Propensity Score Matching and Parental Job-Loss Effects on Parents and Children

Seminar: Econometric Impact Evaluation

Institute of Econometrics and Economic Statistics

Prof. Dr. Mark Trede

Dr. Andreas Masuhr

Stella Martin, M.Sc.

Winter term 2020/21

Chris Vennemann

c_venn01@uni-muenster.de

Economics M.Sc.

Matr.-No.: 429793

Contents

List of Figures

List of Tables

1 Introduction

Over the recent years, an extensive branch of the economics literature has developed, dedicated to understanding how job-losses affect the various dimensions of a person's life. This paper studies the effects job losses have on parents and their children. Particular fields of interest include, among others, parent's health and economic well-being, family cohesion, as well as children's career prospects and educational attainment.

Disclosing the causal effects and mechanisms of parental job-loss is important for a number of reasons. First, the nature of the consequences is typically permanent or at least long-lasting. When a parent loses its job, the effects are often present even years after the initial workplace closure.[1] This makes it especially hard to recover from job losses. Second, job losses exhibit negative spillover effects onto children. Unemployment not only damages the future employment chances of the parent but sometimes also diminishes their children's academic achievement.[2] This can be an obstacle that affects the children's academic career and may even harm their future career opportunities and income. Third, being employed is for the vast majority of people desirable for reasons that a regular income facilitates a good standard of living and that employment plays a deciding role in the societal norm, paving the way for financial, emotional, and physical well-being.[3]

Ultimately, this paper aims to review the effects from both paternal and maternal job-loss on parents, their children, and the family as a whole. This is done by providing a motivation for the use of propensity score matching in observational studies and a comprehensive summary of the study by Mörk, Sjögren, and Svaleryd (2020).[4] For parent's affected outcome variables, MSS focus on parent's unemployment, earnings, disposable income, mortality, hospitalizations, and separation status. For the children, the studied outcomes are hospitalizations, their grade point average (GPA), high school completion, and whether they are unemployed or receive social assistance by age 20-23. To capture these effects, the authors make use of several Swedish administrative databases that allow for a connection of the observed outcomes with plant closures. Using data from plant closures to capture effects from job-loss is also regularly done in other papers to remedy — at least in parts — potential selection bias among individuals who become

[1] See Mörk, Sjögren, and Svaleryd (2020); Hilger (2016); Jacobson, LaLonde, and Sullivan (1993); Stevens (1997)

[2] See Schaller and Zerpa (2019); Lindo (2011); Rege, Telle, and Votruba (2011)

[3] See Eliason and Storrie (2009a) and Eliason and Storrie (2009b); Eliason (2014)

[4] The paper is abbreviated as MSS, hereafter.

unemployed. In fact, in observational studys, selection bias is often the primary threat to identifying the causal effect. Selection bias means that individuals who experience the treatment vary from individuals who do not experience treatment in *systematic* ways — that is, other variables confound the treatment selection. After all, individuals may have self-selected into the closed workplaces based on some other underlying characteristics. In cases like these, naive estimation results are often biased and fail to uncover the true causal effect.

Propensity score matching is a prominent statistical method to solve problems of selection bias. It ensures that treatment and control groups are balanced on covariates so that the estimated effects better approximate the unbiased effects of a similar study where the treatment selection was entirely randomized. Hence, propensity score matching has considerable merits whenever randomization of treatment groups is not prevalent. This makes it a frequently used technique in fields of medicine as well as health- and labor economics. In fact, MSS use propensity score matching to ensure balance between the groups of parents who lost their job (treatment) and parents who did not (control).

Section 2 gives an overview of the literature that deals with job-loss and the effects on parents and children. In section 3, a motivation for propensity score matching and notes on implementation are presented. Section 4 encompasses all about the data, descriptive statistics, and setting of the study. Section 5 deals with the specific estimation method and section 6 summarizes the reported results by Mörk, Sjögren, and Svaleryd (2020). Section 7 gives a brief evaluation of the study and section 8 concludes the most important insights of this paper.

2 Literature Review

The reviewed study of this paper is most similar to Eliason and Storrie (2006), who find that using Swedish plant closure data from 1987, displaced worker's unemployment probability rises by an immediate 13% after the initial job-loss. Although these effects diminish rapidly, a 2-3% higher probability of unemployment was still prevalent in 1990. They also find similar effects for earnings as earnings drop initially, recover over the years, but remain at a lower level years after the initial closure. The findings of Hilger (2016) suggest an initial negative lay-off effect on after-tax US-family income of 14% and a persistent effect of 9% after five years. Lasting negative earnings effects are also reported in Jacobson, LaLonde, and Sullivan (1993) and Stevens (1997). For parent's health-related outcomes, there are closely related Swedish studies. Eliason and

Storrie (2009a) study short and long-run mortality following job-loss from plant closures and find evidence for short-run increased mortality for men. Eliason and Storrie (2009b) find increased alcohol-caused hospitalizations for men and women, and Eliason (2014) further manifests these results. Lastly, Eliason (2012) finds that only job-loss for men leads to a significant increase in divorce risk of couples.

Evidence for job-loss induced effects on children's health are reported by Schaller and Zerpa (2019) who find that job-loss of fathers has a strong negative effect on children's health in families with a low socioeconomic profile, while positive effects are presented when mothers lose their jobs. Similar effects with respect to paternal job-loss are presented in Lindo (2011). Regarding early adulthood-effects on children, Rege, Telle, and Votruba (2011) find — using Norwegian data — evidence for a negative paternal job-loss effect on children's school performance, but no significant effect from maternal job-loss. Bratberg, Nilsen, and Vaage (2008) find no effects from displaced fathers on children's earnings in later years. Supportive findings by Hilger (2016) suggest limited paternal effects on children's earnings and their college enrollment.

3 Propensity Score Matching

3.1 Motivation

Estimating unbiased treatment effects is relatively simple in a randomized controlled trial (RCT). The advantage of RCTs is the absence of selection bias because treated units do not differ systematically from untreated units. This is a very convenient property as it facilitates estimation of the treatment effect. Formally, the treatment effect of observation i is given by

$$\Delta_i = y_{1i} - y_{0i}, \tag{3.1.1}$$

where y_{1i} is the outcome variable if unit i was treated and y_{0i} the outcome if unit i was not treated. Clearly, (3.1.1) can not be calculated because every observation i can only either be treated or not treated so that either y_{1i} or y_{0i} is an unobserved counterfactual. This does not pose a problem in RCTs because, when the condition of random treatment selection is met, the *average treatment effect (ATE)*,

$$E(y_1 - y_0) = E(y_1) - E(y_0), \tag{3.1.2}$$

reliably captures the treatment effect (3.1.1). It is simply the difference between the means of the treatment and the control group. When this condition is not met, however, the ATE is a biased estimator of the treatment effect because the treatment group differs systematically from the control group and so (3.1.2) would diverge from the true treatment effect.[5]

Propensity score matching can help to remedy selection bias. The propensity score is the estimated conditional probability of receiving the treatment conditional on the observed covariates, $p(X) \equiv Pr(d = 1 \mid X)$.[6] It reduces the problem of balancing the treatment and control groups on k confounders to simply balancing on a scalar. In order to obtain unbiased estimates of the desired treatment effect from matching on the propensity scores, two assumptions must hold. Rosenbaum and Rubin (1983) refer to them as strong ignorability assumptions. If they hold, treatment assignment is said to be strongly ignorable.[7]

The *conditional independence assumption (CIA)*,

$$(y_1, y_0) \perp d \mid X, \tag{3.1.3}$$

states that outcomes and treatment assignment are conditonally independent given the observed covariates. It implies that, after controlling for the covariates X, units are assigned to the treatment as if treatment selection was random. Sometimes, (3.1.3) is referred to as the *unconfoundedness* or *selection on observables* assumption because it requires that all variables that confound the treatment selection are observed and included in X.[8]

The *common support assumption (CSA)*,

$$0 < Pr(d = 1 \mid X) < 1, \tag{3.1.4}$$

implies that for every realization of X the probability of being treated as well as the probability of not being treated strictly lie in the unit interval. Hence, it ensures that there is common support between the treatment and control group in order to find good matches.[9] As both assumptions are detrimental to obtaining the desired treatment effects, it is important to evaluate whether they hold after the matching on propensity scores was conducted.

Besides the ATE, experimental studies often aim to estimate the *average treatment effect on*

[5] Cf. Zhao (2004) p.92
[6] Cf. Rosenbaum and Rubin (1983) pp.42-43
[7] Cf. Rosenbaum and Rubin (1983) p.43
[8] Cf. Abadie and Imbens (2016) p.783
[9] Cf. Abadie and Imbens (2016) ibid.

the treated (ATET),

$$E(y_1 - y_0 \mid d = 1) = E(y_1 \mid d = 1) - E(y_0 \mid d = 1). \tag{3.1.5}$$

The ATET captures the difference between the average outcome of those who were treated, $E(y_1 \mid d = 1)$, and the average outcome of them had they not been treated, $E(y_0 \mid d = 1)$. Again, this poses the problem of estimation because $E(y_0 \mid d = 1)$ is a counterfactual. One can, however, observe the average difference between treated and control units given by

$$E(y_1 \mid d = 1) - E(y_0 \mid d = 0) = E(y_1 - y_0 \mid d = 1) + \big(E(y_0 \mid d = 1) - E(y_0 \mid d = 0)\big). \tag{3.1.6}$$

The observable difference is equal to the ATET plus a selection bias term. Equation (3.1.6) shows that simply estimating the left hand side to estimate the ATET is spurious whenever treatment assignment suffers from selection bias. However, applying the law of iterated expectations and the CIA to (3.1.5) yields the unbiased estimator of the ATET,

$$\tau_{ATET} = E\big[E(y_1 \mid p(X), d = 1) - E(y_0 \mid p(X), d = 0) \mid d = 1\big].^{10,11} \tag{3.1.7}$$

Equation (3.1.7) is a central result due to Rosenbaum and Rubin (1983). Since it does not contain counterfactual outcomes, it can be easily estimated from the data. Furthermore, matching on the scalar propensity score has considerable merits over balancing on a possibly large number of covariates.

3.2 Practical Implementation

The following paragraphs briefly summarize a structured procedure when implementing propensity score matching. Generally, this procedure can be separated into four steps:

3.2.1 Estimation of the Propensity Scores

This is usually done using a logistic regression model with the binary treatment variable d as the dependent variable and the covariates X as predictors. Since the fitted values are estimates of the conditional probability that a unit is assigned to the treatment (given the covariate values

[10] See A.2 for a derivation.

[11] Cf. Angrist and Pischke (2008) p.52; Abadie and Imbens (2016) p.784; Zhao (2004) p.92

$X = x$), the estimated propensity scores are bound to lie in the unit interval. Regarding the choice of which covariates to use as as predictors of the treatment, the functional form should be such that it most appropriately mimics the unknown selection process, be it in the sense of an active selection like employers evaluating potential candidates for a job based on some criteria (e.g. education level, years of education) or employees self-selecting into the workplaces based on employer-criteria (e.g. pay, benefits, firmsize). On a more general note, any covariate that affects both the treatment and the outcome variable should be included. It is often preferable to include an insignificant variable than to be at risk of omitting a potentially significant one.[12] It is important to note that only pre-treatment variables should be included as predictors of the treatment.[13,14]

3.2.2 Matching

Matching describes the procedure of finding the most similar pair of treated and untreated observations. There are a handful of possible ways to perform matching. Nearest Neighbour matching is a commonly used method that matches each treatment unit to the closest (1:1) or the k closest (1:k) control units based on the absolute distance of their propensity scores. Methods also vary in terms of replacement such that a control unit may be matched to more than just one treated unit. The advantage of replacement becomes apparent when there are only few control, but many treated units with a propensity score in a given range. Not replacing a control unit either causes poor matches for the other treated units in that range because the few control units are already matched to other treated ones or may even prevent matches completely if they are not similar enough.[15] Closely related are caliper and radius matching. Caliper matching is like nearest neighbour matching but imposes a restriction on the maximum allowed distance of propensity scores. Units whose propensity scores fall outside the allowed distance are not matched. Radius matching is the same as caliper matching but allows a treated unit to be matched to several untreated units inside the caliper.[16] Since the caliper must be specified by the researcher, this poses a problem of parameter tuning to obtain good matches.

[12] Cf. Stuart (2010) p.5
[13] Cf. Caliendo and Kopeinig (2008) p.7
[14] Cf. Rosenbaum and Rubin (1983) p.42
[15] Cf. Caliendo and Kopeinig (2008) pp.9-10
[16] Cf. Pan and Bai (2018) p.7

3.2.3 Evaluating the Matching Quality

A combination of statistical and visual inspection typically help to access whether the matching has achieved covariate balance. Researchers frequently use standarized biases for each covariate in this context.[17,18] A standardized bias below three to five percent after matching is considered low enough to ensure balance of a particular covariate.[19] Another popular method to check covariate balance are sample t-tests of equal treatment and control means.[20] Some other statistical methods are available, however standarized biases and t-tests are typically sufficient to indicate covariate balance or imbalance.[21] Additionally, visual inspection of propensity score distributions of treated and control groups complements the matching quality evaluation in terms of finding common support. Evaluation of common support rarely goes beyond visual inspection, however Caliendo and Kopeinig (2008) argue for more precise accessment of common support based on formal metrics.[22]

3.2.4 Outcome Analysis

If the matching has achieved balanced treatment and control groups, the desired treatment effects can be estimated by comparing the means of the treatment and control groups. Whenever control units are matched to several treated units, the control units need to be adjusted by weights. For matching algorithms that match one-to-one estimation of the treatment effect can also be done by ordinary least squares regression on the matched data sample with the outcome as the dependent variable and the treatment as the regressor. If some covariates remain unbalanced after the matching, adding the unbalanced covariates to the right-hand side of the regression model is a useful way to adjust for the remaining imbalance. In either model, the estimated coefficient of the treatment variable is the desired estimate of the ATET. For matching algorithms that match one-to-many, a weighted least squares regression model should be fit to account for the imbalance of treated and control units in a match (radius matching) or the frequency of control units (replacement).[23]

[17] See equation (A.1.1) in Appendix A.
[18] Cf. Rosenbaum and Rubin (1985) p.34 Table 1
[19] Cf. Caliendo and Kopeinig (2008) p.15
[20] Cf. Pan and Bai (2015) pp.9-10; Caliendo and Kopeinig (2008) p.16
[21] See Caliendo and Kopeinig (2008) p.16 for related methods or Pan and Bai (2018) p.329; Pan and Bai (2015) p.9; Cochran and Rubin (1973) for percent bias reduction.
[22] Cf. Caliendo and Kopeinig (2008) pp.12-14
[23] Cf. Pan and Bai (2015) p.10; Stuart (2010) pp.12-13

4 Data

The following sections deal with the study by Mörk, Sjögren, and Svaleryd (2020). The first few sections encompass all about the data and setting of the study, followed by a review of the estimation method and empirical results.

4.1 Data Sample

The data is obtained from administrative Swedish data that allows for a connection of closed plants to workers and their children. Analyzed are all plant closures in the years from $t = 1995 - 2000$. For every year t, all workers who work at the same workplace in t as in $t-3$ and $t-2$ and who have children of any age between 2 and 18 in year t, are selected. Workers whose workplaces close down in year t are assigned the treatment. From the workers whose workplaces did not close down, MSS draw a 25% random sample to construct the control group. The data panel allows to observe the outcomes of the parents and their children five years prior to and up to ten year after the plant closure.[24]

The studied outcome variables for the parents are as follows. `Unemployed` is a binary variable that equals 1 if the parent is unemployed at least once in a year. `Earnings` measures the earnings from employment and self-employment, while `disposable income` captures all income minus taxes. The variable `separated` indicates whether parents live in separated households. `Mortality` captures instances where a parent deceased and `hospitalization` indicates whether a parent was hospitalized for any cause, whereas `alocohol` and `mental` indicate specific hospitalization causes. Child outcomes capture a range of health and education-related variables. `Hospitalization` is similar to the parent's but excludes pregnancies or child birth. Hospitalizations are further devided into `avoidable`, capturing child hospitalizations due to any causes that were preventable[25], and `mental and behavior`, which captures conditions due to mental illness, alocohol abuse, self-harm or external abuse. `GPA` measures childrens standardized grade point average in the final year of compulsory school (at age 16). `High school` indicates whether children have completed high school by age 20. Lastly, `unemployed` and `SA` indicate whether children suffer from unemployment or live in a household which receives social assistance at ages

[24] Cf. Mörk, Sjögren, and Svaleryd (2020) pp.3-4

[25] E.g. causes that can be prevented through vaccination, antibiotics, lifestlye changes, pharmaceutical treatment or other medical intervention.

20 to 23.[26]

4.2 Study Setting

Since the sample is drawn from Swedish administrative databases, the setting of the study is special in a number of ways. Frist, the treatment assignment is contructed using information from plant closures. As initially stated, a study where treatment is constructed using plant closure data may yield different results than a study where the treatment was constructed using different definitions of job-loss. Plant closures likely capture job-loss in a more exogenous manner than do job firings, for instance.[27] Second, Swedish families are generally well equipped against unemployment. Unemployed parents, who were with an employment insurance fund and took part in requisite labor market programs, were granted substanial insurance payouts with earnings replacement rates of 75-80 percent in the studied time frame. Third, the human capital formation of Swedish children is rather independent of the parent's income or employment status because of extensive subsidized childcare. Financial support range from free school meals to free university tuition, subsidized student loans, and either free or subsidized drug prescription. Forth, Sweden has a dual-earner norm. Unlike in other countries, Swedish mothers and fathers are incentivized to both provide for the family's income due to individual taxation, high marginal tax rates, sick or parental leave benefits, and pensions.[28]

4.3 Descriptive Statistics

This section gives an overview of the treatment and control group means before matching. Covariate means are measured in $t-2$ and divided into data for mothers and data for fathers. Table 1 summarizes pre-matching comparisons of each covariate. Regarding covariates for the children, the data shows systematic differences for children's age and GPAs between children of parents whose workplaces close and whose workplaces do not. On average, children of treated parents are younger and have lower GPAs. For the characteristics of mothers, all but one listed covariates indicate a systematic difference: Treated mothers are younger, less likely to have graduated university, more likely to be foreign-born, as well as more likely to live separated from the fathers. Furthermore, mothers differ (negatively) in terms of tenure, unemployment, earnings, and hospitalizations. The father's characteristics are similar to those of mothers, with the ex-

[26] Cf. Mörk, Sjögren, and Svaleryd (2020) p.4
[27] Cf. Mörk, Sjögren, and Svaleryd (2020) p.2 footnote 6; Eliason and Storrie (2006) pp.845-846
[28] Cf. Mörk, Sjögren, and Svaleryd (2020) p.3

ception that treated fathers are not more frequently hospitalized than untreated mothers. In terms of firm-level characteristics, closing workplaces belong to larger and newer firms and their employees had children with lower GPAs.[29] Hence, there is evidence for a systematic difference between parents at closing and non-closing workplaces which justifies the use of matching on propensity scores to control for selection bias.

5 Estimation Method

Estimation of the propensity score is done using logistic regression with a broad range of possible confounding variables.[30] As specified in an earlier section, the included variables are measured pre-treatment whenever it was feasible due to data availability constraints. Matching is done using nearest neighbor matching with replacement of untreated units.[31] After matching, the covariate means between treated and untreated units are significantly better balanced, as shown in Table 2. Only firm-level GPA of children in the paternal data remained unbalanced. Figure 5 additionally shows that the standardized biases of covariates diminished considerably. Furthermore, an inspection of the distributions of treatment and control groups in Figure 4 indicates that matching has achieved common support.[32] In both the maternal and paternal samples, the treated and control group distributions are left-skewed. However, the skewness is slightly more pronounced for the control group. Overall, MSS report that there is (sufficient) common support.[33] MSS use the ATET estimator from Abadie and Imbens (2016) on the matched sample.[34]

6 Results

6.1 Effects on the Parents

Figure 1 displays the estimated effects on parent's probability of unemployment. There are no significant differences between treated and untreated parents up to two years before the plant closure. Once the workplace closes down in year 0, mothers have a 7 percent points higher probability of being unemployed and fathers have a 8.5 higher probability. The treatment effect appears to diminish gradually over time, but the probability is still around 1 percent point

[29] Cf. Mörk, Sjögren, and Svaleryd (2020) p.4
[30] See Table 3
[31] Cf. Mörk, Sjögren, and Svaleryd (2020) p.5
[32] Propensity scores are modeled as the conditional probability of *not* receiving the treatment.
[33] Cf. Mörk, Sjögren, and Svaleryd (2020) p.5
[34] Cf. Abadie and Imbens (2016) p.785

higher for treated mothers and fathers even ten years after the treatment. Earnings show a similar pattern. Displaced mothers (fathers) have 5% (6%) lower earnings one year after the lay-off. Both earnings recover slowly in the years after, however fathers earnings recover more slowly. The treatment effect on disposable income is overall less drastic. While there appears to be a slight downward trend after the treatment in year 0 for parents overall, mother's disposable income is most reduced, by around -4%, five years after. The father's disposable income effect flucuates around -2% to -4.5% in the years after.[35]

Effects for parent's separation status and mortality are shown in Figure 2. The estimated treatment effect on parent's separation status is only significant for the maternal sample. Treated mothers live 2.2% more often separated from their spouses 2-5 years after the workplace closes down. After that, there is no significant effect. Contrary to separation status, mortality treatment effects are only significant for fathers 6-10 years after treatment. On average, the number of deceased fathers who lost their jobs is 1.2-1.9 per 1000 higher than fathers who kept their jobs.[36] Table 5 shows effects on parent's hospitalizations. Hospitalization effects are only significant in terms of alcohol-related hospitalizations for mothers. Displaced mothers are 13% more likely to be hospitalized due to alcohol causes in any of the ten years after job-loss. No significant effects are found for overall or mental health hospitalizations.[37]

6.2 Effects on the Children

No evidence can be found for treatment effects on children's health variables (hospitalizations, avoidable, mental and behavior). The estimates for hospitalization causes, overall and specific, 0-10 years after the treatment are all insignificant and also hint towards effects that are small in magnitude. Furtermore, any possible pre-treatment effects can be ruled out since estimates for hospitalizations 3-5 years before treatment are also not significant, as shown in Table 6.[38]

Figure 3 and Table 7 summarize the estimated effects on children's school performance, unemployment, and social assistance. Estimates for effects on children's GPA suggest that only maternal job-loss has significant effects. Since GPA is only measured once at age 16, the estimates do not correspond to the typical effects for every year, but instead are such that they capture

[35] Cf. Mörk, Sjögren, and Svaleryd (2020) p.6

[36] Using (cumulative effect) results from regression tables. See Table 4. In the figures, scales are usually in percent. Mortality effects are hard to interpret in percent becaues of the very low comparison value of mortality.

[37] Cf. Mörk, Sjögren, and Svaleryd (2020) p.6

[38] Cf. Mörk, Sjögren, and Svaleryd (2020) pp.6-8

the effect of treatment on children who were 6-21 years old when the parent lost his or her job. Estimates are only significant for children whose mothers were displaced 2-4 years before the children's graduation and suggest that their GPA is on average 5-7% of a standard deviation lower than children of non-displaced mothers. Lastly, the effects on highschool completion by age 20, social assistance at ages 20-23, and unemployment at ages 20-23 are reported. There is no evidence for a significant effect on highschool completion for both maternal and paternal job-loss but there is a significant increase in the probability of receiving social assistance and being unemployed at ages 20-23 for maternal job-loss. These effects are small however as the increase, compared to the controls, is 4.4% for social assistance and 1.7% for unemployment.[39]

7 Evaluation

Only little can be said for the internal validity of the study because there is no real benchmark to compare the estimation results to. Qualitative evaluations can therefore only be based on whether the two assumptions of conditional independence and common support are fulfilled and on closeness to results of earlier studies with similar settings.

The CIA may not hold if not all confounders are included in the estimation of the propensity score. Since MSS include many variables, which capture a wide range of characteristics at the worker- and firm-level, it is unlikely that confounding variables are unobserved or not included, but it cannot be ruled out. Furthermore, as Figure 5 indicates, the covariates have sufficiently low standardized biases in the matched sample. A small inconsistency stands out, however. MSS mention that only firm-level GPA has statistically significant differences in means after matching, but Table 2 also shows low p-values for other covariates (e.g. unemployed, education).[40] Lastly, Figure 4 shows that matching has achieved common support.

The estimated effects for parent's probability of unemployment (7%-8.5%) are close to those obtained by Rege, Telle, and Votruba (2011), who find a 5% increase for Norwegian parents. On the other hand, Eliason and Storrie (2006) report a 13% increase for Swedish workers. Earnings and disposable income effects are broadly similar to other related studies in nordic settings but different from studies with dissimilar settings. Hilger (2016), for example, reports stronger effects on after-tax income. The finding that displacement of fathers has a negative effect on

[39] Cf. Mörk, Sjögren, and Svaleryd (2020) pp.8-9

[40] This inconsistency may be explained by the fact that the T-statistic takes the sample size into account, whereas standardized biases do not. Also, the values in the differences column in Table 2 are likely rounded so that for small values, differences of 0 are displayed.

their mortality is in line with results of Eliason and Storrie (2009a). Furthermore, MSS report a higher chance of alcohol-related hospitalizations for displaced mothers, whereas Eliason and Storrie (2009b) find such effects for both mothers and fathers. Regarding the effects on children's health outcomes, no significant results were found, which stands in contrast to the negative effects of earlier studies. There is also no indication of a positive effect of displaced mothers on children's health as occasionally reported by related works. Effects that are inconsistent with the literature are also found for children's GPA. MSS find a small but negative effect for children with displaced mothers (5-7% of a standard deviation 2-4 years before graduation at 16) but no significant effect for children with displaced fathers, whereas Rege, Telle, and Votruba (2011) obtain results of similar magnitude and age (6% of a standard deviation in early teenage years) for displaced fathers instead of mothers. Lastly, the estimated insiginifcant effects of paternal job-loss on children's unemployment and social assistance at ages 20-23 are also found by other studies.[41]

External validity is limited whenever there are differences between the studied population or the sample drawn thereof and the population to which the results ought to be generalized. Unemployment insurance, unconditional childcare, the dual-earner norm, and the measurement of job-loss (plants closures) were identified as the main characteristics of the studied setting. Therefore, results may not be generalized to other populations where these characteristics are not prevalent and may therefore likely explain both the similar results of other nordic studies and the diverging results from dissimilar studies.[42]

8 Conclusion

Observational studies often suffer from selection bias — units that receive the treatment differ from untreated units in systematic ways. Therefore, researchers must be cautious in accessing treatment effects on a certain subpopulation of observed units (ATE, ATET). Simply averaging over observable outcome differences between treated and untreated units more often than not leads to spurious estimations of treatment effects because of selection bias. Such problems can be overcome by propensity score matching. By matching on the estimated propensity score, selection bias vanishes, and the estimation of the desired treatment effect no longer suffers from problems that are due to unobservable outcomes.

In the study by MSS, propensity score matching is used to eliminate selection bias between

[41] Cf. Mörk, Sjögren, and Svaleryd (2020) pp.9-10
[42] Cf. Eliason and Storrie (2006) p.832

parents whose workplace closed down and parents whose workplace did not. Nearest neighbor matching with replacement has improved the covariate balance considerably. Only one variable displayed a too high residual imbalance afterward.

While the magnitudes of estimated effects are broadly in line with what other nordic studies find, there are some dissimilarities with respect to which parent's job-loss induces a significant treatment effect. Parent's unemployment and earnings are negatively affected shortly after treatment, recover slowly over the years, but remain deteriorated even after a couple of years. Disposable income seems to be less strongly affected. Swedish unemployment insurance likely absorbs some of the negative consequences of displacement and shields families from economic misfortune. Contrary to other findings, mothers whose workplace closes down live more often separated from the fathers. Furthermore, displaced fathers face higher mortality than do non-displaced fathers. On the other hand, displaced mothers are more frequently hospitalized for alcohol-related conditions. No significant treatment effects are found for children's health outcomes, which likely reflect the generously subsidized childcare in Sweden. In terms of children's school performance, only maternal job-loss appears to negatively affect children's GPA. The estimates amount to only small effects, however. The same is true for effects on children's unemployment and social assistance at ages 20-23. Again, this result may be explained by the extensive Swedish childcare system. While job-losses have considerable negative effects on Swedish parents, no or only negligible effects are found for their children.[43]

[43] Cf. Mörk, Sjögren, and Svaleryd (2020) pp.10-11

References

Abadie, Alberto and Guido W Imbens (2016). "Matching on the estimated propensity score". In: *Econometrica* 84.2, pp. 781–807.

Angrist, Joshua D and Jörn-Steffen Pischke (2008). *Mostly harmless econometrics: An empiricist's companion*. Princeton university press.

Bratberg, Espen, Øivind Anti Nilsen, and Kjell Vaage (2008). "Job losses and child outcomes". In: *Labour Economics* 15.4, pp. 591–603.

Caliendo, Marco and Sabine Kopeinig (2008). "Some practical guidance for the implementation of propensity score matching". In: *Journal of economic surveys* 22.1, pp. 31–72.

Cochran, William G and Donald B Rubin (1973). "Controlling bias in observational studies: A review". In: *Sankhyā: The Indian Journal of Statistics, Series A*, pp. 417–446.

Eliason, Marcus (2012). "Lost jobs, broken marriages". In: *Journal of Population Economics* 25.4, pp. 1365–1397.

— (2014). "Alcohol-related morbidity and mortality following involuntary job loss: Evidence from Swedish register data". In: *Journal of studies on alcohol and drugs* 75.1, pp. 35–46.

Eliason, Marcus and Donald Storrie (2006). "Lasting or latent scars? Swedish evidence on the long-term effects of job displacement". In: *Journal of Labor Economics* 24.4, pp. 831–856.

— (2009a). "Does job loss shorten life?" In: *Journal of Human Resources* 44.2, pp. 277–302.

— (2009b). "Job loss is bad for your health–Swedish evidence on cause-specific hospitalization following involuntary job loss". In: *Social science & medicine* 68.8, pp. 1396–1406.

Hilger, Nathaniel G (2016). "Parental job loss and children's long-term outcomes: evidence from 7 million fathers' layoffs". In: *American Economic Journal: Applied Economics* 8.3, pp. 247–83.

Jacobson, Louis S, Robert J LaLonde, and Daniel G Sullivan (1993). "Earnings losses of displaced workers". In: *The American economic review*, pp. 685–709.

Lindo, Jason M (2011). "Parental job loss and infant health". In: *Journal of health economics* 30.5, pp. 869–879.

Mörk, Eva, Anna Sjögren, and Helena Svaleryd (2020). "Consequences of parental job loss on the family environment and on human capital formation-Evidence from workplace closures". In: *Labour Economics* 67, pp. 1–11.

Pan, Wei and Haiyan Bai (2015). *Propensity score analysis*. Guilford Publications.

Pan, Wei and Haiyan Bai (2018). "Propensity score methods for causal inference: an overview". In: *Behaviormetrika* 45.2, pp. 317–334.

Rege, Mari, Kjetil Telle, and Mark Votruba (2011). "Parental job loss and children's school performance". In: *The Review of Economic Studies* 78.4, pp. 1462–1489.

Rosenbaum, Paul R and Donald B Rubin (1983). "The central role of the propensity score in observational studies for causal effects". In: *Biometrika* 70.1, pp. 41–55.

— (1985). "Constructing a control group using multivariate matched sampling methods that incorporate the propensity score". In: *The American Statistician* 39.1, pp. 33–38.

Schaller, Jessamyn and Mariana Zerpa (2019). "Short-run effects of parental job loss on child health". In: *American Journal of Health Economics* 5.1, pp. 8–41.

Stevens, Ann Huff (1997). "Persistent effects of job displacement: The importance of multiple job losses". In: *Journal of Labor Economics* 15.1, Part 1, pp. 165–188.

Stuart, Elizabeth A (2010). "Matching methods for causal inference: A review and a look forward". In: *Statistical science: a review journal of the Institute of Mathematical Statistics* 25.1, pp. 1–21.

Zhao, Zhong (2004). "Using matching to estimate treatment effects: Data requirements, matching metrics, and Monte Carlo evidence". In: *Review of economics and statistics* 86.1, pp. 91–107.

Appendices

A Equations and Formulas

A.1 Standardized Bias

The standardized percentage bias for covariate k is given by

$$SB_k = \frac{\mu_{1k} - \mu_{0k}}{\sqrt{\frac{\sigma_{1k}^2 + \sigma_{0k}^2}{2}}} \times 100, \tag{A.1.1}$$

where μ_{1k} and μ_{0k} are the means of the k-th covariate in the treatment and control group and σ_{1k}^2 and σ_{0k}^2 are the respective variances.

A.2 Unbiased Estimator for the ATET

Applying the law of iterated expectations to (3.1.5) yields

$$E\big[E(y_1 \mid X, d = 1) - E(y_0 \mid X, d = 1) \mid d = 1\big]. \tag{A.2.1}$$

Then, given that the CIA holds and

$$(y_1, y_0) \perp d \mid X \implies E(y_0 \mid X, d = 0) = E(y_0 \mid X, d = 1), \tag{A.2.2}$$

(A.2.1) is equal to

$$E\big[E(y_1 \mid X, d = 1) - E(y_0 \mid X, d = 0) \mid d = 1\big]. \tag{A.2.3}$$

Using the propensity score, $p(X) = Pr(d = 1 \mid X)$, as a balancing score, Rosenbaum and Rubin (1983) showed that the conditional independence and common support assumption imply

$$(y_1, y_0) \perp d \mid p(X) \tag{A.2.4}$$

$$0 < p(X) < 1. \tag{A.2.5}$$

With analogous steps as above, this yields the unbiased estimator of the ATET

$$\tau_{ATET} = E\big[E(y_1 \mid p(X), d = 1) - E(y_0 \mid p(X), d = 0) \mid d = 1\big]. \tag{A.2.6}$$

B Tables and Figures

All tables and figures are taken from Mörk, Sjögren, and Svaleryd (2020).

Table 1: Mean comparisons of treatment and control groups before matching

	Maternal sample				Paternal sample			
	Surviving workplace	Closing workplace	Difference	(p-value)	Surviving workplace	Closing workplace	Difference	(p-value)
Child characteristics								
Girl	0.48	0.49	-0.00	0.11	0.49	0.49	-0.00	0.36
Age	8.72	8.55	0.17	0.00	7.76	7.71	0.05	0.00
Hospitalization*	44.75	45.22	-0.46	0.62	52.23	51.49	0.74	0.37
Mental and behaviour*	1.51	1.87	-0.36	0.04	1.59	1.43	0.16	0.28
Avoidable*	6.06	6.54	-0.48	0.17	8.79	8.30	0.50	0.15
GPA at age 16 **	0.13	0.03	0.10	0.00	0.11	0.05	0.06	0.00
Parental characteristics								
Age	37.9	37.5	0.37	0.00	39.2	39.1	0.10	0.00
Compulsory education	0.12	0.16	-0.04	0.00	0.19	0.20	-0.01	0.00
Secondary education	0.51	0.54	-0.03	0.00	0.48	0.49	-0.00	0.03
University education	0.37	0.31	0.07	0.00	0.33	0.32	0.01	0.00
Swedish born	0.90	0.88	0.02	0.00	0.90	0.89	0.01	0.00
Separated	0.18	0.22	-0.03	0.00	0.16	0.17	-0.01	0.00
Tenure***	4.73	4.25	0.47	0.00	4.99	4.48	0.51	0.00
Unemployed ****	0.06	0.09	-0.02	0.00	0.06	0.09	-0.03	0.00
Disposable income	364,172	362,102	2,070	0.12	363,634	365,354	-1,720	0.12
Earnings	186,363	185,262	1,101	0.00	304,215	300,570	3,645	0.00
Hospitalization*	50.50	54.44	-3.94	0.00	44.27	43.89	0.38	0.62
Alcohol*	0.60	1.09	-0.49	0.00	1.55	1.49	0.06	0.69
Mental*	37.89	37.52	0.37	0.00	2.76	2.60	0.16	0.40
Firm characteristics								
Firm size	8,763	10,810	-2,046	0.00	5,012	6,276	-1,264	0.00
New firm	0.15	0.26	-0.11	0.00	0.27	0.38	-0.11	0.00
GPA std. at firm *****	0.09	0.02	0.08	0.00	0.05	0.00	0.05	0.00
No obs.	950,616	53,032			1,102,675	77,738		
No obs. GPA age 16	60,742	3,272			56,405	3,915		

Note: Base years 1995–2000. All variables measured in $t-2$. For surviving workplaces, we have drawn a 25% random sample. * individuals per 1,000 hospitalized at least once during the year; ** for children who were 16 years old in $t-2$; *** censored at 7 years; **** being registered at the PES as unemployed or in an active labour market program at least one day during the year; ***** mean for workers' children graduating before $t-2$.

Table 2: Mean comparisons of treatment and control groups after matching

	Maternal sample				Paternal sample			
	Surviving workplace	Closing workplace	Difference	(p-value)	Surviving workplace	Closing workplace	Difference	(p-value)
	Child characteristics							
Girl	0.49	0.49	-0.00	(0.65)	0.49	0.49	0.00	(0.28)
Age	8.56	8.55	0.01	(0.70)	7.68	7.71	-0.03	(0.21)
Hospitalization*	46.05	45.22	0.83	(0.52)	51.31	51.49	-0.18	(0.87)
Mental and behaviour*	2.04	1.87	0.17	(0.54)	1.30	1.43	-0.13	(0.50)
Avoidable*	6.43	6.54	-0.11	(0.82)	9.18	8.30	0.88	(0.07)
GPA at age 16 **	0.05	0.03	0.01	(0.59)	0.05	0.05	0.01	(0.72)
	Parental characteristics							
Age	40.13	40.15	0.00	(0.92)	39.02	39.06	-0.04	(0.28)
Compulsory education	0.22	0.22	-0.00	(0.48)	0.20	0.20	-0.00	(0.89)
Secondary education	0.49	0.48	0.00	(0.55)	0.49	0.49	0.01	(0.05)
University education	0.30	0.30	-0.00	(0.93)	0.31	0.32	-0.00	(0.05)
Swedish born	0.88	0.88	0.00	(0.23)	0.89	0.89	0.00	(0.15)
Separated	0.22	0.22	0.00	(0.45)	0.17	0.17	-0.00	(0.30)
Tenure***	4.92	4.90	-0.02	(0.19)	4.48	4.48	0.00	(0.96)
Unemployed ****	0.16	0.16	-0.00	(0.34)	0.09	0.09	-0.00	(0.00)
Disposable income	359,046	356,801	1,603	(0.24)	367,283	365,354	1,930	(0.25)
Earnings	263,086	261,008	811	(0.12)	302,218	300,570	1,648	(0.09)
Hospitalization*	52.67	51.74	0.74	(0.60)	44.43	43.89	0.54	(0.61)
Alcohol*	3.73	3.30	0.00	(1.00)	1.31	1.49	-0.18	(0.35)
Mental*	6.18	5.47	0.19	(0.59)	2.83	2.60	0.23	(0.39)
	Firm characteristics							
Firm size	5,450	5,840	41.5	(0.70)	6,237.33	6,276.37	-39.04	(0.55)
New firm	0.31	0.30	-0.00	(0.15)	0.38	0.38	-0.00	(0.22)
GPA std. at firm ****	0.04	0.05	0.01	(0.07)	0.02	0.00	0.02	(0.00)
No obs.	49,739	53,032			72,363	77,738		
No obs. GPA age 16	3,148	3,272			3,675	3,915		

Note: Base years 1995–2000. All variables measured in t-2. * individuals per 1,000 hospitalized at least once during the year; ** for children that were 16 years old in t-2; *** censored at 7 years; **** being registered at the PES as unemployed or in an active labour market program at least one day during the year; ***** mean for workers' children graduating before t-2.

Table 3: Covariates for propensity score estimation

Variable	***Definition***
Age t-2 (child, worker)	Years of age
Age^2 t-2 (child, worker)	Years of age, squared
Girl (child)	=1 if girl
Hospitalization t-2 and t-3 (worker), t-2 or t-3 (other parent) and t-2 (child)	=1,000 if admitted to a hospital that year
Hospitalization for diagnoses indicating mental health problems t-2 or t-3 (worker)	=1,000 if admitted to a hospital with a diagnosis code indicating mental health problems according to Table B 1.
Hospitalization for diagnoses indicating alcohol abuse t-2 or t-3 (worker)	=1,000 if admitted to a hospital with a diagnosis code indicating alcohol-related disease according to Table B 1.
Hospitalization for diagnoses indicating alcohol abuse or mental health problems t-2 or t-3 (other parent)	=1,000 if admitted to a hospital with a diagnosis code indicating alcohol-related disease or mental health problems according to Table B 1.
Hospitalization for diagnoses indicating mental health and behaviour problems in t-2 (child)	=1,000 if admitted to a hospital with a diagnosis code indicating mental health problems, self-destructive behaviour, alcohol-related conditions, i.e., disease or abuse according to Table B 1.
Separated (child) t-2	Dummy indicating that the biological parents do not live together
Years in Sweden t-2 (worker, other parent)	Dummy indicating time living in Sweden (8) 0: born in Sweden 1: time in Sweden < 6 years 2: 5 < time in Sweden < 11 3: 10 < time in Sweden < 16 4: 15 < time in Sweden < 21 5: 20 < time in Sweden < 31 6: 30 < time in Sweden < 41 7: time in Sweden > 40
Unemployed in t-2 and t-3 (worker) and in t-2 (other parent)	Dummy indicating whether the individual is registered at the PES
Unemployed long t-2 (worker, other parent)	Dummy indicating whether the individual has been registered at the PES more than 180 days
Income from employment t-2 (worker)	Income from employment or self-employment, deflated with CPI to 2014 prices
Household disposable income t-2 (worker, other parent)	Log household disposable income in 100 s SEK, deflated with CPI to 2014 prices
Income from social assistance t-2 (worker, other parent)	Log social assistance in 100 s SEK, deflated with CPI to 2014 prices in the individual's household
Swe * social assistance t-2 (worker, other parent)	Interaction variable between born in Sweden and income from social assistance in the individual's household
Education t-2 (worker, other parent)	Dummy variables for years of schooling (3) 1: years of school < 10 2: 9 < years of school < 13 3: years of school > 12
Tenure t-2 (worker)	Dummy variables for number of years employed at the current workplace. Categories: 2, 3, 4 and 5 or more years.
Size of workplace t-2 (worker)	Number of workers at workplace
Size of workplace ^2 t-2 (worker)	Number of workers at workplace, squared
Small workplace t-2 (worker)	Dummy variable indicating whether the workplace has fewer than 50 workers
Medium-sized workplace t-2 (worker)	Dummy variable indicating whether the workplace has more than 49 but fewer than 250 workers
Industry sector t-2 (worker)	Dummy variables for industry sector, SNI code (9)
New firm t-2	=1 if the firm was established less than 7 years ago
GPA std. at firm t-2	Average GPA of children of workers at the workplace
County t-2(worker)	Dummy variables for county of residence (25)
Base year	Dummy variables for sample year 1995-2000

Figure 1: Parental effects (unemployment, earnings, disposable income)

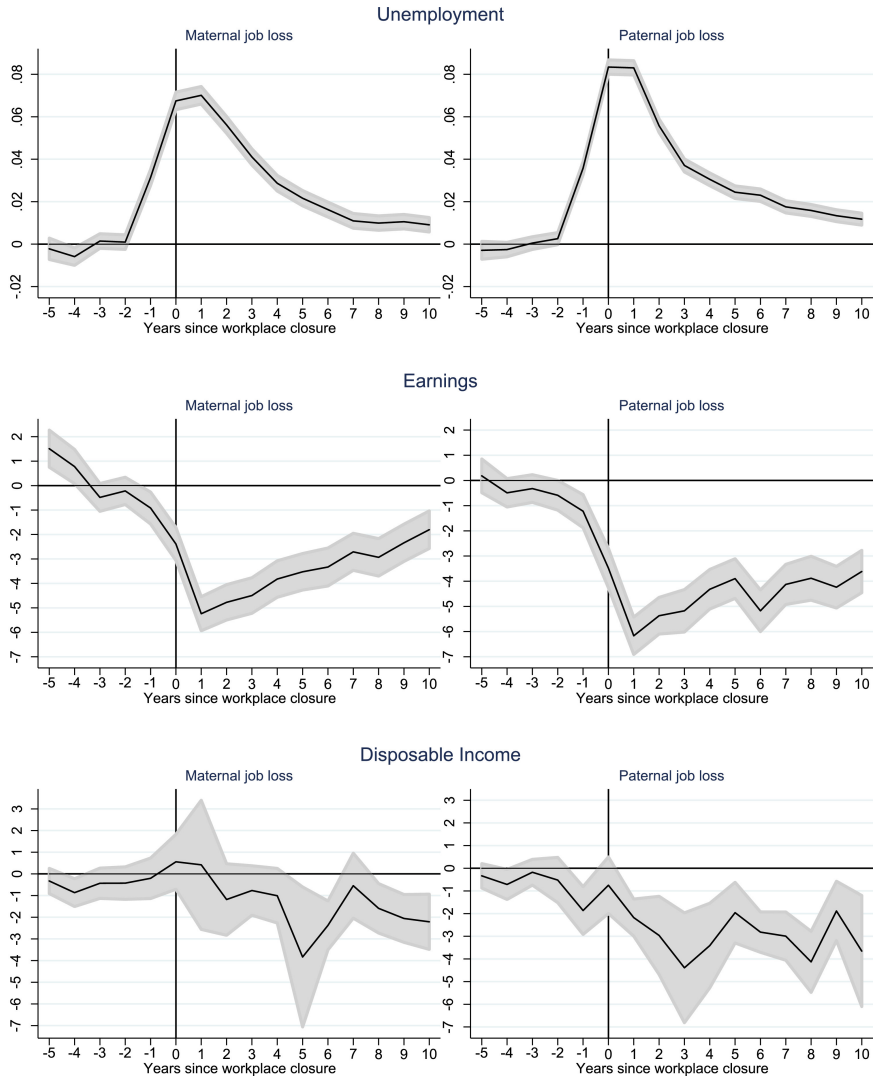


Figure 2: Parental effects (separation, mortality)

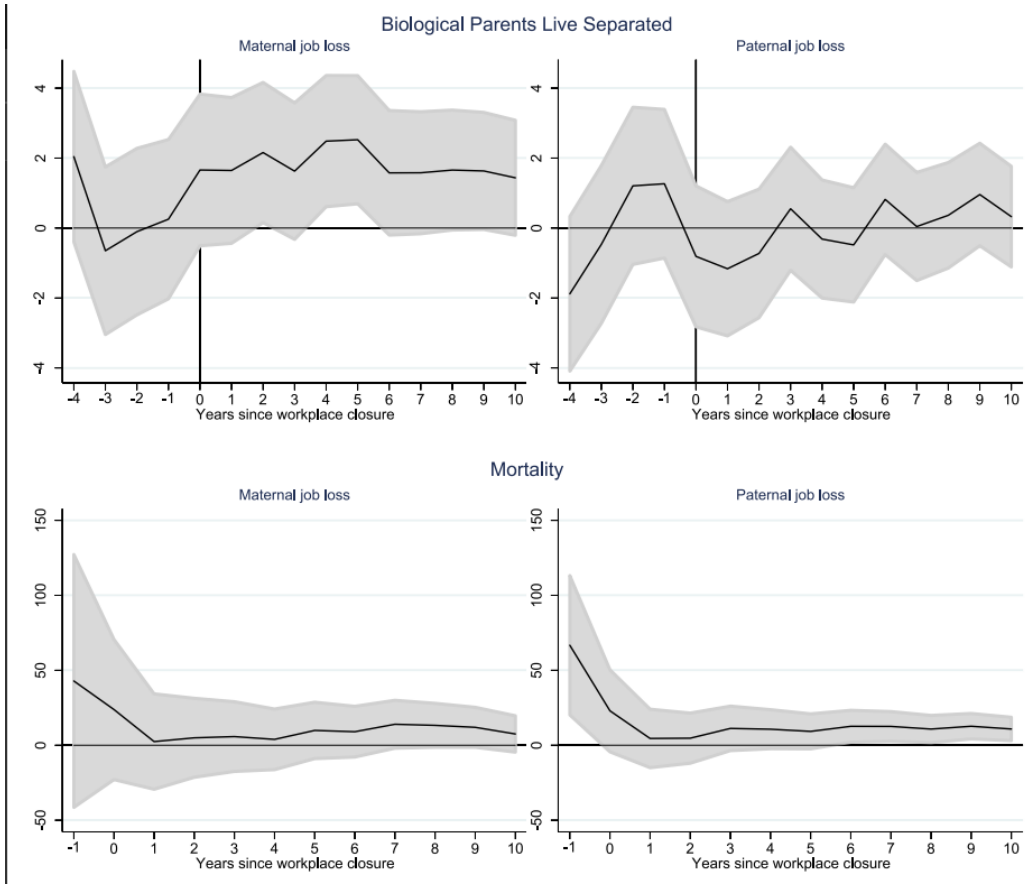


Figure 3: Effects on children (GPA)

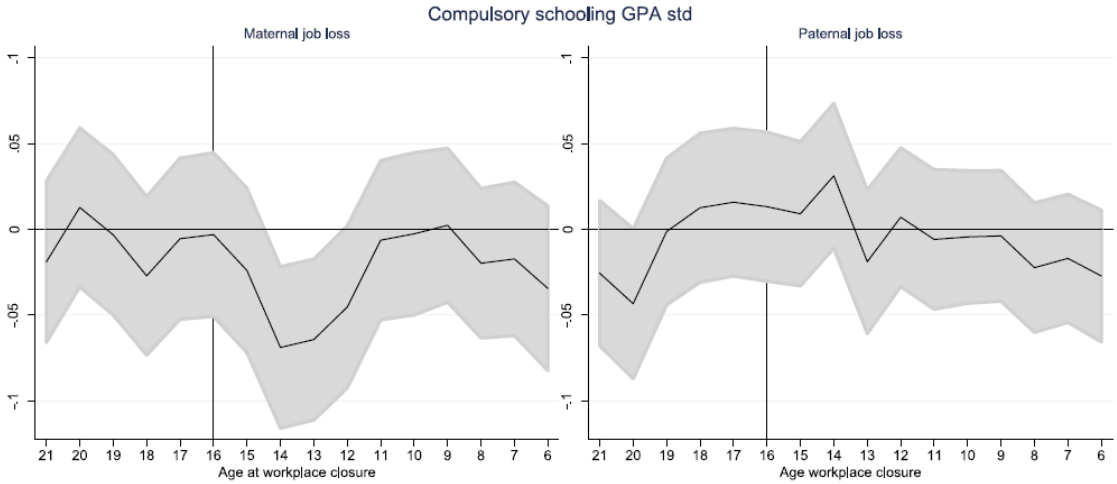


Table 4: Paternal effects (separation, mortality)

Period	Estimate	Std. error	# obs.	Estimate	Std. error	# obs.
	Separated			Mortality		
t-4	-0.00331	0.00198	1,215,605			
t-3	-0.000797	0.00196	1,218,928			
t-2	0.00205	0.00196	1,218,928			
t-1	0.00238	0.00204	1,213,660	0.423**	0.151	1,218,928
t	-0.00167	0.00212	1,208,817	0.349	0.212	1,218,928
t+1	-0.0026	0.00218	1,204,667	0.125	0.274	1,218,928
t+2	-0.00173	0.00224	1,201,022	0.174	0.318	1,218,928
t+3	0.00140	0.00228	1,197,702	0.548	0.370	1,218,928
t+4	-0.000853	0.00233	1,193,975	0.673	0.419	1,218,928
t+5	-0.00137	0.00236	1,190,567	0.722	0.466	1,218,928
t+6	0.00242	0.00239	1,187,053	1.196*	0.516	1,218,928
t+7	0.000129	0.00243	1,182,878	1.382*	0.557	1,218,928
t+8	0.00116	0.00247	1,178,394	1.395*	0.599	1,218,928
t+9	0.00317	0.00248	1,173,615	1.868**	0.640	1,218,928
t+10	0.00111	0.00251	1,168,611	1.868**	0.688	1,218,928

Note: Estimates from propensity score matching (nearest neighbour with replacement). Standard errors, taking into account that the propensity score is estimated, in parentheses. ** p<0.01, * p<0.05.

Table 5: Parental effects (health)

	Maternal job loss				Paternal job loss			
	Hospitalization	Mental health	Alcohol	Mortality	Hospitalization	Mental health	Alcohol	Mortality
0–10 years	-4.854	0.543	1.331*	0.787	1.815	0.220	-0.143	1.868**
after job loss	(3.027)	(1.062)	(0.641)	(0.650)	(2.485)	(0.868)	(0.665)	(0.688)
# observations	1,011,726	1,011,726	1,011,726	1,034,025	1,173,243	1,173,243	1,173,243	1,218,928
# treated children	53,361	53,361	53,361	54,614	77,131	77,131	77,131	80,295
Mean of outcome	356.740	28.579	9.689	10.492	326.471	27.5116	16.102	17.237
3–8 years	2.249	0.0372	-0.0372		2.512	0.423	0.487	
before job loss	(2.818)	(0.591)	(0.286)		(2.097)	(0.519)	(0.370)	
# observations	1,021,327	1,021,327	1,021,327		1,186,795	1,186,795	1,186,795	
# treated children	53,800	53,800	53,800		78,023	78,023	78,023	
Mean of outcome	274.331	8.848	2.082		196.993	9.613	4.755	

Note: Estimates from propensity score matching (nearest neighbour with replacement). Standard errors, taking into account that the propensity score is estimated, in parentheses. ** p<0.01, * p<0.05. Means of outcomes calculated for the matched controls.

Table 6: Effects on children (health)

	Maternal job loss			Paternal job loss		
	Hospitalization	Avoidable	Mental and behaviour	Hospitalization	Avoidable	Mental and behaviour
0–10 years *after* job loss	0.00943	0.415	0.0566	1.048	1.434	-1.325
	(2.849)	(1.077)	(1.212)	(2.350)	(0.910)	(0.938)
# observations	1,003,648	1,003,648	1,003,648	1,180,413	1,180,413	1,180,413
# treated children	53,032	53,032	53,032	77,738	77,738	77,738
Mean of outcome	278.257	29.265	37.732	276.765	31.674	33.096
3–8 years *before* job loss	-1.054	0.614	0.000	-1.781	0.913	0.143
	(2.226)	(0.994)	(0.381)	(1.959)	(0.905)	(0.344)
# observations	933,544	933,544	933,544	1,010,322	1,010,322	1,010,322
# treated children	48,852	48,852	48,852	66,254	66,254	66,254
Mean of outcome	129.442	22.763	3.316	137.562	25.334	3.585

Note: Estimates from propensity score matching (nearest neighbour with replacement). Standard errors, taking into account that the propensity score is estimated, in parentheses. ** p<0.01, * p<0.05. Means of outcomes calculated for the matched controls.

Table 7: Effects on children (GPA, highschool, unemployment, social assistance)

	Compulsory school GPA	High school completion at age 20	Social assistance at age 20–23	Unemployed at age 20–23
		Maternal job loss		
Parental job loss	-0.0230**	-0.00170	0.00493*	0.00810*
at age 6–16/18	(0.00718)	(0.00257)	(0.00237)	(0.00367)
# observations	719,042	743,453	753,755	753,755
# treated children	37,395	38,773	39,430	39,430
Mean of outcome	0.0977	0.860	0.115	0.473
Parental job loss	0.00934	0.00132	0.00713	0.00723
at age >16/22	(0.00970)	(0.00359)	(0.00408)	(0.00496)
# observations	419,300	377,290	412,864	412,832
# treated children	21,179	18,577	20,124	20,123
Mean of outcome	0.0371	0.545	0.194	0.635
		Paternal job loss		
Parental job loss	-0.00817	-0.00252	0.00403	-0.000380
at age 6–16/18	(0.00512)	(0.00222)	(0.00210)	(0.00323)
# observations	776,892	770,028	782,219	782,219
# treated children	50,788	50,557	51,382	51,382
Mean of outcome	0.103	0.869	0.117	0.484
Parental job loss	-0.0199*	-0.00511	0.00462	0.000441
at age >16/22	(0.00894)	(0.00324)	(0.00372)	(0.00454)
# observations	381,129	332,505	362,754	362,728
# treated children	25,322	22,002	23,818	23,816
Mean of outcome	0.0837	0.569	0.187	0.646

Note: Estimated using propensity score matching (nearest neighbour with replacement). Standard errors, taking into account that the propensity score is estimated, in parentheses. ** p<0.01, * p<0.05. Means of outcomes calculated for the matched controls.

Figure 4: Comparison of treatment and control distributions

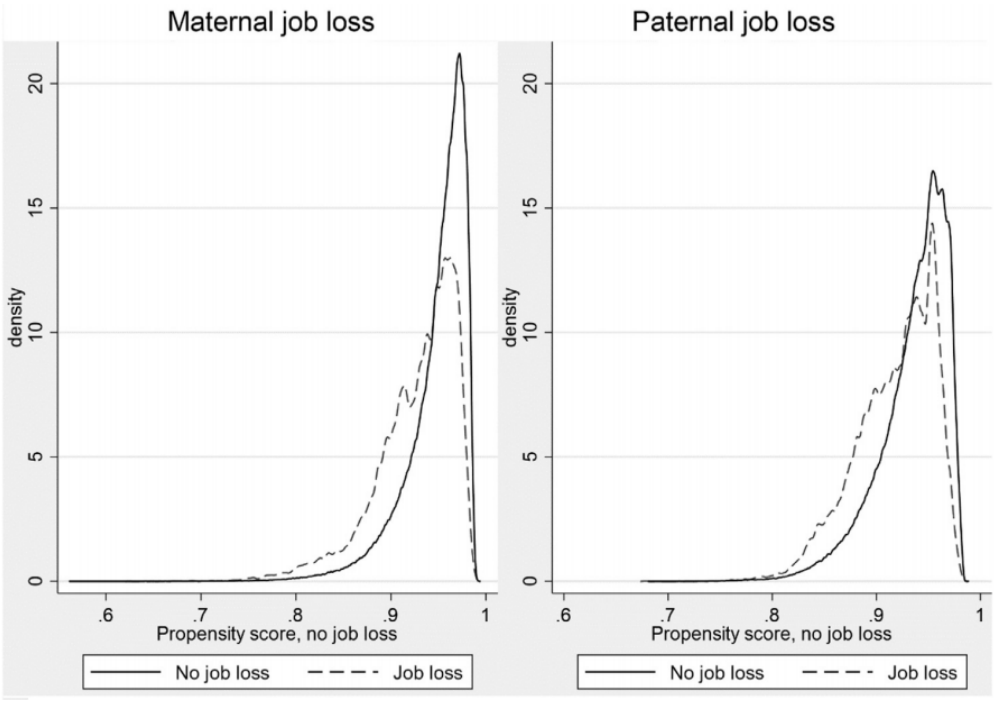
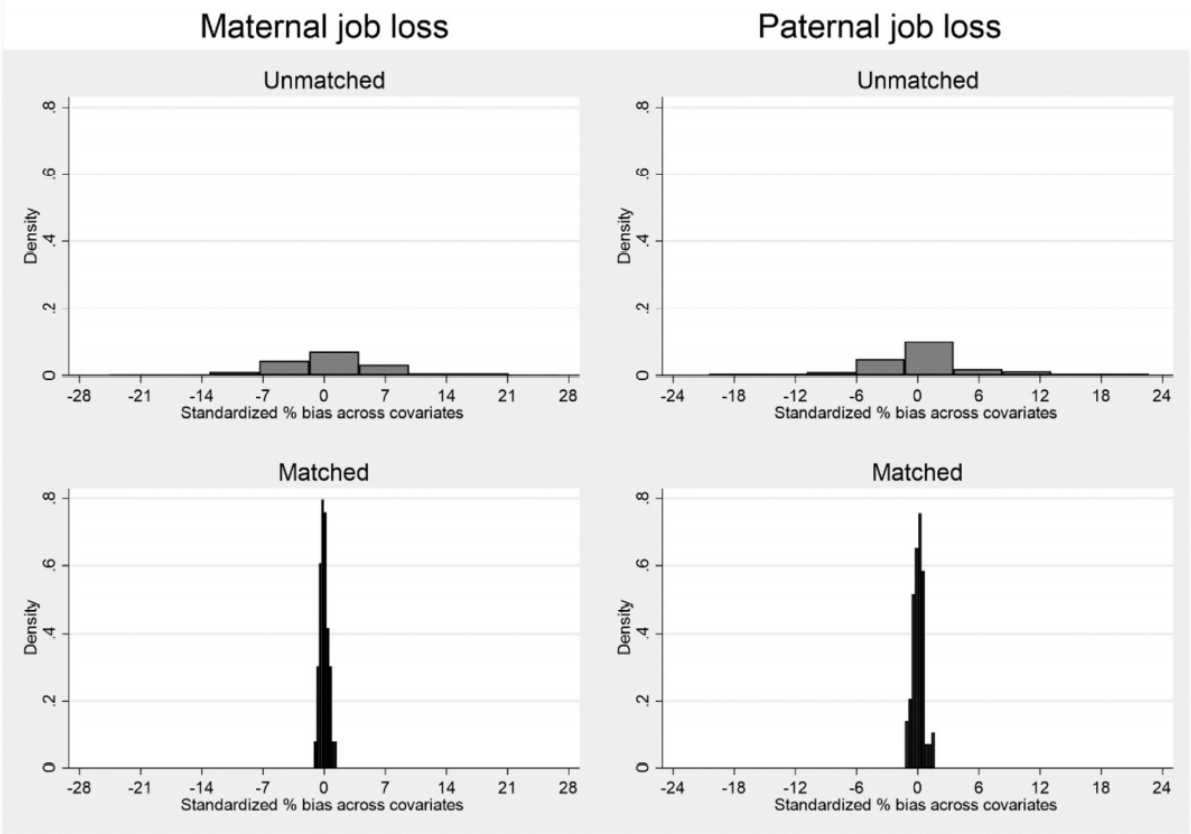


Figure 5: Covariate balance pre and post matching



Declaration of Authorship

I assure that I wrote this seminar paper/bachelor thesis/master thesis on my own without the usage of aids other than indicated. I only used the indicated sources. All literally or analogously translated paragraphs of the original sources are marked as such. The work has not been submitted to any examination office in the same or a similar form. Hereby I agree that the digital version of the work may be checked by plagiarism detection software.