DEPARTMENT OF ECONOMICS
JOHANNES KEPLER UNIVERSITY OF LINZ

The Fatal Consequences of Grief

by

Bernhard SCHMIDPETER

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Johannes Kepler University of Linz
Department of Economics
Altenberger Strasse 69
A-4040 Linz - Auhof, Austria
www.econ.jku.at

bernhard.schmidpeter@jku.at
phone: +43 732 2468 5372

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Bernhard Schmidpeter

Johannes Kepler University Linz

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Abstract

In this paper we investigate the effect of stress on the survival probability using a child's death as the triggering event. Employing a propensity score weighted Kaplan-Meier estimator, we are able to explore the associated time pattern of grief without imposing assumptions on the underlying duration process. We find a non-monotonic relationship between time and relative survival rates: decreasing for 13 years after the event and slowly reversing afterward. However, even 19 years after the event bereaved parents have significantly lower survival probabilities compared to the hypothetical case, that the event had not occurred. Investigating the main reason for this development, our results indicate that bereaved parents have a higher probability of dying from natural causes, especially circulatory diseases. Interestingly, our results reveal that bereavement has a stronger impact on fathers, while we find only modest evidence for mothers. This is a novel and surprising finding as males are in general regarded as more stress resilient than females. However, this research shows that this perception is not true.

Keywords: Bereavement, Child death, Death, Adjusted Kaplan Meier

JEL Classification Numbers: I12, J14, C41

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Contact details: bernhard.schmidpeter@jku.at, Johannes Kepler University Linz, Department of Economics: Altenberger Str. 69 - A-4040 Linz, Austria. This research was funded by the Austrian Science Fund (FWF): National Research Network S103, The Austrian Center for Labor Economics and the Analysis of the Welfare State and the Christian Doppler Laboratory "Aging, Health and the Labor Market".

1 Introduction

It is well documented that the death of a loved one has adverse effects on the surviving persons. Bereaved individuals have an increased risk of deteriorating physical as well as psychological health and excess mortality (see for example Byrne & Raphael (1997), Chen et al. (1999), Gerra et al. (2003), Espinosa & Evans (2008), van den Berg et al. (2011) and Simeonova (2013)). However, most of the research which studies the consequences of bereavement concentrate on the effect on spouses, while comparatively little attention has been given to parents who lost their child.[1] Yet, the death of a child can be regarded as possibly the most horrendous and detrimental life event parents can suffer from.

Given that most parents share a unique bond with their children, a child loss is often associated with a higher level of stress and severe adjustment problems compared to other forms of bereavement (Rubin (2003) and Sanders (2003)). Psychological stress of a child loss accompanied by possible behavioral changes such as drinking or smoking can lead to ill health such as an increased the risk of myocardial infarction (Li et al. (2002)) and diabetes (Olsen et al. (2005)). Moreover, parents might find it difficult to adapt to their loss even over a longer time horizon. As a consequence bereavement can have a negative effect on mental health, leading to depression, anxiety or other forms of affective disorders (Lehmann et al. (1987), Li et al. (2005), Rogers et al. (2008) and van den Berg et al. (2012)). These health effects might moreover affect the long term labor market supply and earning prospect of bereaved parents. Furthermore, increasing time absence from the labor market might create a vicious cycle, where bereaved parents find it more and more difficult to re-enter employment (van den Berg et al. (2012)). In the most extreme, child bereavement leads to heightened mortality of parents (Lehmann et al. (1987), Li et al. (2003), Rostila et al. (2012) and Espinosa & Evans (2013)).

Despite the evidence that high levels of bereavement stress lead to a higher mortality risk, the exact time pattern and pathways seem not to be clear. For example, Espinosa & Evans (2013) find an increase in overall mortality for mothers only during the first two years after the bereavement, while Li et al. (2003) find a significant effect after the first to the third year and again after nine years for mothers but no significant effects for fathers.

[1] See the literature review of Stroebe et al. (2007).

Rostila et al. (2012) do not find significant time effects either for mothers or for fathers who have experienced the death of a minor child but an increase in the mortality risk for mothers when all time periods are pooled together.

In this study we investigate the time pattern of stress on mortality in greater detail using a child's death as an especially distressing event for parents. Our analysis contributes to the existing literature in three important ways: First, we apply a semiparametric estimation method, the adjusted Kaplan Meier Estimator proposed by Xie & Liu (2005) and disussed in Sant'Anna (2014), to estimate the effect of child death on parents' mortality. This approach allows us to combine propensity score weighting with nonparametric estimation of the survival function, which removes all bias associated with observable covariates while it does not impose any restriction on the underlying duration process. This has the advantage that our estimation procedure is more robust to possible misspecification. For example, in our analysis we find a violation of the proportional hazard assumption in the majority of our cases. Hence relying too much on functional form restrictions would lead to wrong conclusions in our setting. Furthermore, with our approach we are able to estimate the bereavement effect for every point in time during the follow up period. While this is theoretically possible using standard duration models, in practice and for computational reasons researcher have to specify time periods over which to hold the treatment effect constant, as for example in Li et al. (2003), Rostila et al. (2012) and Espinosa & Evans (2013). However, the decision over which time span the treatment effect remains constant is somewhat arbitrary and can mask important patterns. The approach used here leads to a clearer understanding how stress is affecting the time pattern of mortality.

Second, our data contain a rich set of demographic, family and work characteristics, which enable us to account for a wide range of possible confounding factors, such as past unemployment spells, family income and sick leaves from work. Socioeconomic background is not only affecting one's own health but is also a strong predictor for health and mortality of children (see for example Blakey et al. (2003) and Currie (2009)). For example, ill health of the parents might transfer to children leading to heightened mortality in both parents and children. Moreover, some forms of death, for example due to cancer or long-term illness, might be anticipated by the parents well before the actual death date. This anticipation effect can lead to unobserved changes in behavior. On one hand, it might

be possible that parents with terminally ill children start to smoke or drink, which would lead to an overestimation of our effects. On the other side, parents could prepare for the death of the child and starting to cope with the situation, leading to less exposure to stress at the actual death date. In this case, our estimates would be lower than the actual effect. To avoid any of these biases we construct an exogenous treatment indicator using the exact information about the death cause of the child and excluding possibly expected death causes as cancer and long-term illness. Previous studies were only partially able to control for socioeconomic characteristics or exact death cause of the child, leading to doubts that bereavement stress is the cause of heightened mortality in parents (see also the comment of van Ommeren & Levav (2003)).

Third, we investigate the effect of stress on more specific diagnoses of parents' death causes. Besides considering overall mortality as well as death due to natural and unnatural reasons, we investigate the effect of bereavement on parents' death by cancer and circulatory diseases. These two death causes are often associated with stressful events. Especially when considering circulatory diseases an exact picture of the time pattern is very useful to understand how stress is affecting mortality. It is likely that the burden of a child death on the circulatory system only comes to effect after a certain time after the loss. Aggregating the time period in the wrong way might therefore conceal important effects. Investigating stress on more specific death causes complements furthermore the existing literature of the loss of a child on parents' health (for example Li et al. (2002), Li et al. (2005) and Bolton et al. (2013)).

In our analysis, we find strong evidence, that an unexpected death of a child by unintentional causes, such as traffic accidents, is associated with a decrease in the overall survival probability for affected parents. However, the associated time pattern is not stable over time. Our results indicate that the survival probability is steadily decreasing until 13 years after the event and shows slight signs of reversal afterward. Nevertheless, even 19 years after the child's death we find that bereaved parents have a significant lower relative survival probability. Our results show that affected parents are at higher risk of dying from natural causes, especially from circulatory diseases, but do not find a significant effect on external causes. We find that in the long run fathers have a higher risk of premature death than mothers. This is a novel and surprising finding as males are in general regarded as

more stress resilient than females. However, his research shows that this perception is not true.

Investigate possible channels through which a child loss might affect the health of the parents, we find that bereaved parents take up more sick leaves shortly after the death and have a higher probability of retiring from work due to health impairments. However, we do not find evidence that bereaved parents suffer from wage loss or have more unemployment days after the child's death. These results support our main findings and indicate that stress is directly affecting parent's health and increases the risk of mortality.

The paper proceeds as follows: In Section (2) we give an overview of the empirical method used in this paper. Section (3) describes the data and the identification of control and treatment groups, while section (4) contains the results from our propensity score estimation. The main part of the paper, section (5) , presents our estimations of the average treatment effect and discusses the results. The robustness of our results are examined in section (6). We investigate possible channels trough which bereavement might affect mortality in section (7). Conclusions are drawn in section (8).

2 Empirical Method

Our goal is to to investigate the time pattern of bereavement on parents in a flexible manner without imposing restrictions on the underlying duration process. For this purpose, we apply the weight-adjusted Kaplan Meier Estimator (WKM) proposed by Xie & Liu (2005), however with some slight variation of the weighting function as we will discuss further below.[2] The construction of the WKM proceeds in two steps. In a first step, we estimate the probability that an individual is observed in the treatment group given our covariates $E[T = 1|X]$. This quantity is called the propensity score and will be denoted by $p(X)$. In a second step, we use the estimated propensity score to construct weights for the Kaplan Meier Estimator.

This approach has several advantages over the usual estimators employed in duration analysis. First of all, by combining reweighting with nonparametric duration analysis we can account for all bias associated with observable covariates, while we do not impose

[2]See also the discussion in Sant'Anna (2014) for a similar approach.

any restrictions on the underlying duration process. This makes our estimation approach robust to functional form misspecifications. Indeed in the majority of our estimations, we reject the proportionality assumption imposed by the popular Cox proportional hazard model. Second, our approach allows us to calculate a treatment effect for every point in time during the follow up period. While this is theoretically possible in standard duration models, in practice and due to computational issues the treatment effect is assumed to be constant over certain time periods. The periods over which the effect is specified to be constant is somewhat arbitrary and can furthermore mask important details of the duration process. The approach used in this study does not rely on functional form assumptions nor do treatment effects have to be hold constant over certain time periods. Therfore it gives a deeper understanding of the impact of stress on the survival probability. In the following we explain our estimation procedure in more detail.

Let the dummy variable T denote the treatment status, with $T = 1$ if the individual has suffered from a child loss, i.e. she belongs to the treatment group, and zero otherwise. Similarly, let $Y(1)$ and $Y(0)$ be the potential outcomes under the treatment and control respectively. We are interested in comparing the outcome of a bereaved parent to the hypothetical outcome the child death had not occurred, i.e. we want to estimate the average treatment effect on the treated (ATET), which is of central interest for policy makers.[3] However, as our outcome variable is subjected to censoring, everything we can observe for an individual i from the data is the minimum between the death date and a censoring time $C(T_i)$, together with a censoring indicator Δ_i.

If there was no censoring we could identify $E[Y(1)|T = 1]$ from the data alone, while $E[Y(0)|T = 1]$ could be identified under unconfoundedness $Y(0) \perp\!\!\!\perp T|X$ and an overlap assumption $P(T = 1|X) < 1$ (Rosenbaum & Rubin (1983)). Here X is a vector of exogenous covariates and $\perp\!\!\!\perp$ means statistical independence. However, ignoring censoring or estimating the treatment effects only for the uncensored sub-population would lead to inconsistent results. Hence, besides unconfoundedness and overlap we furthermore assume that censoring is independent of our treatment, outcome and explanatory variables as in Honoré et al. (2002) and Lee & Lee (2005).

[3]To be more precise, the effect measured in this paper is a distributional treatment effect. However, we stick to the more familiar term of average treatment effect on the treated in the following.

Rosenbaum & Rubin (1983) show that if unconfoundedness given the vector X and the overlap assumption holds, so does it for the propensity score. Hence, to obtain an estimate of the hypothetical outcome $E[Y(0)|T = 1]$, we can re-weight our data using the estimated propensity score. This adjustment removes any bias associated with the observable covariates (Rosenbaum & Rubin (1983)). We estimate the propensity score by means of a logistic regression using a set of baseline covariates, which includes family characteristics, labor market outcomes and a set of demographic variables.[4] The predicted probabilities from our model $p(X)$ are then used to construct weights of the form $w_i = T_i + (1 - T_i)\frac{p(X_i)}{1-p(X_i)}$.[5]. With this in hand we can estimate the counterfactual quantity of interest.

Denote by D_i the time of death or censoring of an individual i. The censoring indicator Δ_i takes the value one if we observe an exit and zero if the individual is censored. We can use our estimated weights w to construct the weighted number of events in group k at time t_j as:

$$d^k_{w,t_j} = \sum_{i:D_i=t_j} w_i \Delta_i Y_i(k) \tag{1}$$

Likewise, the weighted number at risk in this particular group at time t_j can be defined as

$$R^k_{w,t_j} = \sum_{i:D_i \geq t_j} w_i Y_i(k) \tag{2}$$

The weight-adjusted Kaplan Meier Estimator for t and group k can be defined analogously to the standard Kaplan Meier Estimator as

$$\widehat{S^k(t)} = \begin{cases} 1 & \text{if } t < t_0 \\ \Pi_{t_j \leq t} \left[1 - \frac{d^k_{w,t_j}}{R^k_{w,t_j}} \right] & \text{if } t \geq t_0 \end{cases} \tag{3}$$

From (3) it is clear that our estimator is only defined if $R_{w,t_j} > 0$ and we have for all observations in the control group $p(X_i) < 1$.

[4] A detailed list of the variables used in the analysis can be found in section (4).

[5] For a proof, why re-weighting the data by w gives us our hypothetical distribution $f_{Y(0)|T=1}$ see for example Kline (2011), p. 533.

Once we have obtained our WKM estimates for the treatment and the control group, we can calculate our parameter of interest, the ATET. Let $S^T(t)$ and $S^C(t)$ be the Kaplan Meier estimates at time t for our control and treatment group respectively. We estimate the ATET then for every time t as

$$ATET(t) = ln(\frac{S^T(t)}{S^C(t)}) * 100 \tag{4}$$

Equation (4) expresses the treatment effect as relative change in the survival probability at time t [6] Besides enabling us to capture the time pattern of the treatment effect, defining the ATET in this way has the advantage that it is more convenient to interpret than a simple difference. For example, a negative ATET of 1 implies that bereaved parents have a 1% lower survival probability compared to the hypothetical event, the child loss had not occurred.

Xie & Liu (2005) treat the propensity score as known to derive the asymptotic variance of the weighted Kaplan Meier Estimator. However, this is a very strong assumption and unlikely to hold in practice. Furthermore, when estimating the ATET, the first step estimation affects the large sample distribution of the estimator and ignoring it might lead to an underestimation of the standard errors.(see Hahn (1998) and Hirano et al. (2003)). To account for this effect when calculating standard errors and confidence intervals we bootstrap the whole estimation process. For each bootstrap sample, we estimate the propensity score and calculate the weights, the Kaplan Meier estimator and the ATET anew. We then obtain the standard errors in the usual way.

3 Data

We conduct our analysis using three unique data sets: the Austrian Social Security Data Base (ASSD), the Austrian Death Register and the Register for Family Allowances. The ASSD is a matched firm-workers data base and contains very detailed information about demographic characteristics, daily labor market states, sick leaves and yearly earnings from 1972 until 2010. Zweimüller et al. (2009) provide a more detailed description of this data

[6] Equation (4) would not be defined if $S^C(t)$ or $S^T(t)$ were zero for some t. While this is a theoretical possibility, it is of less concern in our empirical analysis as the number of censored observations are sufficiently large at the end of our observation period for both the control and treatment group.

base. We use the ASSD to construct pre-treatment labor market outcomes to account for possible differences between parents in the treatment and the control group.

The Austrian Death Register contains the exact death date of an individual covering the time span from 1970 until 2010. Moreover, it includes detailed information about the underlying causes of death through the *International Classification of Diseases* (ICD9/ ICD10). The Death Register can be linked to the ASSD using a unique person identifier. A drawback is, that only around 80% of all deaths in Austria can be uniquely identified from the Death Register. Hence, in some circumstances we have to take the information from the ASSD to identify the death date of an individual. However, in those cases we are not able to identify the exact death cause.

The Register for Family Allowances contains information about child benefits, the person who is granted the benefits and the child for whom the benefits are paid. Furthermore, in case a spouse exists, information for her/him is supplied. This enables us to link parents with children. We also have information, if a family is paid additional child-disability benefits. They are granted if the degree of the disability is at least 50%. The unique person identifier, available in all data sets, allows us to link parents to their work history and both parents and children to demographic characteristics and the death register.

3.1 Definition of Treatment and Control Group

For each quarter between the beginning of 1991 and the second quarter of 2005, we identify those parents aged 20-60 as treated, when they lost their child during the respective quarter and the child was below the age of 22 at the time of death. In total, this gives us 3,057 treated individuals. However, parents, who can anticipate the death of their child, might adjust their behavior. As this adjustment is unobserved including those individuals in our estimation would lead to inconsistent results. Hence, following the discussion in van den Berg et al. (2012), from our original data we only include individuals in the treatment group who lost their child due to death by unanticipated or external causes (ICD-9:800-949,970-999; ICD-10: V01-X59, Y10-Y98).

Table (1) gives an overview over the broader categories of the death cause and their share considered in our study. The vast majority of all deaths are due to accidents,

especially traffic accidents, followed by accidental poisoning and falls and drops. Note that we exclude fatalities due to any cause of assault and suicides. The reason why we exclude suicides, is similar to the argument brought forward above. Parents might foresee or anticipate suicide intentions of their child, which might lead to a change in their behavior. Furthermore, death by assault might be a consequence of child abuse. However, as we are unable to identify if the offense happened within the family or outside, we prefer to obtain a strictly exogenous treatment indicator and exclude those cases.

Once we have obtained our treatment group, we randomly draw for each quarter in which we observe a child death (base quarter) individuals for our control group. In particular, for each base quarter we randomly choose around 2,500 individuals aged 20-60 who have at least one child below 22 years in the respective quarter and have not lost any child during their life. We exclude parents from our analysis who suffered from a child loss and the child was aged 22 or above at the time of death. This gives us in total 134,892 observations in the control group. From our final data set, we discard all those persons from our analysis which have at least one disabled child in the base quarter. Having a disabled child in the family might lead to unobserved adjustment in the behavior of the parents. After all these adjustments we end up with 130,518 observations in the control group and 1,649 individuals in our treatment group.

3.2 Outcome Variables

We are able to follow parents until the end of 2010, so for every member in both the control and treatment group we have a minimum of at least five years to follow. For every quarter during the follow up period, we determine if the individual died during the quarter or not. If the person died, we calculate the duration as the difference between the death and the base quarter. If an individual has not died until the end of 2010, we regard this observation as censored. This implies that some individuals are censored after five years, while others are being censored after 15 years, depending on their base quarter.

We consider various death outcomes in our analysis. First, we study all cause mortality and mortality due to natural and unnatural reasons (ICD-9:800-999; ICD-10: S00-T98, V01-Y84). Furthermore, we investigate specific causes of death often associated with stress. In particular we study death caused by cancer (ICD-9:149-239; ICD-10:C00-C97)

and circulatory diseases (ICD-9:390-459; ICD-10: I00-I99). When considering specific outcomes, we are censoring all persons at their death date if the person died and the reason of death does not belong to the cause studied or we do not have exact information about the death cause of a person.

Table (2) shows summary statistics for our sample. As we will discuss the propensity score estimation and observable differences between treatment and control group later, we do not show the distribution of the covariates in the table. In total our treatment group consists of 1,649 individuals while our control group comprises of 130,518 observations. From this simple descriptive statistic one can already deduce, that in general bereaved parents tend to have a higher mortality rate compared to those which have not experienced a child loss. Especially fathers seem to be affected by a sudden death of the child.

4 Propensity Score

The propensity score is estimated via a logit function for the whole sample and for fathers and mothers separately. In our estimation we account for a wide range of personal and socioeconomic characteristics which possibly can explain the difference between treatment and control group. More specifically, we control for age of the parents, family income in the past two years previous to the base quarter, unemployment duration and length of sick leaves up to four years before the base quarter, the employment sector in the base quarter, education, family size as well as residence and time dummies. Furthermore, we include dummies if the individual is single, widowed, foreigner, blue collar worker and/or female. We convert continuous explanatory variables into a number of categorical variables. This provides us with some functional flexibility when estimating the propensity score, but it is entailed with a certain loss of information. However, we try to minimize this loss by incorporating a very large set of categories for each continuous variable. In total we include 189 categorical variables in our estimation.

For expositional reasons we do not report estimation details. However, not surprisingly, those persons living in a metropolitan region are more likely to have lost a child due to external causes. Furthermore, working in the agricultural sector increases the probability of being in the treatment group while being employed in the health or service sector

reduces it. Our results indicate that, holding all other covariates constant, older individuals, singles and blue collar workers have a higher probability of suffering from a child loss. In contrast higher education is associated with a lower probability of being in the treatment group. Furthermore, individuals with inferior health, measured by sick-leave days from work, tend to be more likely to suffer from bereavement.

After estimating the propensity score, it remains to verify that there is sufficient overlap between the treatment and control group and our weighted sample is balanced. This is key for an unbiased estimation of our treatment effects.

4.1 Distribution of the Propensity Score

In general the estimation bias of the ATET can be split up into three terms: a bias term due to a lack of overlap in the distributions between control and treatment group, a term arising from misweighting the data and a component due to selection bias (Heckman, Ichimura, Smith & Todd (1998)). As argued before, we do not think that the unconfoundedness assumption is violated in our setting and hence we expect our selection bias to be negligible. Moreover, similar to matching on the propensity score, our weighting approach should eliminate all bias due to misweighting. We will assess potential bias arising from weighting the data when we discuss the balancing property of the propensity score. However, having sufficient overlap is crucial to estimate the ATET accurately, especially when using a weighting approach (Busso et al. (2013)).

We assess the overlap in our propensity score estimation graphically. To do so, we first estimate the density of the estimated propensity score for each group separately using kernel density estimation. As recommended by Imbens (2004), we choose a bandwidth smaller than the optimal one to undersmooth our estimation. We use in this analysis a smoothing bandwidth of $h_{smooth} = h_{opt}\ n^{-\frac{1}{10}}$, where h_{opt} is the optimal bandwidth determined by Silverman's rule (Silverman (1998)).[7] The distribution of the estimated propensity score by treatment status and sub-sample is given in figure (1). Inspecting the figure, it is obvious that bereaved parents have a substantial higher probability of being observed in the treatment group than non affected ones for each of our samples

[7] We were experimenting with various smoothing parameters but the results were virtually identical to those reported in the paper.

under consideration. For all our groups the predicted probabilities are smaller than one. This is especially important for the control group, as otherwise our estimator would not be defined.[8] Furthermore, the majority of the distributions between the treatment and control group are overlapping. However, there might be some common support problem at the upper part. Here the estimated density is non-zero for our treatment group but there are no comparable individuals in the control group.

To get a better assessment of the possible lack in overlap, we have to determine the region of common support. Following Heckman, Ichimura & Todd (1998) and Smith & Todd (2005) we define the set of all individuals within the common support as those for which

$$S_{trimmed} = \Big\{ p(x) : \hat{f}(p(x)|T=1) \cap \hat{f}(p(x)|T=0) > \epsilon \Big\} \tag{5}$$

holds. Here $\hat{f}(\cdot)$ is an estimate of the density and ϵ is a positive constant. The advantage of this definition is, that we can account for insufficient overlap over the whole range of the estimated propensity score. Hence, this approach is more robust than just disregarding observations above or below a certain threshold at the extremes of the distributions (see e.g. Dehejia & Wahba (1999)). In our analysis, we use a kernel density estimator with the bandwidth defined by h_{smooth} and an ϵ of .05.[9]

Table (3) shows summary statistics of our estimated propensity score for the whole and the trimmed sample. The figures confirm the conclusion from the graphical analysis. At the upper part of the propensity score distribution there are no comparable controls for the treatment group. However, once we apply our trimming, the support of the treatment group is almost identical to that of the control group. One can see that the number of observations which are outside of the common support region is very small; around .66% in the treatment and .09% in the control group. The same is true if we look at the distribution of the propensity score separately by gender: for both mothers and fathers the overlap is around 99% in both the treatment and control group. Furthermore, from the table it is clear that no person in the control group is assigned a large weight and hence dominates our analysis. For example, the maximum weight in the control group is .21 in the full

[8] As we are interested in the ATET we can relax the usual assumption $0 < p(x) < 1$ to $p(x) < 1$.

[9] For robustness check, we conducted the analysis with an ϵ of .1 and .2. However, the obtained results, are similar to those reported.

sample and reduces to .11 in the trimmed one.

Although a lack in overlap seems not to be of any concern given the high number of individuals within the common support, we will investigate the sensitivity of our results in section (6) by re-estimating the ATET for the common support only. The trimmed ATET is in general only a valid measure of the treatment effect for the subgroup within the common support and does not allow for a generalization to the whole population. However, if we find that the ATET for the trimmed and the complete sample does not substantially differ, we can safely conclude that a lack of overlap is of no concern in our analysis.

We furthermore investigate the sensitivity of our results with respect to the method applied. Frölich (2004) has argued that re-weighting estimators are not competitive to matching. However, as pointed out in Busso et al. (2013) the performance of weighting estimators depends on good overlap of the propensity score. As an additional robustness check, we compare the results of our weighted estimator to those obtained from matching.

4.2 Balancing Property

If the treatment indicator can be regarded as exogenous after weighting is in general not directly testable. One way to indirectly evaluate the unconfoundedness assumption is to test the effect of the treatment indicator on a set of outcomes, which are known to be unaffected by the treatment. For example, one could determine the effect of the treatment on outcome variables determined prior to the treatment. In some settings, it is possible to account for time-fixed unobservable characteristics which might affect selection into treatment using the estimators proposed in Heckman & Hotz (1989) and then use the testing procedures as described in their paper.[10] To apply either of these approaches, we have to observe our variable of interest prior and after the event, which is impossible in our setting. However, what we can test is the underlying assumption that our propensity score is a balancing score which eliminates all bias associated with our observable covariates. That is we can test that $X \perp\!\!\!\perp T|p(x)$. This condition is necessary for unconfoundedness given the propensity score. Hence, if we find significant differences

[10] However, not rejecting the null hypothesis in those tests does not necessarily imply that the unconfoundedness assumption holds (see Imbens (2004) for a discussion on theses tests.)

in the covariates between treatment and control group, the unconfoundedness assumption is most likely to be violated. Moreover, as our covariates are transformed into dichotomous variables the balancing test gives also an assessment of the bias due to misweighting.[11]

We test the balancing property of the propensity score by comparing the standardized difference or distance (SDM) of each covariate before and after weighting. The standardized difference is given by the absolute difference of the mean of the covariate between the treatment and control group, standardized by the pooled sample variance (Rosenbaum & Rubin (1985), Flury & Riedwyl (1986))

$$SDM_x = \frac{|\overline{X}_{treat} - \overline{X}_{control}|}{\sqrt{\sigma^2_{Pooled}}}$$

The SDM is commonly used in the literature to assess the balancing properties of the propensity score (see for example Lechner (2000), Hansen (2004), Sianesi (2004) and Levinsohn et al. (2013)) and has the advantage that it does not depend on the sample size, as for example the t-test. However, a potential drawback is, that there is no unique cut off value under which the sample is regarded balanced and hence its determination is somewhat arbitrary. For example, Rosenbaum & Rubin (1985) regard a SDM above 20% as large, while Normand et al. (2001) see already the danger of potential bias in the analysis with a SDM above 10%. In this analysis, we follow the more conservative cut off and regard a SDM of above 10% as a potential problem. For completeness we also report t-values for the difference in means.

Table (4) reports the SDM and t-values for difference in means of our covariates before and after weighting by treatment status for the whole population. In the appendix we furthermore report detailed results for mothers and fathers. We can see from the table, that before weighting there was a large imbalance between bereaved and non affected parents especially in age and relation status, mirroring our findings from the propensity score estimation. However, applying our threshold of 10%, we furthermore find an imbalance in sick leaves and unemployment durations. In particular, the SDM implies that bereaved

[11] The bias due to misweighting is zero if $f(X|T=0) = f(X|T=1)$ (see Heckman, Ichimura, Smith & Todd (1998)). To get a feeling for a possible bias due to misweighting, we can test if $E[X|T=0] = E[X|T=1]$ over the common support, as our covariates are dichotomous. Hence, if we observe larger difference between both groups this will indicate a greater bias due to misweighting. Note, although the results in table (4) are obtained using the untrimmed sample, the results for the region of common support are virtually identical.

parents tend to be longer sick and have longer unemployment duration. After weighting our sample, there is no evidence of a systematic difference between our treatment and control group. Our estimated SDMs are all below 4%.[12]. We arrive to a similar conclusion if we test for balancing separately by gender (see tables (A.1) and (A.2) in the appendix). Hence, we can conclude that our estimation sample is balanced. Moreover, as the estimated differences between treatment and control groups are close to zero, we do not see bias due to misweighting as a particular concern in our analysis.

5 The Consequences of Bereavement

In this section we present our estimation results. We consider as the maximum time span for calculating the ATET the last point in the follow up period where we observe an event in either group. As our data admits a follow up period of 19 years and we conduct our analysis on a quarterly the estimated ATET is depicted graphically together with 95% confidence intervals. All confidence intervals are based on 500 bootstrap replications.

To obtain a comprehensive picture of the fatal consequences of stress, we estimated the bereavement effect on various death outcomes. In particular, we examine the effect on a coarser set of death causes comprising of overall mortality, natural and external causes but also on a finer subset of specific causes, i.e. cancer and circulatory diseases. For better readability we split this section up into three subsections: the first one is concerned with overall mortality while the second one reports results for the two large groups death by natural causes and external causes. The third subsection contains our estimation for two specific causes often associated with stress: death by cancer and circulatory diseases.

Bereavement Effect on Overall Mortality

Figure (2) depicts our estimations result for overall mortality. Panel (a) shows the result for the overall sample. Our estimated treatment effect is negative for almost the complete follow up period of 19 years and the associated time pattern is far from being constant over time. Over the first two years, we see a sharp decline in the relative survival probability

[12] As with the propensity score estimation, we do not show the results for the residence, sector and time dummies for expositional reasons, but none of our estimated standardized difference for these variables was larger than .08%.

which remains fairly stable over the next four years. It then drops sharply from year six onward and the ATET reaches a minimum at year 13. The survival probability for bereaved parents is 1.72% lower compared to the hypothetical event, that the child loss had not occurred. This effect is quite substantial.[13] From this low, the effect slightly reverses afterward but the survival probability remains substantially lower than during the first twelve years after the bereavement. Our gain in flexibility comes however at a loss of efficiency compared to specifying a parametric model. The confidence bands indicate that the ATET is significant only from year eight after the bereavement onward. Nevertheless, we can conclude that a child loss is associated with a long and dramatic fall in the survival probability which remains substantially lower even 19 years after the bereavement.

Panel (b) and (c) show our estimations for fathers and mothers respectively. Comparing panel (b) with panel (c), we can see that mothers suffer from a child loss stronger during the first six years after the bereavement than fathers do, but the effect remains smaller in the long run. Our estimated ATET for mothers is close to significance only for some periods of the follow up time.[14] Bereaved fathers, on the contrary, exhibit a strong and sudden fall in survival probability after seven years and our estimated ATET reaches a minimum at -2.74% in year 13. As was it the case for the overall sample, after this point our ATET increases slightly. For both fathers and mothers we find that bereavement has a negative effect on the survival probability.

In contrast to the previous literature on bereavement effects, our estimates imply a stronger effect for fathers than for mothers. These results are somewhat surprising, as we 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 a child loss acts as a long term stressor for fathers with the full effect coming into force years after the actual event, while mothers seeking external help and try to cope with the loss from the very beginning.[15] If this was the case, we would expect to find strong effects of death due to natural causes, especially circulatory causes, for fathers and only modest or non-existing effects for mothers. We will investigate this pattern further in the next subsections.

[13] This effect translates to a cumulative hazard ratio of approximately 1.65.

[14] The effect is, however, significant at a 10%-level from year 4 to year 10.

[15] There is evidence that women deal differently with stress than men. Females tend to cope with stressful situations by turning to friends or family when stressed and rely less on the social support of the spouse (Taylor et al. (2000)).

Bereavement Effect on External and Natural Causes

To deal with the loss of a child, parents might, on one hand, do self-harm or commit suicide. If this was the case, we would expect to see a decrease in the relative survival probability for external causes. On the other hand, parents might be affected by long term stress of the death leading to an increased mortality due to circulatory diseases or heart attacks. If this was true, we would observe a fall in the relative survival probability for natural causes. We investigate this hypothesis in this section, considering two broad measures of death outcomes. Death due to external causes includes for example suicide, self-inflicted harm or traffic accidents, while death due to natural causes includes heart attacks, circulatory diseases or cancer. If an individual died during the follow up from any other cause than the one under consideration, the observation will be considered as censored at the death quarter.

Figure (3) shows our results for external causes of death. Inspecting panel (a) and (c), we see that there is no evidence that bereavement has an effect on excess mortality for external causes in the overall and mothers-only sample. The estimates effects are all close to zero and not statistically significant. The results for fathers, reported in panel (b), indicate even a slightly positive effect on the survival probability during the first years after the bereavement. This might be the case as father could reduce their labor supply and hence the probability of being involved in a fatal traffic accident reduces.[16]. We conclude that bereavement is not associated with an increase in the mortality due to external causes for mothers while it even has a slightly protective effect for fathers during the first years.

The picture changes if we consider only natural causes as the outcome of interest, depicted in Figure (4). The results for the whole sample, given in panel (a), show a sharp decline in the relative survival probability during the first three years of the follow up period, stabilizes in the medium run and steeply falls from year six until year 13. Our estimates become statistically significant after 7.5 years and remain so until the end of the observation period. Comparing this pattern to the one obtained when only considering fathers (panel (b)), one can see that the whole effect in the medium to lung run is mainly

[16] We investigate the effect of bereavement on labor supply in section (7). We tried to investigate the effect for more specific causes like accidents, suicides and alcohol-related mortality. However we did not find any effect.

driven by them. Although the estimated ATET for fathers is indistinguishable from zero during the first 8.5 years after the bereavement, it becomes highly significant afterward and stays so until the end of our follow up period. At its low in year 13, fathers have a 1.96% lower survival probability and even after 19 years the effect still amounts to -1.7%. In contrast, the results in panel (c) show that mothers have steeper fall in the relative survival probability over the first few years, but it is stabilizing on a higher level afterward. The estimates from year 4 to year 10 are significant on a 10% level. This is in line with our findings for death by all cause mortality. In general, fathers seem to suffer more from a child loss than mothers, especially in the long run.

Our results in this section indicate that there is no evidence that parents tend to extreme actions or self-harm after a child loss. Our estimated ATET for external causes is statistically zero for mothers, while there is a short-run protective effect for fathers. The outcomes for death by natural causes suggest that a child death is acting as a medium- to long term stressor in particular for fathers. For this reason, we have a closer look at two outcomes often associated with stress, cancer and circulatory diseases, in the next section.

Bereavement Effect on Cancer and Circulatory Diseases

Stress might lead cells to develop into tumours (Wu et al. (2010)) or to accelerates the spread of cancer (Wolford et al. (2013)). In light of these findings, we examine the effect of bereavement on cancer. The estimated ATET is depicted in figure (5). Although our estimates are mostly negative when considering the full sample or fathers only, they are not close to be statistically significant. The time pattern for fathers is similar to the one previously estimated, with a decline in later quarters. We can see that the same is true for mothers from half a year after the bereavement onward. Conclusive, we do not find evidence that stress increases the death probability due to cancer neither in general nor for bereaved mothers and fathers.

Bereavement can affect the circulatory system in multiple ways, directly through exposure to unhealthy high levels of chronic stress or indirectly by risk factors associated with a stress induced life style change, e.g. smoking, drinking or lack of exercising. The results of our analysis are depicted in figures (6). In general, we find that bereaved parents have a smaller relative surviving probability with the ATET being negative over the whole

observations period and statistically significant from eight years after the bereavement onwards. Panel (b) shows that stress due to bereavement affects the circulatory system of fathers strongly in the medium to long run. We can observe a pronounced drop in the survival probability around 6 years after the event. The effect for mothers, presented in panel (c), is virtually non-existing. Our estimated treatment effect stays close to zero for the whole observation period.

The findings in this section confirm our conjecture, that stress affects adversely the circulatory system in the long run but only for fathers. Furthermore, it seems to be the main channel through which bereavement affects mortality. Our estimates for fathers are substantial and stand in contrast to previous findings in the literature. The channel for mothers is not that clear. While we find that in general the survival probability decreases, especially for natural death causes, we do not find any significant effect when considering more specific causes of death.

6 Robustness Checks

6.1 Assessing Possible Lack of Overlap

In this section we examine the effect of a possible lack in the overlap of the propensity score on our estimated ATET. If our results were driven by individuals outside of the common support, the estimated ATET would be biased and our results do not hold in general (Heckman et al. (1997), Black & Smith (2004) and Rothe (2015)). Hence, to obtain valid inference we would have to restrict ourselves to individuals in the common support and estimate the ATET consistently only for this subgroup.[17] If, however, we do not find any different results for the trimmed and the full sample, we can conclude that limited overlap is not of a particular concern in our analysis.

We define the common support as in equation (5) and choose an ϵ of .05, where the density is estimated as described in section (4).[18] This leads us to disregard individuals on the right tail of the propensity score distribution, and hence observations with

[17] See also the discussion in Lechner (2008) and possibilities to incorporate information outside the common support.

[18] Another possibility is the method proposed by Crump et al. (2009), which calculates an optimal cut off value but does not take into account possible overlap problems in the middle of the distributions. We applied this method as well. The results are, however, similar to those reported in this section.

larger weights in our control group. However, in the total sample we lose only around .66% and .09% of our individuals in the treatment and control group respectively, a fairly small amount. Likewise, in our sample for fathers the trimmed sample in both groups is around 99% of the original one.[19] To obtain confidence intervals, we bootstrap the whole estimation procedure including the trimming process 500 times.

Restricting our estimation to the common support does not alter our conclusions from the previous sections. In general, our estimates for the trimmed sample indicate a slightly lower survival probability for bereaved parents and lower variability compared to the original sample, see the figures (B.1)-(B.3) in the appendix.[20] However, our trimmed estimates exhibit the same shape and time pattern as in the untrimmed analysis. However, all of our conclusions from the previous sections remain valid. Furthermore, the time pattern of the bereavement effect remains the same as when considering the untrimmed samples. We conclude that the bias due to a lack in overlap is negligible in our analysis.

6.2 Results obtained from Matching

Using simulation studies Frölich (2004) finds that weighting estimators perform worse with respect to finite sample properties than those based on matching. Reassessing his conclusion, that weighting is not competitive with matching, Busso et al. (2013) find that the performance of reweighting estimators are comparable if the overlap in the propensity score is good. Although, as shown in the previous section, our results seem not to suffer from a lack of overlap, we re-estimate the bereavement effect by applying a matching approach. However, our choice of which matching estimator to use is restricted by the possibility to obtain valid inference via bootstrap. As was it the case for our weighting estimator, ignoring the estimation error in the propensity score can lead to wrong inference when estimating the ATET via matching (Abadie & Imbens (2012)). Abadie & Imbens (2008) show that the standard bootstrap is not valid using the popular nearest neighbor matching, while they conjecture that it is for kernel based matching methods. In light of this discussion we use kernel matching on the propensity score with an Epanechnikov

[19] To avoid dependence of our results on an inappropriately chosen ϵ we also conduct the analysis for .10 or .20. The results were, however, almost identical to the one described in the text.
[20] We do not report trimmed results for external causes of death and cancer.

kernel and an optimal bandwidth given by Silverman's rule (Silverman (1998)).[21] We obtain standard errors by bootstrapping the whole estimation process.

Detailed results from the matching estimator can be found in the appendix (C). The results are in line with those obtained from weighting. In general, the ATETs obtained from matching indicate a smaller effect at the beginning but a stronger decline in the medium to long run. Furthermore, they are less precisely estimated compared to the effects obtained from propensity score weighting. However, our matching estimates do not lead to any different conclusions, neither in the case of the treatment effect nor in the time pattern or shape. Therefore, we can conclude that our results are robust to the method used.

7 Possible Factors affecting Mortality

Our results indicate that a child loss is associated with a higher mortality risk especially for fathers. In this section, we briefly investigate possible channels, trough which bereavement might affect mortality. van den Berg et al. (2012) find for Sweden that the death of a child is associated with long run adverse labor market outcomes and health problems. We explore these channels and compare the career path of bereaved parents to those not affected by a child loss.[22] To do so, we use a simple inverse probability weighting estimator using our ATET weights and adjusting the standard errors for the uncertainty in the propensity score estimation as for example in Newey & McFadden (1994).

We first investigate the difference in labor force participation, days worked and days spend in unemployment between members of the treatment and control group. An individual is considered as in the labor force for a given year, if she was at least one day employed or unemployed. In our analysis, we consider all types of employment, that is we include for example marginal workers, and assign individuals zero working days if they were in the labor force but were not employed.[23] Our estimated effects are depicted in figure (7). One can see from the figures that bereavement does neither affect the labor

[21] An alternative would be to match directly on the vector of explanatory variables, as our covariates are discrete. However, as in total we have more than 180 covariates, the number of exact matches is very low when using this method.

[22] Due to data limitations we are not able to do an in-depth analysis as van den Berg et al. (2012).

[23] Our results do not change if we conduct our analysis conditionally on non-zero employment days or a narrower definition of employment.

force participation rate nor unemployment for both fathers and mothers. Only mothers seem to have less employment days the year after the child has dies, but recover quickly afterward. As we do not observe hours worked in our data, we are not able to assess if bereaved parents tend to work less or more hours than individuals in the control group.

Considering every type of employment might mask the job quality. As bereavement might affect the performance and stamina in a job, we investigate if bereaved parents tend to be more employed in risky jobs and suffer from wage losses. We consider an individual to have a risky job if she is employed as a marginal worker, works in a subsidized job or are employed via a freelancer service contract.[24]. When investigating the effect of bereavement on wages, we include only blue- or white collar workers in our analysis. The reason for this is that we only have information on wages for individuals paying social security. Furthermore, we assess if parents in our treatment group have more sick-days absence. This can be a sign of serious ill health through which the stress of a child loss is affecting mortality. In the ASSD data sick-leaves are observed when the employer is not required to continue paying wages in case of sickness absence. The exact date depends on the tenure in the respective firm and the status, i.e. blue- or white collar worker. In general, during the first five years of tenure a sick leave is recorded if an individual is absent for longer than 6 weeks and this increases to 12 weeks after 25 years for blue and 26 years of tenure for white collar workers.

Our estimations are presented in figure (8). The first panel give the results for our broad category of job quality. On average, neither beavered mothers nor bereaved fathers tend to work more days in risky jobs. A similar picture emerges if we consider wages. For both individuals in the treatment and control group wage evolve in a similar fashion and the difference is negligible, as can be seen in panel (b). However, for both genders there is a significant increase in average sick-absence days the year following the death of the child. The jump is higher for mothers, suggesting that they suffer stronger from the bereavement in the short run. This result supports our findings from the previous sections that the stress of a child loss affects mortality through ill-health.

In a last step, we investigate the effect of the child death on the retirement pattern of

[24]Individuals employed via freelancer service contract or "Freier Dienstvertrag"' are independent of the organization of the firm and the contract is signed only over a pre-specified period.

parents, where we also have a closer look at retirement due to health impairment, the so called disability-retirement. An individual can enter disability retirement not only if she has physical impairment but also psychic or mental problems. Disability retirement is, as our definition of sick-leaves, an indicator for serious ill-health. As we regard the inflow into retirement as an absorbing state and our sample for retirement contains censored spells, we estimate the ATET in these cases as the difference in the cumulative hazard rates between both groups. This is done using a propensity score weighted Nelson-Aalen estimator with bootstrapped standard errors. Figure (9) presents the results. Panel (a) depicts our results for overall retirement regardless of the reason. One can see that there is a significant difference in the retirement pattern around 8 years after the bereavement for fathers and very close to the child's death date for mothers. Panel (b) shows our estimates for disability retirement. From the figure it is obvious that the retirement pattern of bereaved parents is driven by retirement due to disability. Furthermore, the time pattern of disability retirement for fathers coincides with the fall in the survival probabilities discussed earlier. These findings are in line with van den Berg et al. (2012), who finds that bereaved parents have a higher likelihood of leaving employment.

The results in this section suggest, that the main channel through which bereavement is affecting mortality is health impairment, at least for those who choose to remain in the labor market. We do not find that labor market outcomes are affected by a child loss, however this might be partly due to some data limitation on hours worked, non-labor income components and career outside the labor market. However, both bereaved fathers and mothers have a higher probability of leaving the labor market due to health impairments. In particular for fathers, the timing of the labor market exit coincides with that point in time, where we can see the largest drop in the survival probability.

8 Conclusion

In this study, we examine the time pattern of stress on mortality using a child's death as the triggering event. This topic has so far been covered only sparsely in the literature, despite its importance. We estimate the time pattern of the bereavement effect in greater detail without imposing functional form restrictions while accounting for various background

characteristics. Given our approach and the rich set of covariates at hand, our analysis captures the causal treatment effect of a child's death on the mortality of the parents.

We find strong and significant bereavement effects on the relative survival probability of fathers. The associated time pattern is far from being stable over time. Our results indicate a strong fall in the relative survival probability during the first 13 years after the event and a slight reversal afterward. Investigating the driving forces in more detail we find that bereaved fathers have a lower relative survival probability for natural causes but do not find any evidence for unnatural ones. Furthermore, we examine the effect of grief on two death causes commonly associated with stress: circulatory diseases and cancer. Our results indicate an increasing risk of dying from circulatory diseases but we do not find significant results for cancer. For mothers, we only find modest evidence and only when considering overall mortality and death due to natural causes. These results are somewhat surprising in light of the previous findings in the literature but might be related to gender specific strategies to deal with stress.

Investigating possible channels through which a child loss might affect the mortality of parents, we do not find any evidence that labor market outcomes are negatively affects. However, we find that for both bereaved fathers and mothers the probability of leaving the labor market due to health-related problems is significantly higher than for their counterparts in the control group. These results support our findings in the main part of the paper and indicate that stress is affecting mortality through ill-health.

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Tables

Table 1: Children's Death Causes by Broad Categories

Cause	**Share in %**
Accidents	6.31
Accidental Poisoning	5.22
Falls & Drops	4.91
Fire	1.39
Medical Complications	0.24
Traffic Accidents	80.11
Weather	1.70

The following grouping was used (according to ICD-9): Accidents: E9100-E9270; Accidental Poisoning: E8500-E8699, E9292; Falls & Drops: E8800-E8880, E9293; Fire: E8900-8990, E9294; Medical Complications: E8700-E8799, E9300-E9499; Traffic Accidents: E8000-E8480, E9290, E9291; Weather: E9000-E9059, E9070, E9080, E9090, E9280, E9281-9282, E9288, E9295.

Table 2: Summary of the Data

	All	Mother	Father
Bereaved			
Sample Size:	1,649	868	781
of which % died	4.37	2.42	6.53
Control			
Sample Size:	130,518	66,668	63,850
of which % died	2.94	1.69	4.24

The construction of the sample is described in the text.

Table 3: Summary Statistic of Estimated Propensity Score

	min. p(x)	max. p(x)	mean p(x)	min. Weight	max. Weight	mean Weight	N
Overall Sample							
Total							
Bereaved	2.87×10^{-4}	0.21	0.024	1.00	1.00	1.00	1,649
Control	8×10^{-6}	0.17	0.012	8×10^{-6}	0.21	0.013	130,518
Trimmed Sample							
Bereaved	2.87×10^{-4}	0.10	0.024	1.00	1.00	1.00	1,638
Control	8×10^{-6}	0.10	0.012	8×10^{-6}	0.11	0.013	130,406
Mothers							
Total							
Bereaved	3.0×10^{-4}	0.25	0.026	1.00	1.00	1.00	868
Control	1.3×10^{-5}	0.23	0.013	1.3×10^{-5}	0.30	0.013	66,668
Trimmed Sample							
Bereaved	3.0×10^{-4}	0.11	0.025	1.00	1.00	1.00	862
Control	1.3×10^{-5}	0.11	0.013	1.10×10^{-5}	0.12	0.014	66,600
Fathers							
Total							
Bereaved	7.15×10^{-4}	0.15	0.026	1.00	1.00	1.00	781
Control	2.0×10^{-6}	0.15	0.012	2.0×10^{-6}	0.17	0.012	63,850
Trimmed Sample							
Bereaved	7.15×10^{-4}	0.11	0.025	1.00	1.00	1.00	775
Control	2.0×10^{-6}	0.11	0.012	2.0×10^{-6}	0.12	0.012	63,801

The weights are calculated as $w = T + (1 - T)\frac{p(x)}{1-p(x)}$, where T is the treatment indicator and $p(x)$ the estimated propensity score. The trimmed sample comprises of all observations with a propensity score within the common support $S_{trimmed} = \left\{p(x) : \hat{f}(p(x)|T = 1) \cap \hat{f}(p(x)|T = 0) > \epsilon\right\}$ as in Smith & Todd (2005). $\hat{f}(\bullet)$ is obtained by kernel density estimation with a Gaussian Kernel and a smoothing parameter determined as in the text. The cut off ϵ is chosen to be .05

Table 4: Balancing Property of Propensity Score (Full Sample)

	Mean before weighting		SDM	t-val	Mean after weighting	SDM	t-val
	Untreated	Treated	in %		Untreated	in %	
Age 20-30	0.03	0.00	16.80	−22.42	0.00	0.01	0.00
Age 31-35	0.11	0.02	28.27	−24.99	0.02	0.02	0.01
Age 36-40	0.22	0.21	2.12	−0.87	0.21	0.04	0.01
Age 41-45	0.29	0.36	16.73	6.36	0.36	0.01	0.00
Age 46-50	0.21	0.24	7.38	2.84	0.24	0.06	−0.02
Age 51-55	0.10	0.12	5.34	2.03	0.12	0.05	0.02
Age 56-60	0.04	0.04	1.09	0.43	0.04	0.04	−0.02
Daily Income (t-1) $X \leq 20$	0.17	0.15	5.30	−2.25	0.15	0.05	−0.02
Daily Income (t-1) $20 < X \leq 40$	0.07	0.07	0.92	−0.38	0.07	0.01	−0.01
Daily Income (t-1) $40 < X \leq 60$	0.11	0.12	2.76	1.08	0.12	0.04	−0.02
Daily Income (t-1) $60 < X \leq 80$	0.12	0.11	3.69	−1.55	0.11	0.07	0.03
Daily Income (t-1) $80 < X \leq 100$	0.14	0.14	0.04	−0.02	0.14	0.02	0.01
Daily Income (t-1) $100 < X \leq 120$	0.13	0.13	0.24	−0.10	0.13	0.03	−0.01
Daily Income (t-1) $120 < X \leq 140$	0.09	0.10	2.31	0.90	0.10	0.03	0.01
Daily Income (t-1) $140 < X$	0.18	0.20	5.19	2.01	0.20	0.02	0.01
Daily Income (t-2) $X \leq 20$	0.17	0.17	1.75	−0.72	0.17	0.09	−0.03
Daily Income (t-2) $20 < X \leq 40$	0.08	0.06	5.17	−2.29	0.06	0.03	0.01
Daily Income (t-2) $40 < X \leq 60$	0.12	0.12	0.15	−0.06	0.12	0.01	0.00
Daily Income (t-2) $60 < X \leq 80$	0.13	0.14	2.90	1.14	0.14	0.01	0.00
Daily Income (t-2) $80 < X \leq 100$	0.14	0.12	4.56	−1.94	0.12	0.06	0.02
Daily Income (t-2) $100 < X \leq 120$	0.13	0.12	1.94	−0.80	0.12	0.02	0.01
Daily Income (t-2) $120 < X \leq 140$	0.08	0.10	4.77	1.80	0.10	0.06	0.02
Daily Income (t-2) $140 < X$	0.16	0.18	5.47	2.11	0.18	0.05	−0.02
Unemployment(t - t-4) $X \leq 1$	0.76	0.73	6.98	−2.71	0.73	0.02	0.01
Unemployment(t - t-4) $1 < X \leq 30$	0.02	0.02	1.60	−0.68	0.02	0.01	0.00
Unemployment(t - t-4) $30 < X \leq 60$	0.02	0.01	3.72	−1.74	0.01	0.02	−0.01
Unemployment(t - t-4) $60 < X \leq 90$	0.02	0.02	0.06	−0.03	0.02	0.08	−0.03
Unemployment(t - t-4) $90 < X \leq 120$	0.01	0.02	4.62	1.60	0.02	0.02	−0.01
Unemployment(t - t-4) $120 < X \leq 150$	0.02	0.02	1.19	0.46	0.02	0.02	−0.01
Unemployment(t - t-4) $150 < X \leq 180$	0.01	0.01	0.93	−0.39	0.01	0.04	−0.02
Unemployment(t - t-4) $180 < X \leq 210$	0.01	0.01	1.02	−0.43	0.01	0.07	−0.03
Unemployment(t - t-4) $210 < X \leq 240$	0.01	0.01	2.74	−1.27	0.01	0.00	0.00
Unemployment(t - t-4) $240 < X \leq 270$	0.01	0.01	0.79	0.31	0.01	0.10	0.04
Unemployment(t - t-4) $270 < X$	0.10	0.14	11.25	4.02	0.14	0.03	0.01
Sick Leaves (t - t-4) $X \leq 1$	0.85	0.82	9.44	−3.52	0.82	0.03	−0.01
Sick Leaves (t - t-4) $1 < X \leq 10$	0.04	0.04	0.13	−0.05	0.04	0.05	0.02
Sick Leaves (t - t-4) $10 < X \leq 30$	0.05	0.06	4.64	1.72	0.06	0.03	0.01
Sick Leaves (t - t-4) $30 < X \leq 50$	0.02	0.03	3.89	1.40	0.03	0.03	0.01
Sick Leaves (t - t-4) $50 < X \leq 70$	0.01	0.01	0.77	0.30	0.01	0.02	−0.01
Sick Leaves (t - t-4) $70 < X$	0.03	0.05	10.08	3.30	0.05	0.03	−0.01
Education: Others	0.01	0.00	3.51	−1.91	0.00	0.01	0.00
Education: No School	0.17	0.22	12.35	4.56	0.22	0.08	0.03
Education: Compulsory School	0.38	0.42	7.56	3.01	0.42	0.03	−0.01
Education: Apprenticeship	0.12	0.11	5.35	−2.30	0.11	0.02	0.01
Education: Junior High School	0.07	0.04	10.62	−5.40	0.04	0.00	0.00
Education: Senior High School	0.08	0.03	16.31	−9.68	0.03	0.02	−0.01
Education: University	0.18	0.18	1.72	0.69	0.18	0.05	−0.02
No. of Children> 2	0.07	0.01	21.25	−17.76	0.01	0.01	0.01
Widowed	0.01	0.01	3.35	1.13	0.01	0.03	0.01
Single	0.05	0.10	24.57	7.15	0.10	0.06	−0.02
Foreigner	0.05	0.03	7.14	−3.44	0.03	0.01	0.01
Mother	0.51	0.53	3.12	1.26	0.51	3.88	1.56
Blue Collar Worker	0.36	0.42	12.54	4.93	0.42	0.01	0.00

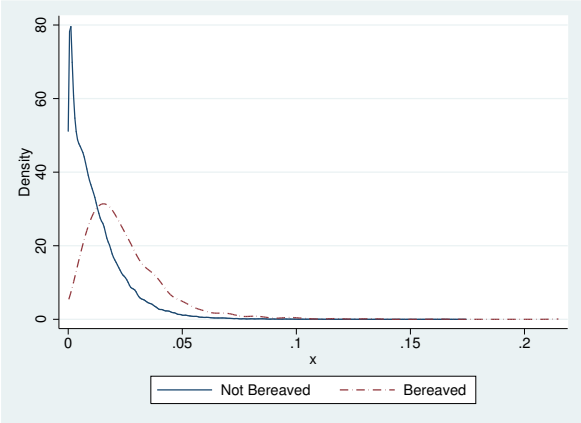
Unemployment refers to the cumulative unemployment duration over the past four years, over which the individual was not holding ANY employment. Sick Leaves refers to the cumulative duration over which sick leave allowance was paid. This happens when an individual is more than six weeks incapable of working.

SDM referst to the standardized differenze in means, which is given by $SDM_x = \frac{|\overline{X}_{treat} - \overline{X}_{control}|}{\sqrt{\sigma^2_{Pooled}}} \cdot 100$

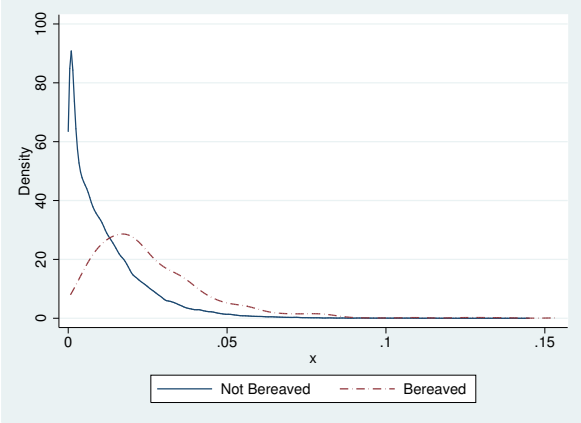
Figures

Figure 1: Distribution of Propensity Score by Treatment Status

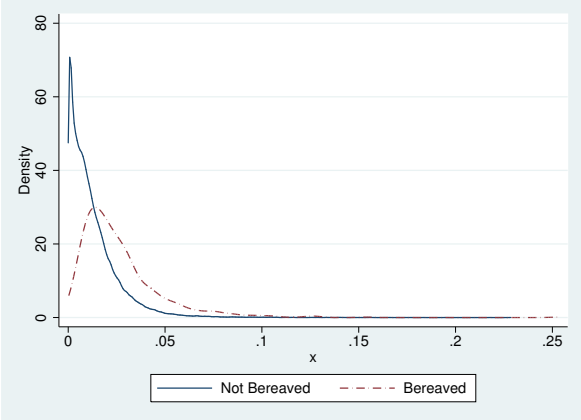
(a) Distribution for the Whole Sample



(b) Distribution for the Fathers-Only Sample



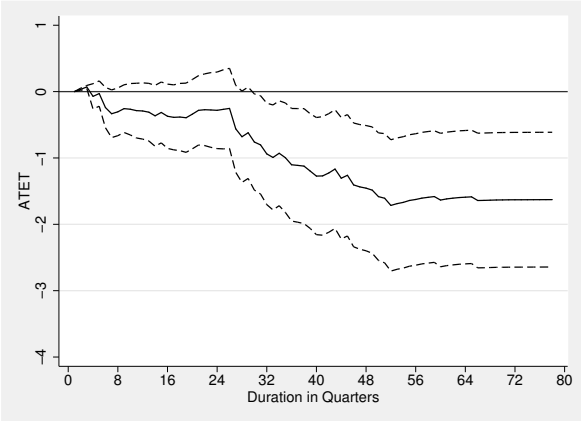
(c) Distribution for the Mothers-Only Sample



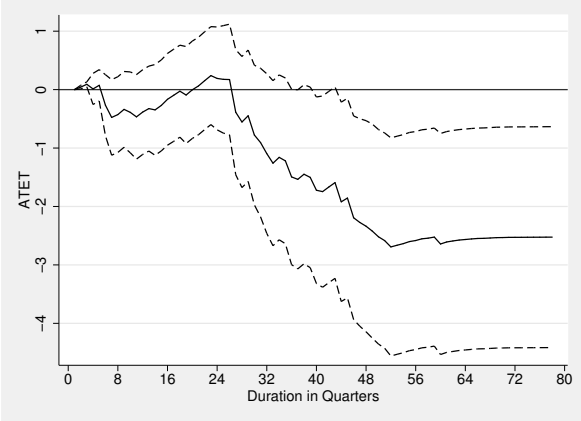
The distributions are estimated by kernel density estimation with a bandwidth $h_{smooth} = h_{opt}\, n^{-\frac{1}{10}}$, where h_{opt} is determined by Silverman's rule (Silverman (1998))

Figure 2: Estimated Treatment Effect for Overall Mortality

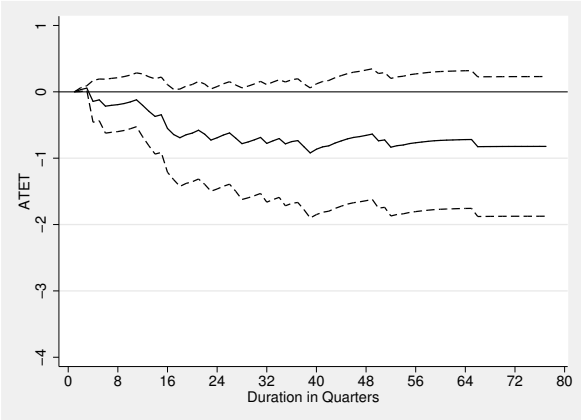
(a) ATET for the Whole Sample



(b) ATET for Father-Only Sample



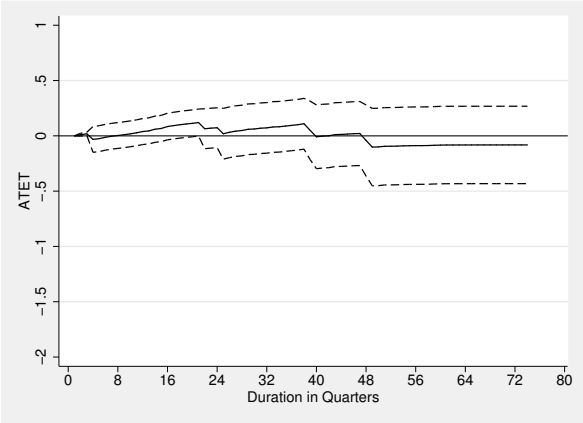
(c) ATET for Mothers-Only Sample



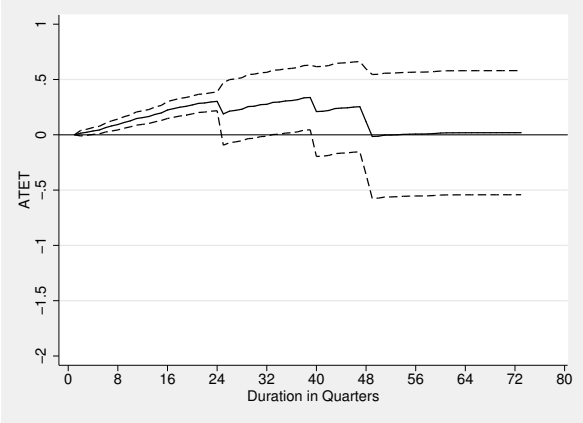
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure 3: Estimated Treatment Effect for External Causes

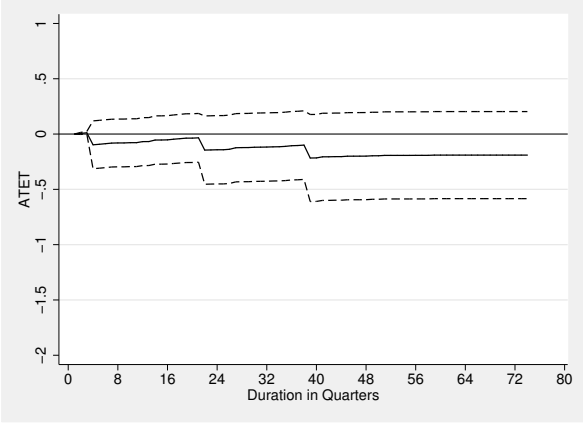
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



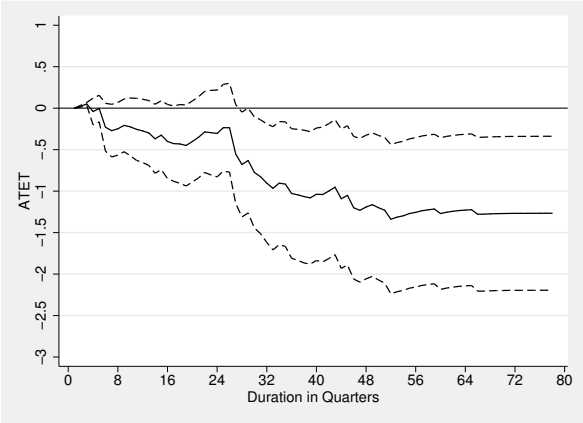
(c) ATET for the Mothers-Only Sample



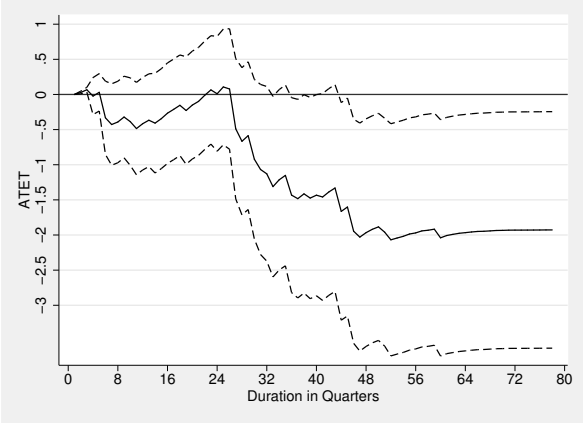
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure 4: Estimated Treatment Effect for Natural Causes

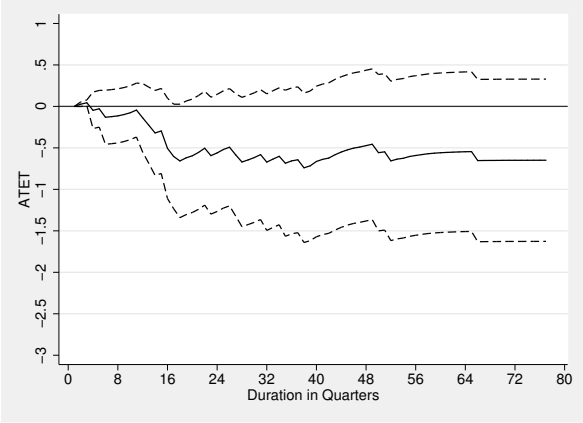
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



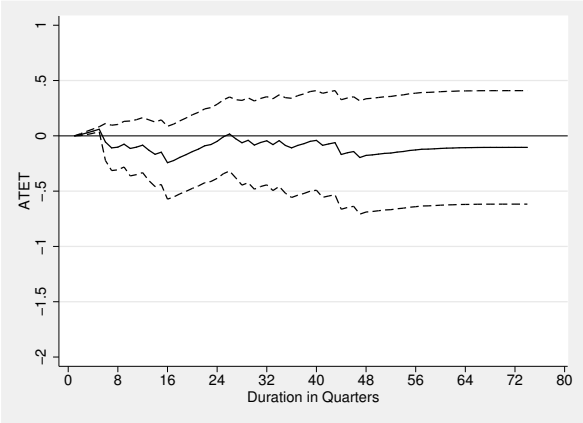
(c) ATET for the Mothers-Only Sample



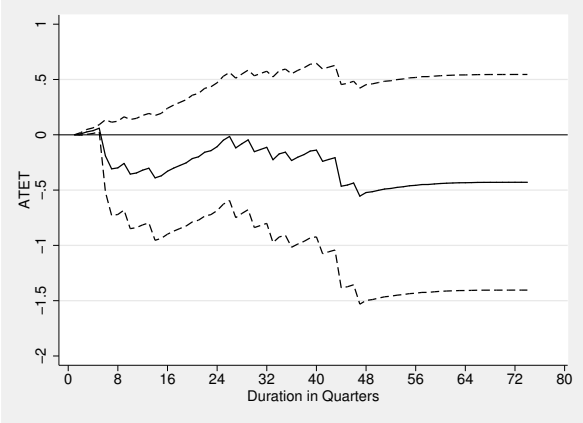
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure 5: Estimated Treatment Effect for Cancer

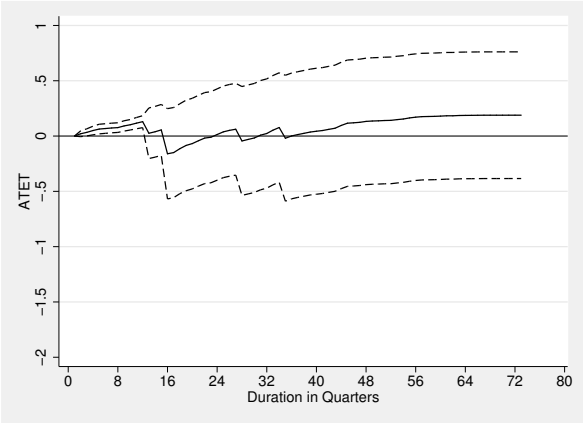
(a) ATET for the Whole Sample



(b) ATET for the Whole Sample



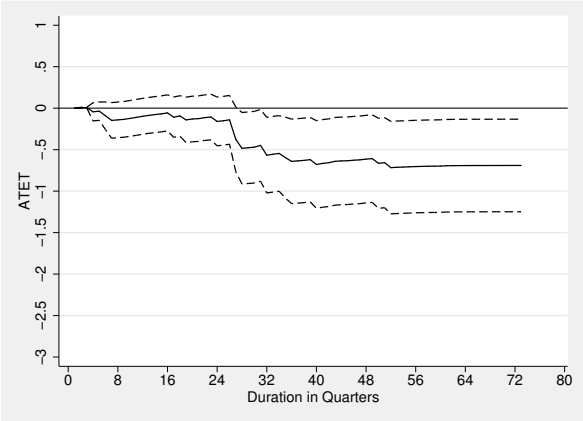
(c) ATET for the Whole Sample



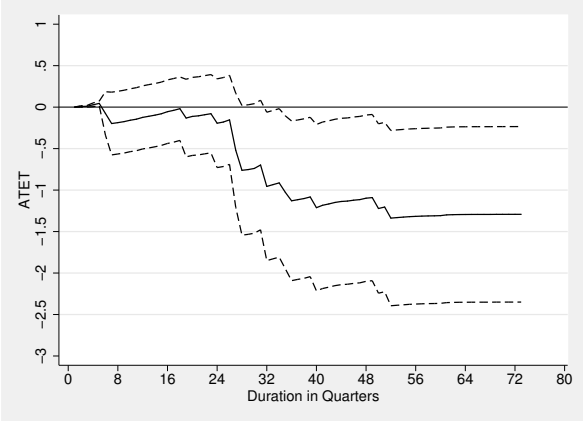
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure 6: Estimated Treatment Effect for Circulatory Diseases

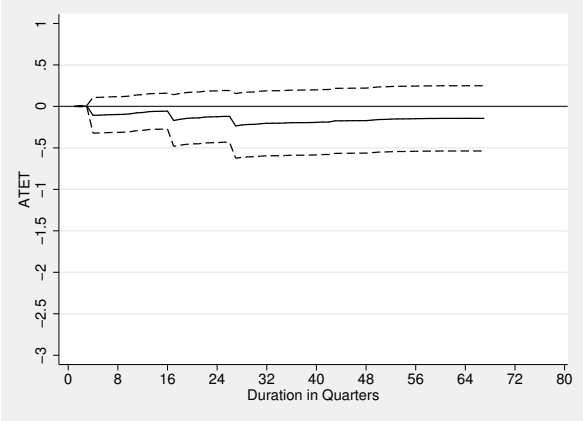
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



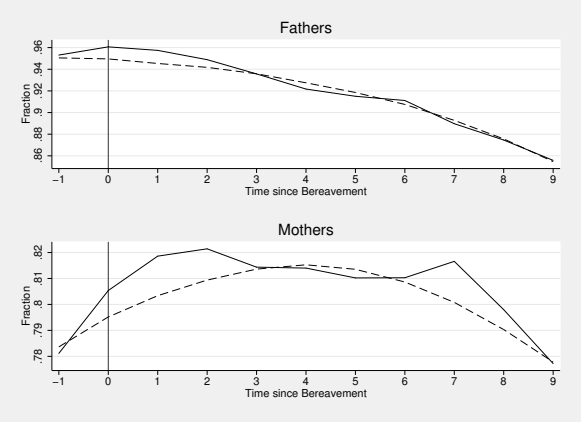
(c) ATET for the Mothers-Only Sample



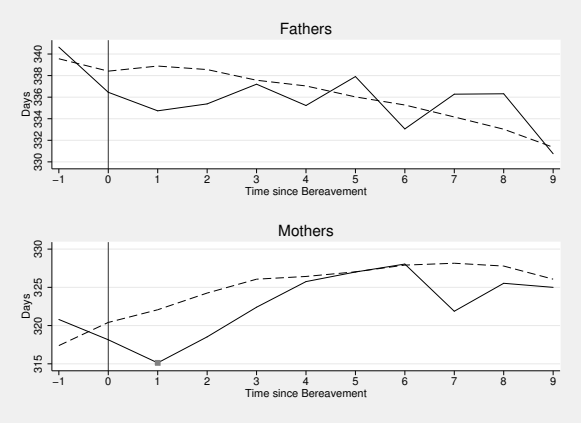
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure 7: Effect on Employment Outcomes

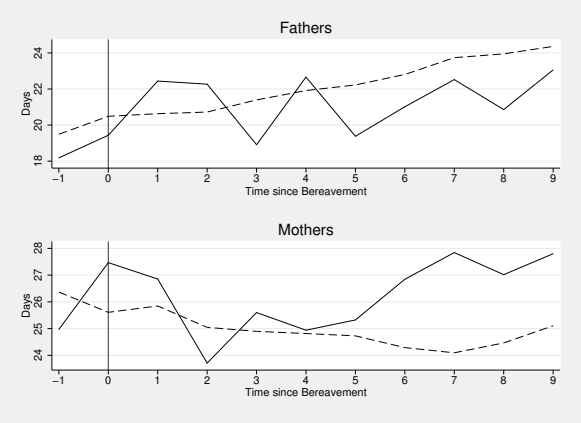
(a) Effect on Labor-Force Participation



(b) Effect on Emplyoment



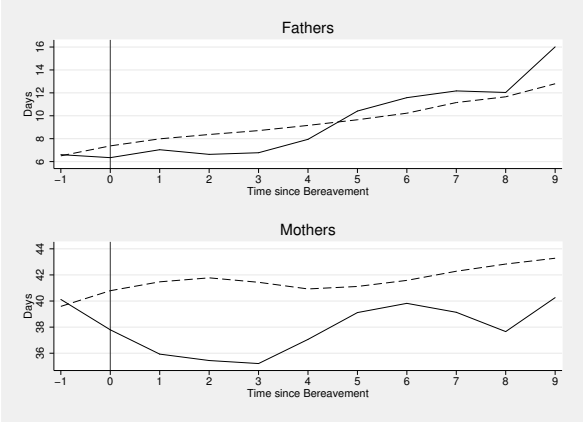
(c) Effect on Unemployment



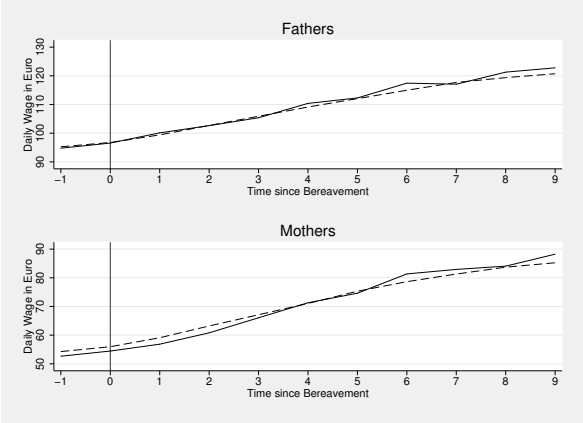
The solid line and the dashed line represent the treatment and control group respectively. The ATET was estimated using a simple IPW-Estimator. The horizontal axis depicts the elapsed time from the base quarter in years. Standard errors are adjusted for the estimation of the propensity score. Dots indicate a significant difference at the 95% level.

Figure 8: Effect on Monetary and Health-Related Outcomes

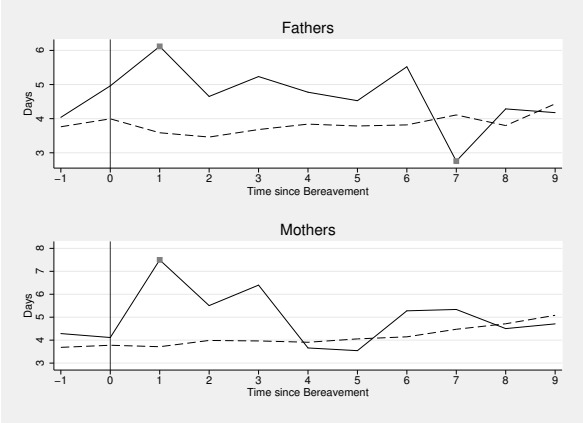
(a) Effect on Risky-Jobs



(b) Effect on Wages



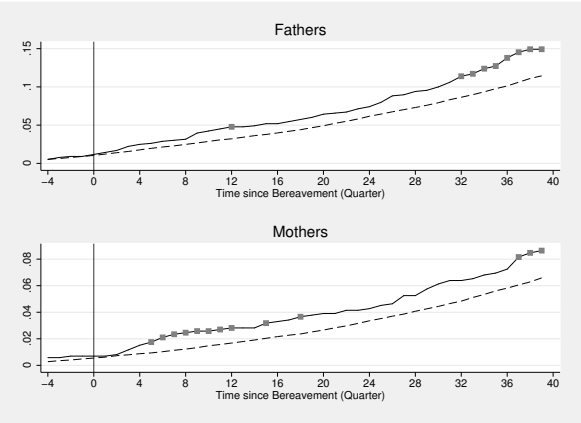
(c) Effect on Sick-Leaves



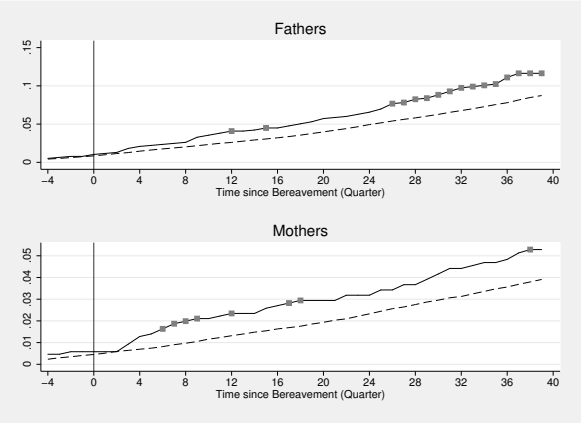
The solid line and the dashed line represent the treatment and control group respectively. The ATET was estimated using a simple IPW-Estimator. The horizontal axis depicts the elapsed time from the base quarter in years. Standard errors are adjusted for the estimation of the propensity score. Dots indicate a significant difference at the 95% level.

Figure 9: Effect on Retirement

(a) Effect on Overall Retirement



(b) Effect on Disability Retirement



The solid line and the dashed line represent the treatment and control group respectively. The ATET was estimated using the difference of the cumulative hazard rate between both groups. The horizontal axis depicts the elapsed time from the base quarter in quarters. Standard errors are obtained after 500 bootstrap replications.. Dots indicate a significant difference at the 95% level.

A Balancing Property by Gender

This section reports detailed balancing properties of the propensity score separately by gender. The standardized difference in means is calculated as given in the main text. As it is the case for the full sample, there is no evidence that our sub-samples are not balanced.

Table A.1: Balancing Property of Propensity Score (Mothers only)

	Mean before weighting		SDM	t-val	Mean after weighting	SDM	t-val
	Untreated	Treated	in %		Untreated	in %	
Age 20-30	0.04	0.00	19.39	−21.06	0.00	0.00	0.00
Age 31-35	0.13	0.03	30.02	−17.19	0.03	0.01	0.00
Age 36-40	0.26	0.29	6.59	1.87	0.29	0.01	0.00
Age 41-45	0.30	0.38	17.57	4.85	0.38	0.03	0.01
Age 46-50	0.18	0.20	4.81	1.36	0.20	0.06	−0.02
Age 51-55	0.07	0.08	2.77	0.78	0.08	0.01	0.00
Age 56-60	0.02	0.03	2.09	0.58	0.03	0.00	0.00
Daily Income (t-1) $X \leq 20$	0.18	0.17	3.91	−1.18	0.17	0.00	0.00
Daily Income (t-1) $20 < X \leq 40$	0.08	0.08	0.48	0.14	0.08	0.04	−0.01
Daily Income (t-1) $40 < X \leq 60$	0.11	0.12	1.32	0.38	0.12	0.16	−0.05
Daily Income (t-1) $60 < X \leq 80$	0.11	0.10	3.45	−1.05	0.10	0.03	0.01
Daily Income (t-1) $80 < X \leq 100$	0.13	0.13	0.21	−0.06	0.13	0.02	0.01
Daily Income (t-1) $100 < X \leq 120$	0.12	0.12	1.06	−0.31	0.12	0.02	−0.01
Daily Income (t-1) $120 < X \leq 140$	0.09	0.09	1.98	0.56	0.09	0.12	0.04
Daily Income (t-1) $140 < X$	0.17	0.19	5.12	1.44	0.19	0.05	0.01
Daily Income (t-2) $X \leq 20$	0.19	0.19	1.04	−0.31	0.19	0.07	−0.02
Daily Income (t-2) $20 < X \leq 40$	0.08	0.07	3.58	−1.11	0.07	0.02	0.01
Daily Income (t-2) $40 < X \leq 60$	0.12	0.11	1.91	−0.57	0.11	0.16	−0.05
Daily Income (t-2) $60 < X \leq 80$	0.13	0.14	3.40	0.96	0.14	0.02	0.00
Daily Income (t-2) $80 < X \leq 100$	0.13	0.12	4.32	−1.33	0.12	0.05	0.01
Daily Income (t-2) $100 < X \leq 120$	0.12	0.11	1.77	−0.53	0.11	0.05	0.01
Daily Income (t-2) $120 < X \leq 140$	0.08	0.09	4.09	1.13	0.09	0.12	0.04
Daily Income (t-2) $140 < X$	0.15	0.17	5.06	1.41	0.17	0.00	0.00
Unemployment(t - t-4) $X \leq 1$	0.75	0.73	5.80	−1.65	0.73	0.03	0.01
Unemployment(t - t-4) $1 < X \leq 30$	0.02	0.03	6.79	1.64	0.03	0.04	0.01
Unemployment(t - t-4) $30 < X \leq 60$	0.02	0.02	0.75	−0.23	0.02	0.02	−0.01
Unemployment(t - t-4) $60 < X \leq 90$	0.02	0.02	2.86	0.76	0.02	0.12	−0.04
Unemployment(t - t-4) $90 < X \leq 120$	0.01	0.01	0.01	0.00	0.01	0.00	0.00
Unemployment(t - t-4) $120 < X \leq 150$	0.02	0.01	1.96	−0.62	0.02	0.06	−0.02
Unemployment(t - t-4) $150 < X \leq 180$	0.02	0.01	0.98	−0.30	0.01	0.05	−0.02
Unemployment(t - t-4) $180 < X \leq 210$	0.02	0.01	4.05	−1.43	0.01	0.05	−0.01
Unemployment(t - t-4) $210 < X \leq 240$	0.01	0.01	4.16	−1.52	0.01	0.04	−0.01
Unemployment(t - t-4) $240 < X \leq 270$	0.01	0.01	1.17	0.33	0.01	0.13	0.04
Unemployment(t - t-4) $270 < X$	0.11	0.13	8.12	2.18	0.13	0.03	0.01
Sick Leaves (t - t-4) $X \leq 1$	0.86	0.84	8.36	−2.27	0.84	0.00	0.00
Sick Leaves (t - t-4) $1 < X \leq 10$	0.04	0.04	0.37	0.11	0.04	0.07	0.02
Sick Leaves (t - t-4) $10 < X \leq 30$	0.05	0.06	4.64	1.72	0.06	0.03	0.01
Sick Leaves (t - t-4) $30 < X \leq 50$	0.02	0.02	3.36	0.89	0.02	0.01	0.00
Sick Leaves (t - t-4) $50 < X \leq 70$	0.01	0.01	3.53	−1.26	0.01	0.04	−0.01
Sick Leaves (t - t-4) $70 < X$	0.03	0.04	10.01	2.34	0.04	0.09	−0.02
Education: Others	0.01	0.00	2.34	−0.81	0.00	0.02	−0.01
Education: No School	0.22	0.28	16.09	4.32	0.28	0.07	0.02
Education: Compulsory School	0.32	0.34	6.29	1.80	0.34	0.04	−0.01
Education: Apprenticeship	0.16	0.13	7.96	−2.53	0.13	0.03	−0.01
Education: Junior High School	0.06	0.04	10.05	−3.69	0.04	0.03	0.01
Education: Senior High School	0.06	0.03	13.61	−5.62	0.03	0.01	0.00
Education: University	0.18	0.17	1.96	−0.58	0.17	0.01	0.00
No. of Children> 2	0.07	0.02	19.01	−9.84	0.02	0.02	0.01
Widowed	0.01	0.01	5.44	1.28	0.01	0.00	0.00
Single	0.06	0.14	32.81	6.67	0.14	0.13	−0.04
Foreigner	0.04	0.03	4.51	−1.48	0.03	0.08	0.02
Blue Collar Worker	0.30	0.35	11.12	3.13	0.35	0.00	0.00

Unemployment refers to the cumulative unemployment duration over the past four years, over which the individual was not holding ANY employment. Sick Leaves refers to the cumulative duration over which sick leave allowance was paid. This happens when an individual is more than six weeks incapable of working.

SDM referst to the standardized differenze in means, which is given by $SDM_x = \frac{|\overline{X}_{treat} - \overline{X}_{control}|}{\sqrt{\sigma^2_{Pooled}}} \cdot 100$

Table A.2: Balancing Property of Propensity Score (Fathers only)

	Mean before weighting		SDM	t-val	Mean after weighting	SDM	t-val
	Untreated	Treated	in %		Untreated	in %	
Age 20-30	0.02	0.00	13.66	−10.67	0.00	0.02	0.00
Age 31-35	0.08	0.01	26.83	−21.92	0.01	0.02	0.00
Age 36-40	0.18	0.13	13.72	−4.37	0.13	0.05	0.01
Age 41-45	0.28	0.35	15.63	4.09	0.35	0.06	−0.02
Age 46-50	0.24	0.28	10.49	2.76	0.28	0.03	−0.01
Age 51-55	0.14	0.17	8.32	2.14	0.17	0.10	0.03
Age 56-60	0.06	0.06	0.99	0.27	0.06	0.05	−0.01
Daily Income (t-1) $X \leq 20$	0.15	0.12	7.36	−2.22	0.12	0.04	−0.01
Daily Income (t-1) $20 < X \leq 40$	0.06	0.06	2.88	−0.84	0.06	0.02	−0.01
Daily Income (t-1) $40 < X \leq 60$	0.11	0.12	4.33	1.14	0.12	0.06	0.02
Daily Income (t-1) $60 < X \leq 80$	0.13	0.12	3.82	−1.11	0.11	0.14	0.04
Daily Income (t-1) $80 < X \leq 100$	0.14	0.14	0.27	0.07	0.14	0.01	0.00
Daily Income (t-1) $100 < X \leq 120$	0.14	0.14	0.76	0.21	0.14	0.07	−0.02
Daily Income (t-1) $120 < X \leq 140$	0.09	0.10	2.72	0.73	0.10	0.03	−0.01
Daily Income (t-1) $140 < X$	0.18	0.20	5.40	1.44	0.21	0.03	−0.01
Daily Income (t-2) $X \leq 20$	0.15	0.14	2.97	−0.85	0.14	0.09	−0.02
Daily Income (t-2) $20 < X \leq 40$	0.07	0.05	7.37	−2.37	0.05	0.03	0.01
Daily Income (t-2) $40 < X \leq 60$	0.12	0.12	1.80	0.49	0.12	0.08	0.02
Daily Income (t-2) $60 < X \leq 80$	0.14	0.15	2.49	0.67	0.15	0.05	0.01
Daily Income (t-2) $80 < X \leq 100$	0.14	0.13	4.72	−1.38	0.13	0.08	0.02
Daily Income (t-2) $100 < X \leq 120$	0.13	0.13	1.98	−0.56	0.13	0.01	0.00
Daily Income (t-2) $120 < X \leq 140$	0.09	0.10	5.58	1.44	0.10	0.02	0.00
Daily Income (t-2) $140 < X$	0.16	0.18	6.03	1.59	0.18	0.13	−0.04
Unemployment(t - t-4) $X \leq 1$	0.77	0.74	8.17	−2.17	0.74	0.02	0.00
Unemployment(t - t-4) $1 < X \leq 30$	0.02	0.00	10.96	−6.47	0.00	0.02	−0.01
Unemployment(t - t-4) $30 < X \leq 60$	0.02	0.01	6.88	−2.62	0.01	0.01	0.00
Unemployment(t - t-4) $60 < X \leq 90$	0.02	0.01	3.42	−1.09	0.01	0.05	−0.01
Unemployment(t - t-4) $90 < X \leq 120$	0.02	0.03	9.73	2.05	0.03	0.07	−0.02
Unemployment(t - t-4) $120 < X \leq 150$	0.01	0.02	4.93	1.16	0.02	0.11	0.03
Unemployment(t - t-4) $150 < X \leq 180$	0.01	0.01	0.95	−0.27	0.01	0.03	−0.01
Unemployment(t - t-4) $180 < X \leq 210$	0.01	0.01	2.76	0.68	0.01	0.06	−0.02
Unemployment(t - t-4) $210 < X \leq 240$	0.01	0.01	1.05	−0.31	0.01	0.03	0.01
Unemployment(t - t-4) $240 < X \leq 270$	0.01	0.01	0.35	0.10	0.01	0.12	0.03
Unemployment(t - t-4) $270 < X$	0.10	0.14	14.77	3.51	0.14	0.02	−0.01
Sick Leaves (t - t-4) $X \leq 1$	0.83	0.79	10.87	−2.78	0.79	0.03	−0.01
Sick Leaves (t - t-4) $1 < X \leq 10$	0.04	0.04	0.54	−0.15	0.04	0.01	0.00
Sick Leaves (t - t-4) $10 < X \leq 30$	0.05	0.06	4.03	1.04	0.06	0.02	0.01
Sick Leaves (t - t-4) $30 < X \leq 50$	0.02	0.03	4.54	1.11	0.03	0.06	0.02
Sick Leaves (t - t-4) $50 < X \leq 70$	0.01	0.02	5.08	1.19	0.02	0.01	0.00
Sick Leaves (t - t-4) $70 < X$	0.04	0.06	10.51	2.39	0.06	0.01	0.00
Education: Others	0.00	0.00	5.11	−2.68	0.00	0.01	0.00
Education: No School	0.12	0.14	6.45	1.67	0.14	0.09	0.02
Education: Compulsory School	0.45	0.50	9.97	2.76	0.50	0.07	−0.02
Education: Apprenticeship	0.09	0.08	2.55	−0.74	0.08	0.05	0.01
Education: Junior High School	0.07	0.04	11.12	−3.89	0.04	0.01	0.00
Education: Senior High School	0.09	0.04	18.78	−7.90	0.04	0.02	−0.01
Education: University	0.18	0.20	5.82	1.55	0.20	0.02	−0.01
No. of Children> 2	0.07	0.01	23.74	−17.96	0.01	0.01	0.00
Widowed	0.00	0.00	0.74	−0.22	0.00	0.14	0.04
Single	0.04	0.07	13.33	2.94	0.07	0.25	0.07
Foreigner	0.06	0.04	9.45	−3.28	0.04	0.00	0.00
Blue Collar Worker	0.42	0.50	15.06	4.14	0.50	0.03	0.01

Unemployment refers to the cumulative unemployment duration over the past four years, over which the individual was not holding ANY employment. Sick Leaves refers to the cumulative duration over which sick leave allowance was paid. This happens when an individual is more than six weeks incapable of working.

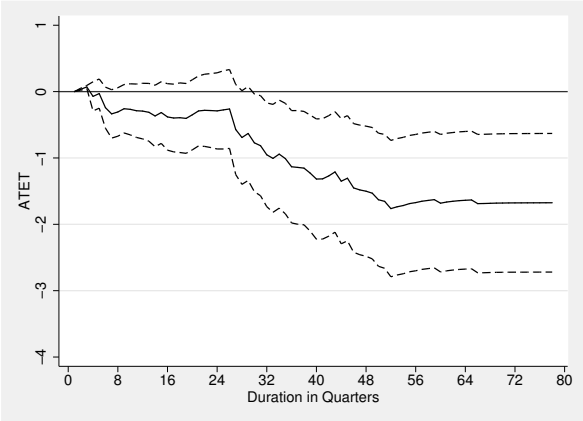
SDM referst to the standardized differenze in means, which is given by $SDM_x = \frac{|\overline{X}_{treat} - \overline{X}_{control}|}{\sqrt{\sigma^2_{Pooled}}} \cdot 100$

B Trimming

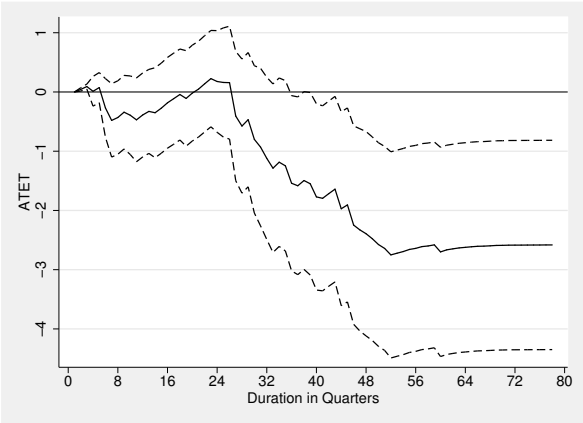
This section reports the results for the trimming samples as described in the text. All standard errors and confidence intervals are obtained from 500 bootstrap replications of the entire trimming process. That is for each bootstrap sample, first the common support is calculated and then the weights and treatment effects for all individuals within this common support.

Figure B.1: Estimated Treatment Effect for Overall Mortality (Trimmed Sample)

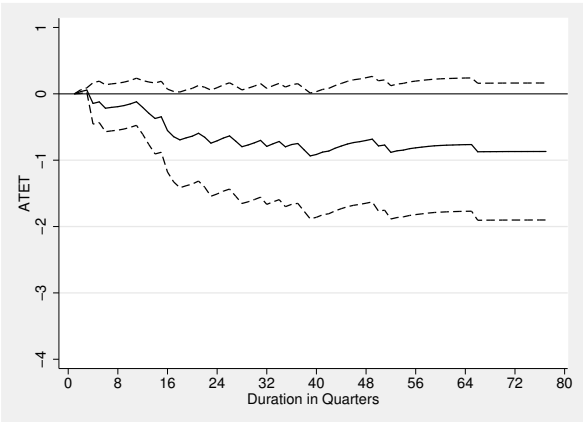
(a) ATET for the Trimmed Sample



(b) ATET for Father-Only Trimmed Sample



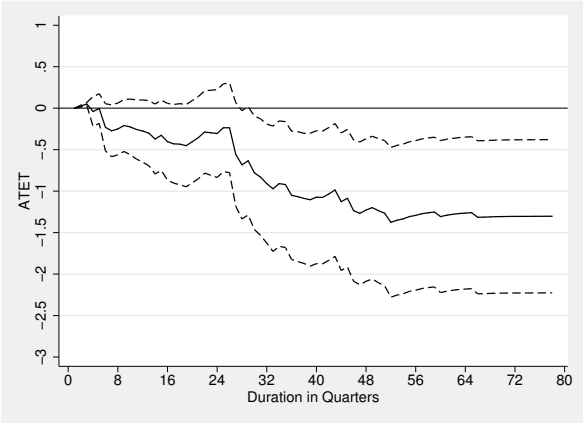
(c) ATET for Mothers-Only Trimmed Sample



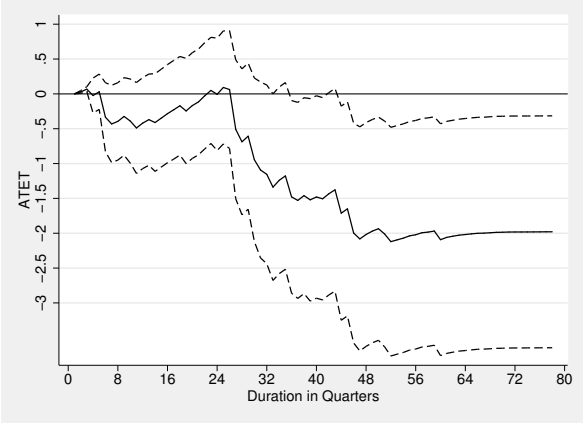
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications of the whole process.

Figure B.2: Estimated Treatment Effect for Natural Causes (Trimmed Sample)

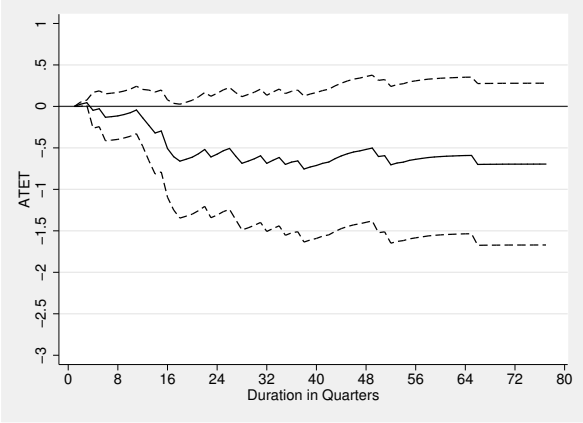
(a) ATET for the Trimmed Sample



(b) ATET for Father-Only Trimmed Sample



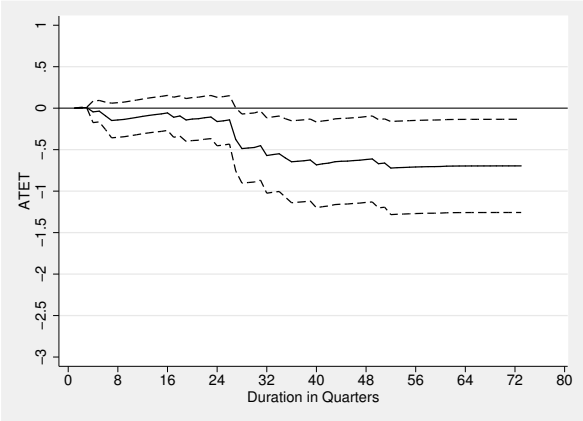
(c) ATET for Mothers-Only Trimmed Sample



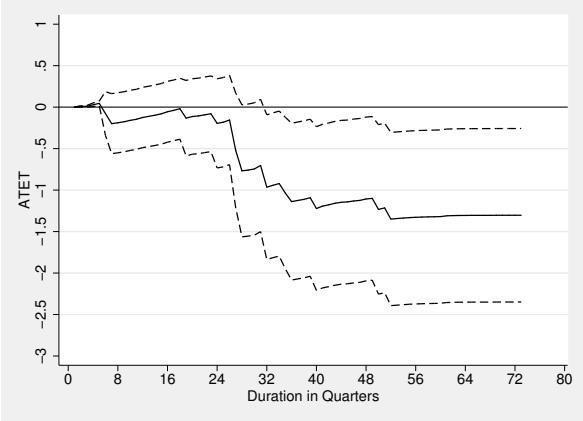
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications of the whole process.

Figure B.3: Estimated Treatment Effect for Circulatory Diseases (Trimmed Sample)

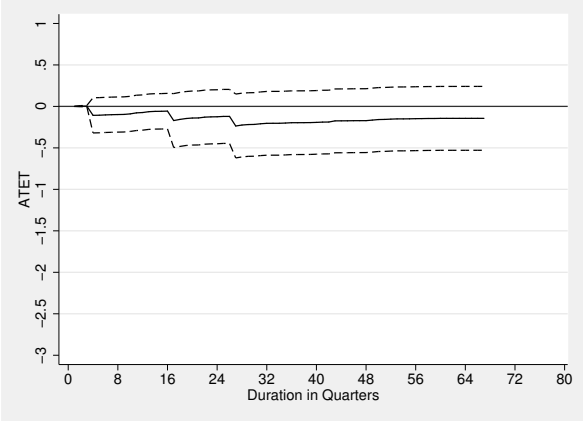
(a) ATET for the Trimmed Sample



(b) ATET for Father-Only Trimmed Sample



(c) ATET for Mothers-Only Trimmed Sample



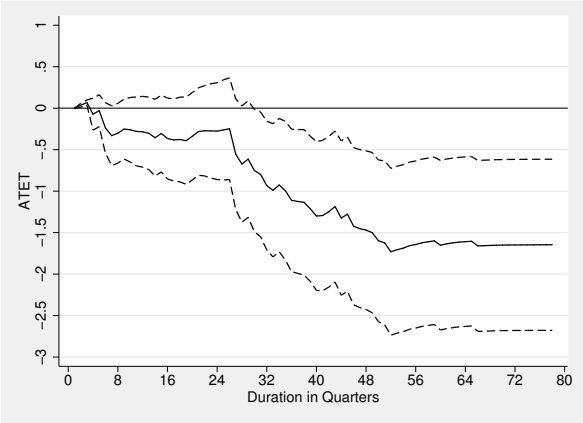
The solid line represents the ATET and was estimated using equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications of the whole process.

C Matching

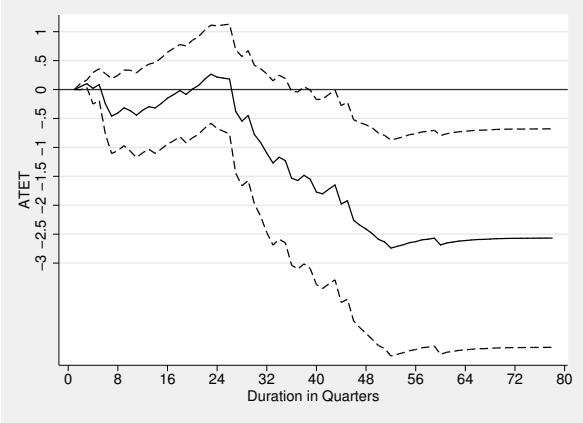
In this section, we report detailed results for our matching estimator, using kernel matching on the propensity score with an Epanechnikov kernel and a bandwidth determined by Silverman's rule (Silverman (1998)). All standard errors and confidence intervals are obtained from 500 bootstrap replications.

Figure C.1: Estimated Treatment Effect for Overall Mortality (Matching Estimates)

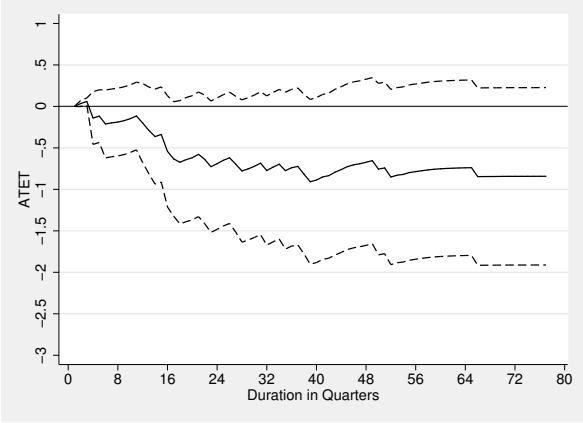
(a) ATET for the Whole Sample



(b) ATET for Father-Only Sample



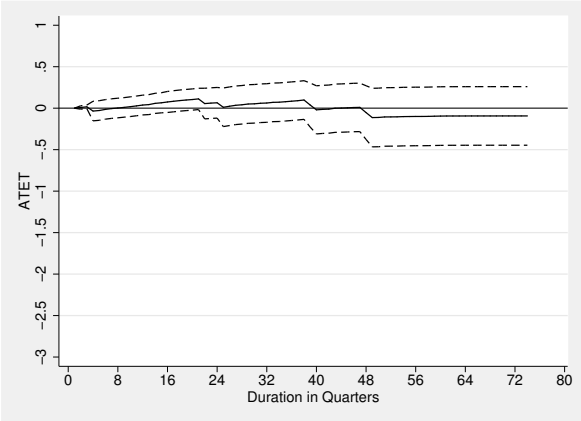
(c) ATET for Mothers-Only Sample



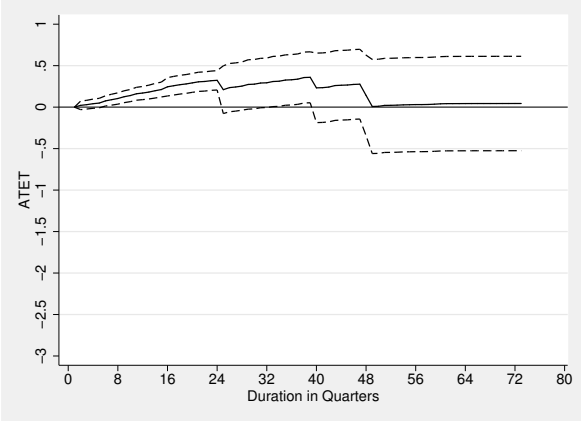
The solid line represents the ATET and was estimated using a matching estimator an equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure C.2: Estimated Treatment Effect for External Causes (Matching Estimates)

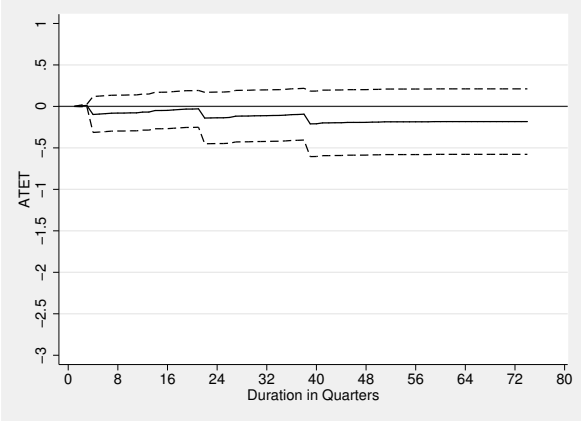
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



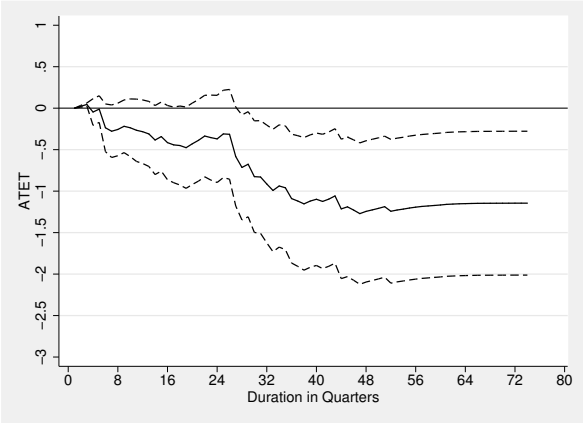
(c) ATET for the Mothers-Only Sample



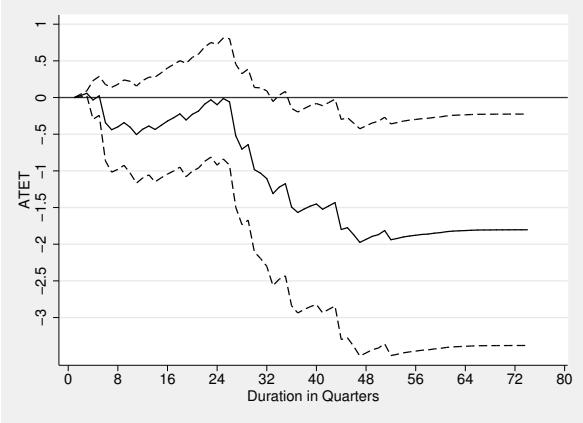
The solid line represents the ATET and was estimated using a matching estimator an equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure C.3: Estimated Treatment Effect for Natural Causes (Matching Estimates)

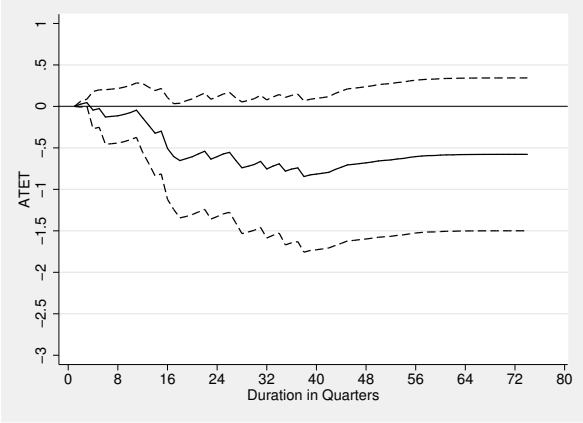
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



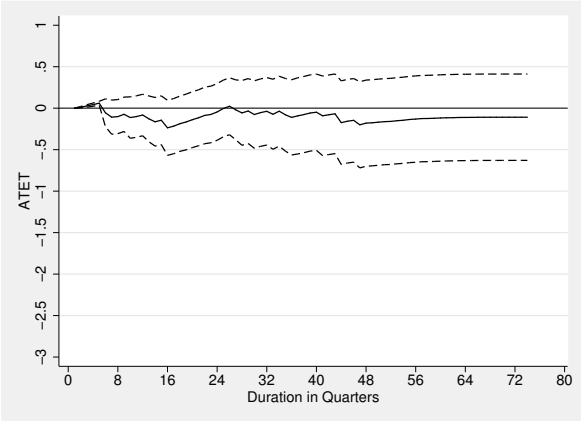
(c) ATET for the Mothers-Only Sample



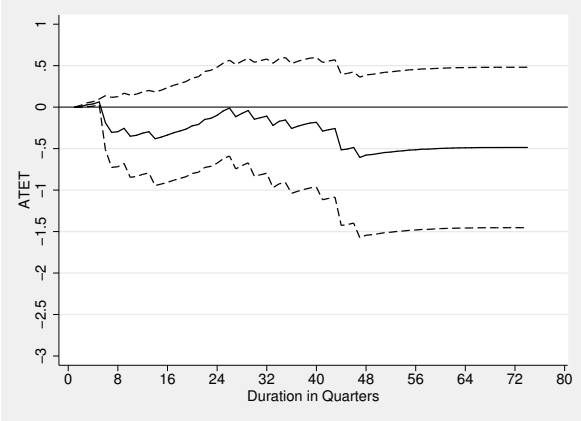
The solid line represents the ATET and was estimated using a matching estimator an equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure C.4: Estimated Treatment Effect for Cancer (Matching Estimates)

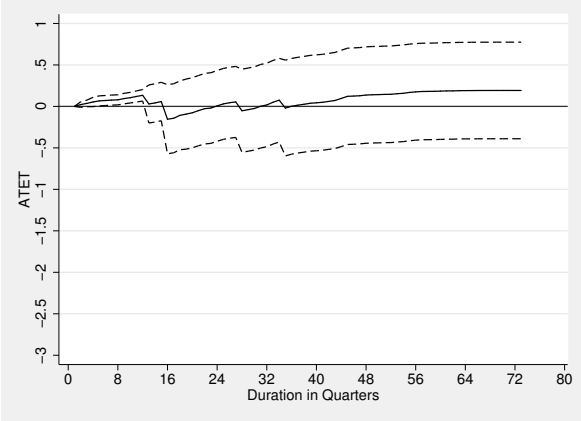
(a) ATET for the Whole Sample



(b) ATET for the Whole Sample



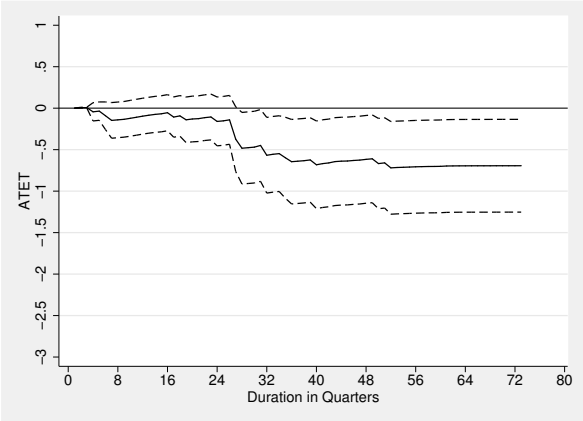
(c) ATET for the Whole Sample



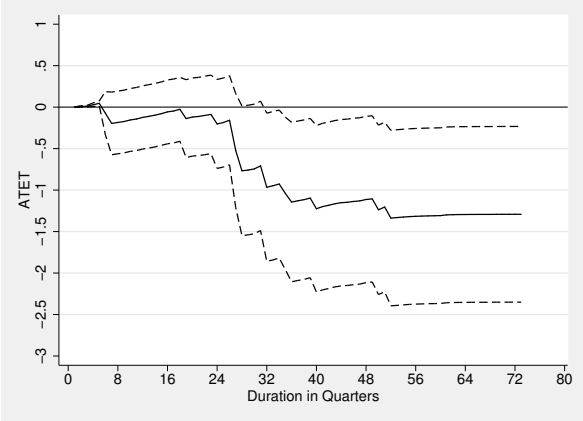
The solid line represents the ATET and was estimated using a matching estimator an equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.

Figure C.5: Estimated Treatment Effect for Circulatory Diseases (Matching Estimates)

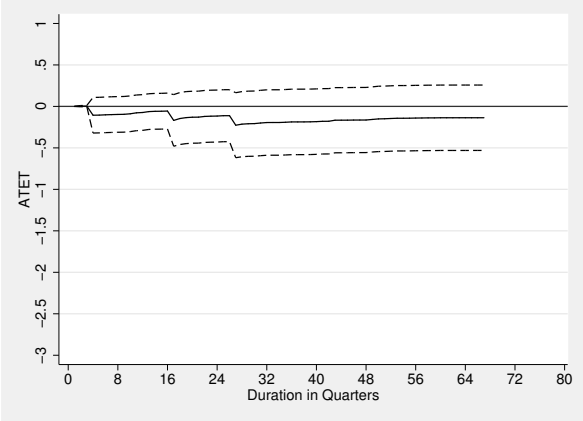
(a) ATET for the Whole Sample



(b) ATET for the Fathers-Only Sample



(c) ATET for the Mothers-Only Sample



The solid line represents the ATET and was estimated using a matching estimator an equation (4) in the text. Dashed line represent 95% confidence intervals based on 500 bootstrap replications.