 onward. The estimates of [Rostila et al. \(2012\)](#) imply an inverted U-shaped pattern for mothers similar to the ones presented here, a lower effect during the first 3 years, increasing from year 4 to year 8, and afterwards decreasing again. For fathers, they find in general an increasing effect with time. In contrast, using similar time intervals but data for Sweden, [Li et al. \(2003\)](#) find that the time pattern for both parents follow a U-shaped pattern with a heightened risk 4 to 8 years after the stressful event. My results highlight the importance of applying a flexible approach when estimating temporal treatment effects. This is also stressed by [Sant'Anna \(2016\)](#) in settings with substantial individual heterogeneity like in my case.

Interestingly, my results also show that women are less affected by stress. This is especially surprising in my setting as one would expect that mothers are more attached to their children and hence suffer more from the loss. One explanation for this development might be that men react substantially different to stress, with the full effect coming into force years after the actual event. In contrast, women seek external help and try to cope with the loss from the very beginning (e.g. [Taylor, Klein, Lewis, Gruenewald, Gurung and Updegraff, 2000](#)). For example, [Buckley, Bartrop, McKinley, Ward, Bramwell, Roche, Mihailidou, Morel-Kopp, Spinaze, Hocking, Goldston, Tennant and Tofler \(2009\)](#) find that cortisol levels, the steroid hormone which helps the body respond to stress, were higher for men than women after the loss of a spouse or parent. They trace the higher levels back to an increased alcohol intake after the stressful event. Likewise, [Vance, Najman, Boyle, Embelton, Foster and Thearle \(1994\)](#) show that men exposed to sudden infant death syndrome had a higher frequency of heavy drinking close to the death date. Thus, gender differences in adverse health behaviour such as excessive smoking and drinking might explain my results.

I investigate this possibility by estimating the effect of stress on alcohol & smoking related mortality. The results are present in Panel b in Figure 2. The estimates mirror

closely the ones obtained when considering overall causes of death for both men and women, albeit they are slightly smaller in magnitude. As before, my estimates for men indicate a flat mortality risk during the first years and a strong and persistent increase afterwards. The estimated overall time pattern for men is consistent with strong negative behavioural adjustments as response to increased stress levels with the negative effects coming into force several years later.

Lastly, I also look at the effect of death due to circulatory diseases. Stress might not only lead to an increased mortality risk because of adverse behavioural changes but might also affect the circulatory system. The results are presented in Panel c of Figure 2. I do not find any significant effects on the long-term mortality risk neither for women nor for men when considering this outcome. While I do find that there is an increase by the end of the observation period for men, the estimates are not statistically significant and substantially lower compared to my previous results. Taken together the results presented here point towards adverse changes in health behaviour as one of the main driving forces behind the impact of stress on mortality for men. The conclusion is not that clear for women who might look for external support as a response with possible mitigating effects. I investigate mental health support as underlying channel in more detail in Section 7.

## 5.2 Heterogeneous Effects

In this section, I consider possible heterogeneous effects of stress on mortality. One important factor how individuals cope with stress is the availability of social interactions. As pointed out in [Taylor et al. \(2000\)](#), especially men are prone to tend to their spouse in stressful situations. I do not have information on the cohabiting status in my data, but I know if the application for child allowance was made single or jointly with a partner. I use this information and estimate the effects for individuals who applied with a partner. The gender of the child is another important factor how parental stress levels can be affected if they have certain preferences for boys or girls. Same sex parent-child pairs can exhibit stronger bonds and this might lead to a different impact of bereavement (e.g. [Feldman, 2003](#)).

Figure 3 presents the results for the couple and same sex parent-child pair samples. The solid (dashed) line presents the estimation results for mothers (fathers) when I restrict the sample to parents who filled in a joint application for child benefits. The dotted (dashed-dotted) line presents the results for mothers (fathers) where the deceased child was a girl (boy).

[Figure 3]

Investigating if social relationships can have a protective effect, I estimate a lower mortality risk compared to the overall sample. Similar protective effects of marriage have also been found by [van den Berg and Gupta \(2015\)](#). For mothers, my effects are virtually zero over the whole follow up period. For fathers, my effects are now smaller during the first years compared to my baseline results, but I still estimate a long-lasting and significant increase in the mortality risk at the middle of my observation period. The increase is, however, less pronounced when compared to my baseline sample. While social interactions seem to lower somewhat the mortality risk for this group it cannot completely offset the negative consequences of stress.

Looking at my estimates if the deceased child was the same sex as the parent, one can see a substantially higher mortality risk for both mothers and fathers when compared to my baseline results. This increase is particularly clear for mothers, albeit the effects are insignificantly estimated. If the deceased child was a girl, mothers have a 3 pp higher mortality risk 8 years after the bereavement compared to a 1 pp increase estimated for my baseline sample. Likewise, the treatment effects for my father-boy sample exhibit the same shape and the same strong increase as in my baseline sample, but my long-run estimates are now up to 25% higher. These findings stand in contrast to those of [Werthmann, Smits and Li \(2010\)](#) but are in line with [van den Berg et al. \(2017\)](#) who find that future labour market outcomes of parents who have the same sex as the deceased child are stronger affected.

The results in this section show that both social interactions and the gender of the child can have an important influence on the heightened mortality risk. One remaining concern is that my results are driven by unobserved characteristics and hence that my unconfoundedness assumption is not satisfied. In the next section, I investigate the robustness of my results to certain departures of my identifying assumptions.

### 5.3 Robustness Check

Despite considering only unexpected causes of death and controlling for a wide range of background characteristics part of my estimated effects might still be driven by unobserved selection into treatment. In this section, I investigate the robustness of my results when there are specific departure from my unconfoundedness assumption following [Ichino, Mealli and Nannicini \(2008\)](#). The idea behind their approach is that the unconfoundedness assumption only holds if an additional unobserved confounder  $U$  is taken into account:  $Y(0) \perp\!\!\!\perp (X, U)$ . [Ichino et al. \(2008\)](#) then propose to simulate the unobserved confounders  $U$  based on the distribution of an observed binary covariate and use the simulated values of  $U$  as an additional control in the estimation of the propensity score;

see also [Rosenbaum \(1987\)](#). Importantly, this approach does not require to specify any parametric model for the construction of  $U$  and is therefore more robust. If my results were sensitive to the inclusion of  $U$  this would point towards violation of my identifying assumptions.

As can be seen from the summary statistics presented in Table 2 and the results of the propensity score estimation reported in A.1 in Appendix A the covariates with the largest difference between treatment and control group are the number of boys, the average age of the children, and if the family lived in one of the major three cities of Austria. Basing my simulation on these three variables should therefore best capture any possible selection. For each estimation based on one of these three variables, I simulate  $U$  1,000 times to estimate the treatment effect.

The results are presented in Figure 4. As one can see, both the magnitude and the time pattern of the treatment effects when including  $U$  in the propensity score estimation are very similar to my results obtained under unconfoundedness. Basing the selection on my binary rural indicator gives me even a slightly higher effect of stress on the mortality risk for men. Overall, the sensitivity analysis shows that my estimates are robust to the inclusion of simulated confounders and give confidence in my identification strategy.

[Figure 4]

## 6 Implications for Other Forms of Stress

My analysis is based on a sudden and unexpected loss of a child, a very particular form of stress. In this light, the implications of my results for other forms of stress are worth discussing. I do so by comparing existing medical studies which measure the changes in cortisol levels due to bereavement to studies relating the change to economic shocks using the relative scale of the studies to weight my estimates ([Persson and Rossin-Slater, 2018](#)). Cortisol is the primary stress hormone in the body which increases sugar in the bloodstream and curb non-essential body functions in stressful situations. Under regular circumstances, hormone levels return to normal once the situation has passed. If stress is persistent, however, these levels remain increased for a longer time period. As different studies use different units to measure cortisol levels, I use the metric  $nmol/l$  and the conversion factor  $\mu/l = 27.59nmol/l$ .

*Bereavement and Cortisol Levels:* There are various medical studies relating bereavement stress to changes in blood (plasma) or saliva cortisol levels. The findings differ between the type of cortisol levels (blood vs. saliva) taken and the type of bereavement investigated. The results in [Pfeffer, Altemus, Heo and Ji \(2009\)](#) show that adults who

lost a spouse during the 9/11 terrorist attacks have around 8.28 nmol/l higher morning saliva cortisol levels after 4 months compared to non-bereaved individuals. Using the death of a spouse or parent and concentrating on the median, [Buckley et al. \(2009\)](#) report a difference in morning blood cortisol levels of 60 nmol/l two weeks after bereavement and of around 66 nmol/l 6 months later between bereaved and non-bereaved individuals. [Irwin, Daniels, Risch, Bloom and Weiner \(1988\)](#) compare women who lost their spouse to non-bereaved ones. They find that blood cortisol levels are 99.3 nmol/l higher in the bereaved group. To the best of my knowledge, the only research which studies the change in cortisol level due to sudden parental bereavement is [Spratt and Denney \(1991\)](#). Surprisingly, they find no statistical difference in the blood cortisol level between bereaved and non-bereaved parents over their study time of 8 months and only briefly, after 6 months, find the average cortisol level of bereaved parents slightly over the ones of the control group, with a difference of 3.59 nmol/l.

*Other Forms of Stress and Cortisol Levels:* There are a number of studies relating economic stress to changes in cortisol levels. [Puterman, Haritatos, Adler, Sidney, Schwartz and Epel \(2013\)](#) compare the saliva cortisol levels of young adults who reported financial hardship to those who did not report any financial problems. The level for individuals who reported to suffer under financial strains was on average 5 nmol/l higher in the morning. [Maier, Egger, Barth, Winker, Osterode, Kundi, Wolf and Ruediger \(2006\)](#) study the changes of blood cortisol level with respect to the duration of unemployment. They compare individuals who were unemployed less than 1 month to those unemployed between one and 18 years. They find that cortisol levels are 50.4 nmol/l higher for the 12-13 months group, 37.52 nmol/l higher for the 14-20 months group, 59.87 nmol/l higher for the 22-36 months group, and 62.35 nmol/l higher for individuals with an unemployment spell of at least 37 months. [Arnetz, Brenner, Levi, Hjelm, Petterson, Wasserman, Petrini, Eneroth, Kallner and Kvetansky \(1991\)](#) find that individuals who lost their job due to a mass-layoff have an increased blood cortisol level of 68 nmol/l one year after displacement compared to those in secured employment situations.

*Relating Bereavement to Other Forms of Stress:* To obtain comparable results, I relate only studies which use the same type of cortisol. I do not consider the results of [Spratt and Denney \(1991\)](#) in my comparison as they suggest that parental bereavement has no effect on stress levels. Under the assumption that individuals suffer at least as much from child bereavement than from spousal one the following comparisons is likely a lower bound on the true effect of other forms of stress on the mortality risk. The findings of [Pfeffer et al. \(2009\)](#) and [Puterman et al. \(2013\)](#) imply that the impact on cortisol levels from financial problems is around 60% ( $= 5/8.28$ ) of the impact of spousal bereavement. Relating this to my estimates for the long run mortality risk (10 to 16 years) implies that financial stress increases the mortality risk between 0.15 and 0.70 pp for women and between 1.9 and 3 pp for men, which is still substantial. The studies of [Arnetz et al.](#)

(1991) and Maier et al. (2006) lead to a similar conversion factor between 60% and 70% of my estimates and therefore I come to similar results when considering changes in the mortality risk due to job insecurity and long-term unemployment.

## 7 Stress and Mental Health Support

My results indicate that stress increases the mortality risk especially for men. The estimates for alcohol & smoking related death causes imply that one of the main reasons for this increase is the change in health behaviour to deal with stress. Interestingly, I do not find similar results for women. In this section, I investigate if mental health support can alleviate the negative impact of stress and account for these gender differences using the decomposition method described in Section 3.

To minimize the selection into mental health care, I measure the mediator within the first quarter after the treatment. I also take into account that the mediator precedes the outcome and consider only deaths after my mediator is measured. This means that I disregard all individuals who died (or were censored) within the first quarter from my estimation for the direct and indirect effects, but I include them in the first step estimation of the propensity scores. In addition, I also provide estimates when extending the time until I measure my mediator to half a year after the base quarter. This gives insights into the importance of selection into mental health support.

Table 3 reports the mediating effect of mental health care utilization. Columns (1) and (2) show the estimated effects when the mediator is measured within the first 3 months after the base quarter for mothers and fathers respectively. Columns (3) and (4) show the estimated effects when the mediator is measured 6 months after the base quarter. To make the effect comparable, I report the indirect effect as the percentage change in the overall mortality risk  $\delta(t) = \frac{\Delta(t)}{\theta(t)} - 1$ .

[Table 3]

Looking at the mediating effects of mental health support for mothers reported in Columns (1) and for fathers in Columns (2), two interesting features emerge. First, mental health support for women is successful in lowering the mortality risk due to stress. I estimate that mental health care treatments taken up during the first 3 months after the base quarter lowers substantially the mortality risk for women between 3% and 10% over my sample period. The estimates for men are substantially lower in magnitude.

Second, gender specific utilization of mental health support likely explains why I do not find the same sharp increase in the mortality risk for women as for men. Looking at the time-varying effect, I estimate the highest protective impact for women at the most



crucial time around six years after the base quarter. At this date, mental health support lowers overall mortality by around 10%. The effect for men is with 4% less than half of the size. These results also support the hypothesis that men rather restore to adverse health behaviours as a mechanism to cope with stressful events than to look for external help .

Interestingly, my estimates for women remain fairly stable when expanding the time over which I measure the mediator and therefore to allow for a longer selection period. This can be seen from the results presented in Column (3) of the table. Women look quite shortly after a stressful event for external help with a relatively low incident of long-run selection into mental health support. In contrast, as can be seen from Column (4), the estimates for men are now larger in magnitude and almost double the size. This suggests that there is a long run sorting of men into health care utilization, likely conditional on stigma effects and having worsening stress symptoms.

The results in this section show that mental health support can be successful in counteracting adverse effects of severe stress. They also help to understand why I do not find stronger effects for women in my analysis. While women benefit from mental health support from the very beginning, the results for men suggest that there is long-term sorting. This might be as there are still stigmata associated with mental health conditions. Taken together with the results from my previous section, this implies that men restore first to adverse health behaviour as coping mechanism, with fatal consequences.

## 8 Conclusion

Stress is ubiquitous in everyday life but its dynamic effect on the mortality risk is not well understood. In this research, I provide new evidence on the dynamic adverse health effects of stress. My method allows me to obtain temporal effects without relying on distributional assumption or restricting treatment effect heterogeneity which is crucial in my setting. To identify variations in stress levels, I use an unexpected death of a child as the triggering event.

I find that (bereavement) stress increases the mortality risk for both men and women. My estimated effects exhibit substantial dynamics over time. For both men and women, I estimate a relatively flat risk during the first years but an increase later. This increase is rather moderate and peaks around ten years after bereavement for women. In contrast, for men my estimates show a strong and persistent increase in the mortality risk. I provide evidence that this is largely due to adverse health behaviour such as excessively smoking and drinking. Conducting a sensitivity analysis, I show that my results are robust to deviations from my identifying assumptions.



Exploring a possible channel behind these gender differences, I find that mental health support provided shortly after the stressful event has a strong and long-term protective effect for women. Although I find that mental health support also decreases the mortality risk for men, the protective effects are considerably smaller. This is likely due to long-term sorting into mental health support as a result of stigma effects and worsening health conditions. Overall, the results suggest that men look for external support too late.

My work gives two important implications for policy makers. First, it highlights the need to provide long term assistance after periods of high levels of stress. The provision of health care is largely focused on the treatment of symptoms rather than prevention, often in order to reduce costs. My results show that this approach can be short-sighted. The negative consequences of stress can build up over a long-time period and suddenly affect health and mortality. The promotion and provision of mental health support early after the stressful event can mitigate these risks. These long run beneficial consequences should be considered when designing public health policies.

Second, the results also highlight the necessity to expand the availability of mental health care and to de-stigmatize the use of it. Especially men seem to under-use mental support and rather resort to adverse health behaviour to deal with high levels of stress. In this light, it is necessary that health and governmental agencies centralize and increase their effort on anti-stigmata programs.

## 9 Tables

Table 1: The Five Main Causes of Child's Death

	Motor Vehicle - Accidents	Other Traffic - Accidents	Falls	Other Accidents	Submersion, Suffocating	N
<b>Total</b>	73.90%	3.27%	4.96%	3.93%	1.78%	1069
<b>Age 10-16</b>	69.49%	6.78%	1.69%	5.08%	3.39%	59
<b>Age 16-18</b>	78.16%	3.40%	3.40%	2.43%	1.94%	206
<b>Age 18-20</b>	75.37%	2.64%	4.69%	4.40%	1.17%	341
<b>Age 20-22</b>	72.41%	3.10%	4.83%	4.83%	3.10%	290
<b>Age 22-24</b>	69.94%	3.47%	8.67%	2.89%	0.00%	173

The Table presents summary statistics for the child's cause of death for parents in the treatment group. The total number of parents in the treatment group is 1,722; 927 are women and 795 are men. The following grouping was used (according to ICD-9): Motor Vehicle Traffic Accidents: E800-E819; Other Traffic Accidents: E820-E849; Falls: E880-E888; Other Accidents: E916-E928; Submersion, Suffocating and Foreign Bodies: E910-E915

Table 2: Summary Statistics by Treatment Status

	Mothers			Fathers		
	Treatment Bereaved $T = 1$	Control Not Bereaved $T = 0$	Difference	Treatment Bereaved $T = 1$	Control Not Bereaved $T = 0$	Difference
<b>Personal Characteristics</b>						
Age (Years)	45.41 (5.38)	44.78 (6.26)	0.63 (6.24)	48.20 (5.21)	47.43 (6.30)	0.77 (6.28)
No Partner (%)	26.54 (44.18)	25.46 (43.57)	1.07 (43.58)	19.50 (39.64)	18.10 (38.50)	1.40 (38.53)
Comp. Schooling (%)	56.96 (49.54)	51.78 (49.97)	5.17 (49.96)	45.03 (49.78)	43.90 (49.63)	1.13 (49.63)
High School (%)	38.08 (48.58)	38.50 (48.66)	-0.42 (48.66)	46.67 (49.92)	41.88 (49.34)	4.79 (49.35)
Higher Degree (%)	4.96 (21.73)	9.72 (29.62)	-4.75 (29.48)	8.30 (27.61)	14.22 (34.92)	-5.91 (34.79)
Living in Major City (%)	15.86 (36.55)	23.78 (42.57)	-7.92 (42.47)	11.95 (32.46)	21.14 (40.83)	-9.19 (40.69)
<b>Children Characteristics</b>						
No. of Children	2.01 (0.88)	1.71 (0.76)	0.30 (0.76)	2.06 (0.87)	1.75 (0.75)	0.31 (0.76)
No. of Boys	1.30 (0.80)	0.90 (0.76)	0.40 (0.76)	1.34 (0.80)	0.92 (0.76)	0.42 (0.77)
Average Age	19.94 (2.62)	18.57 (4.03)	1.37 (4.01)	19.96 (2.49)	18.52 (4.02)	1.43 (3.99)
<b>Previous Labor Market Outcomes</b>						
Av. Income (in '000)	13.79 (13.19)	15.21 (15.29)	-1.42 (15.25)	25.09 (21.00)	26.95 (22.48)	-1.86 (22.45)
Av. Unemployment (Days)	23.22 (61.73)	18.01 (53.14)	5.20 (53.35)	22.91 (60.18)	16.47 (52.08)	6.45 (52.28)
Av. Sick Leaves (Days)	2.78 (11.91)	2.43 (13.12)	0.35 (13.10)	4.67 (17.58)	3.06 (15.07)	1.61 (15.13)
Not in Labor force $t - 3$ (%)	14.99 (35.72)	17.00 (37.56)	-2.00 (37.52)	9.43 (29.25)	8.20 (27.44)	1.23 (27.48)
Not in Labor force $t - 2$ (%)	20.06 (40.07)	21.53 (41.11)	-1.47 (41.08)	10.31 (30.43)	9.26 (28.99)	1.05 (29.02)
Not in Labor force $t - 1$ (%)	21.04 (40.78)	21.18 (40.86)	-0.14 (40.85)	11.70 (32.16)	10.60 (30.78)	1.10 (30.82)
Blue Collar Worker	25.78 (43.77)	22.10 (41.50)	3.68 (41.55)	41.01 (49.22)	32.79 (46.95)	8.22 (47.01)
Regular Retirement (%)	4.21 (20.09)	3.90 (19.37)	0.30 (19.39)	8.55 (27.99)	6.59 (24.81)	1.97 (24.88)
Health Retirement (%)	2.16 (14.54)	1.82 (13.38)	0.33 (13.41)	4.65 (21.08)	3.42 (18.17)	1.23 (18.24)
<b>Outcome</b>						
Dead (%)	2.48 (15.56)	1.97 (13.90)	0.51 (13.94)	7.42 (26.23)	4.98 (21.75)	2.44 (21.86)
No. of Observations	927	41,136		795	35,320	

The treatment group consists of all parents who lost a child aged 10-24 due to unanticipated causes, see Section 2 for details. The control group consists of all parents who had at least one child in the same age range but did not suffer from bereavement. No Partner Specified refers to all parents who applied for child allowances without specifying a partner. All individuals residing in Graz, Linz or Vienna are regarded as living in a major city. Parental average income, unemployment, and sick leaves were measured using the 3 years preceding the reference date. Standard deviations are reported in parentheses.

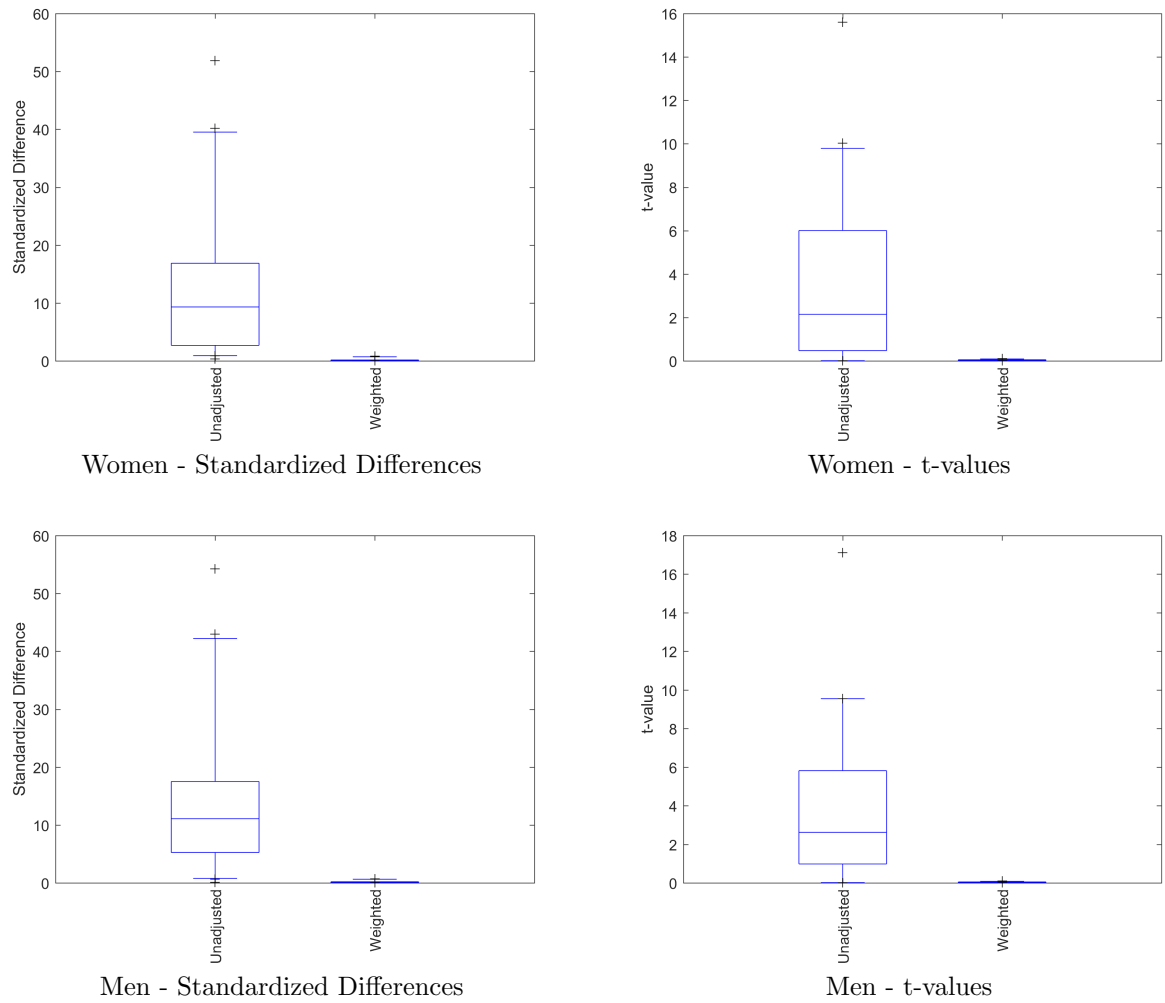
Table 3: The Mediating Effect of Mental Health Support

Duration	(1)	(2)	(3)	(4)
	Short-Term Mediator		Medium-Term Mediator	
	$\delta^S$		$\delta^M$	
	Mothers	Fathers	Mothers	Fathers
1	-6.04	-0.72	-6.34	-1.67
2	-5.40	-1.62	-6.30	-7.12
3	-2.79	-1.12	-2.59	-2.58
4	-2.65	-2.68	-2.53	-7.01
5	-3.17	-4.65	-3.05	-9.90
6	-10.27	-4.02	-10.34	-7.87
7	-10.27	-2.71	-10.34	-4.43
8	-7.53	-4.26	-8.88	-6.41
9	-8.41	-4.77	-9.95	-4.48
10	-8.41	-4.80	-9.95	-4.41

The table presents estimates of the mediating effect of mental health support on the mortality risk  $\delta$ .  $\delta$  is calculated as the %-reduction (increase) in the total treatment effect due to mental health support; see the description in Section 3. Mental health support is measured by the number of treatments provided in the area of Neurology, Psychiatry, and Psychotherapy. Short-Term Mediator refers to the situation where the mediator is measured during the first quarter after the reference date and Medium-Term Mediator refers to the situation where the mediator is measured during the first two quarters after the reference date. In all these cases, individuals who died (or were censored) before the end of the period during which the mediator is measured do not contribute to the estimates of  $\delta$ . The sample consists of all individuals who held insurance provided by the *Gebietskrankenkasse* in Upper Austria from the year 1998 to 2005. The number of individuals in the treatment group is 258 (120 men and 138 women) and the number of individuals in the control group is 7,771 (3,574 men and 4,197 women).

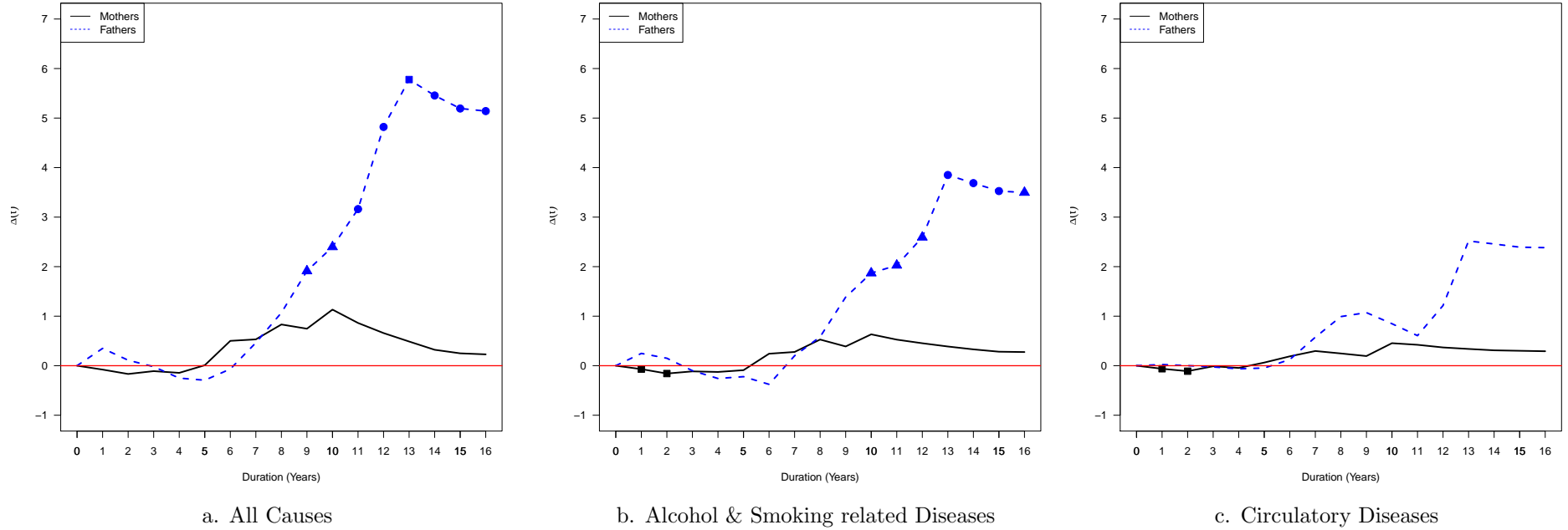
## 10 Figures

Figure 1: Balancing Property of the Propensity Score



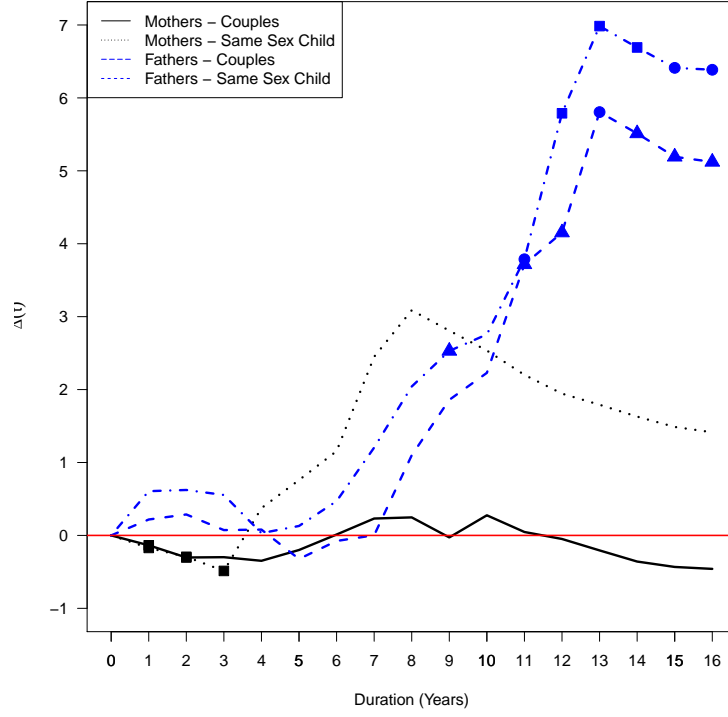
The figures show box-plots for the absolute Standardized Differences in Means ([Rosenbaum and Rubin, 1985](#)) and t-values for difference in means. Within each graph, the first box plot shows the unadjusted differences and the second box plot the propensity score weighted differences. The vertical line in the middle of the box corresponds to the median. The lower and upper ends of the box represent the 25th and 75th percentile. The vertical lines (whiskers) extend to the 95th and 5th percentile respectively. The crosses represent extreme observations.

Figure 2: Effect of Stress on the Mortality Risk



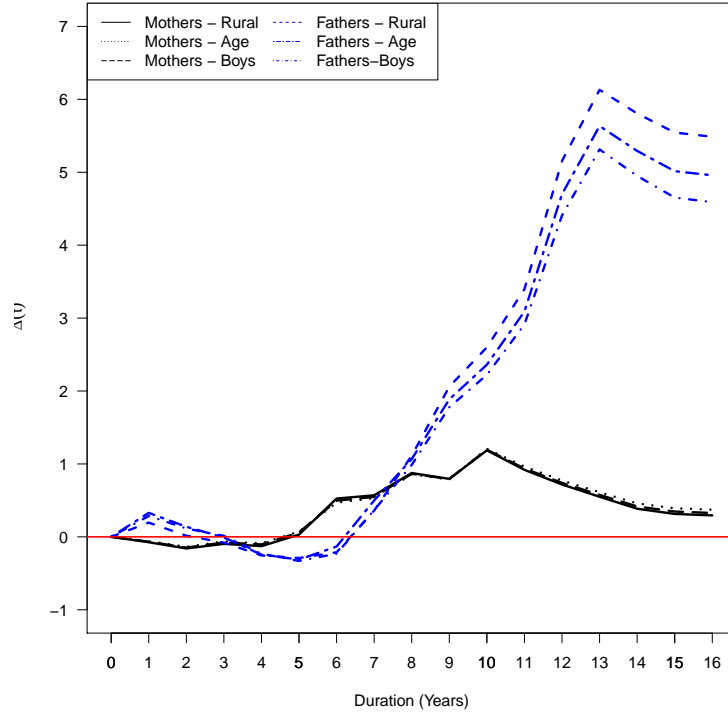
The figure shows the estimated difference in the mortality risk between the treatment and control group  $\Delta(t)$  which was estimated using the approach described in Section 3. The solid line shows the treatment effect for mothers and the dashed line the treatment effect for fathers. Deaths due to Circulatory Diseases are defined as death causes according to ICD-9: 390-459 and ICD-10: I00-I99. Death causes due to Smoking-related Diseases are taken from Malarcher et al. (2000) and death causes due to Alcohol-related Diseases are all direct and indirect chronic as well as all direct acute causes as defined by the Center for Disease Control and Prevention ([https://nccd.cdc.gov/DPH\\_ARDI/Info/ICDCodes.aspx](https://nccd.cdc.gov/DPH_ARDI/Info/ICDCodes.aspx)). When estimating the effect of the treatment on Circulatory Diseases and Alcohol & Smoking related Diseases, individuals who died of other causes are censored at the death date. Marker indicate the significance level of the estimates: a triangle indicates significance on a 10% level, a circle on a 5% level, and a square on a 1% level. Inference is based on 999 bootstrap replications.

Figure 3: Effect of Stress on the Mortality Risk - Heterogenous Effects



The figure shows the estimated difference in the mortality risk between the treatment and control group  $\Delta(t)$  for specific subgroups which was estimated using the approach described in Section 3. The solid line (dashed) line shows the treatment effect for mothers (fathers) who specified a partner at the child benefit application process. The dotted line (dashed-dotted) line shows the treatment effect for mothers (fathers) where the deceased child was a girl (boy). Marker indicate the significance level of the estimates: a triangle indicates significance on a 10% level, a circle on a 5% level, and a square on a 1% level. Inference is based on 999 bootstrap replications.

Figure 4: Effect of Stress on the Mortality Risk - Sensitivity Analysis



The figure shows the estimated difference in the mortality risk between the treatment and control group  $\Delta(t)$  when the conditional independence assumption only holds if taking an additional confounder  $U$  into account:  $Y(0) \perp\!\!\!\perp T|(X, U)$ ; see the described in Section 5.  $U$  is simulated following the approach suggested by Ichino et al. (2008) and is based on a binary indicator for living in a rural area, having children with age above the mean, and the number of boys in the family is above average. The solid (dashed) line shows the results when basing  $U$  on the rural indicator for mothers (fathers), the dotted (dash-dotted) line the results when basing  $U$  on the age indicator, and the long-dashed (short-dashed) line when basing  $U$  on the boy indicator. Results were obtained simulating each category of  $U$  1,000 times.



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