Werther at Work: Intra-firm Spillovers of Suicides

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Abstract

Suicide is a leading cause of death worldwide and a critical public health concern. We examine the hypothesis of suicide contagion within the workplace, investigating whether exposure to a coworker's suicide increases an individual's suicide risk. Using high-quality administrative data from Austria and an event study approach, we compare approximately 150,000 workers exposed to a coworker's suicide with a matched group exposed to a "placebo suicide." We find a significant increase in suicide risk for exposed individuals, with a cumulative treatment effect of 0.04 percentage points (33.3 percent) over a 20-year post-event period. Exposed individuals who also die by suicide are more likely to use the same method as their deceased coworker, strongly suggesting a causal link. Two placebo tests bolster this interpretation: workers who left the firm before the suicide and those exposed to a coworker's fatal car accident do not show an elevated suicide risk.

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Keywords: Suicide, workplace, contagion hypothesis, Werther-effect, mental health.

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1 Introduction

Suicide is a tragic event with long-lasting effects on those left behind in families and communities. It occurs across the lifespan and is a leading cause of death, particularly among younger men. Suicide is a global phenomenon and a major public health problem in all regions of the world. It is the tragic result of a complex set of factors, including mental health, personal struggles, societal pressures, and lack of support systems. To devise effective prevention strategies, it is crucial to deepen our understanding of these underlying causes. An important layer of suicide prevention is also providing support for those who are bereaved by suicide.

Social scientists have developed and tested several theories about suicide. One important hypothesis is that of suicide contagion. The idea that exposure to suicide may affect one's own suicidal behavior, dates back to the release of Johann Wolfgang von Goethe's novel, "*The Sorrows of Young Werther*" in 1774. In this novel, the hero, named Werther, died by suicide. It was widely believed that von Goethe's book led to a wave of young men across Europe deciding to end their lives, many dressed in the same clothes as von Goethe's description of Werther and using similar guns (Thorson and Öberg, 2003). Phillips (1974) coined the term "*Werther effect*" to refer to a positive effect of media coverage on suicide rates. In contrast, the so-called "*Papageno effect*" suggests that media coverage can prevent suicides (Niederkrotenthaler et al., 2010).[1]

In this paper, we empirically test the hypothesis of workplace suicide contagion. The workplace is a social network in which individuals typically spend a considerable amount of time. It is therefore not surprising that previous literature on peer effects has shown that coworkers influence individual decisions in many domains, such as fertility (Pink et al., 2014), parental leave decisions (Dahl et al., 2014), retirement decisions (Duflo and Saez, 2003), investment behavior (Hvide and Östberg, 2015), and intertemporal consumption decisions (De Giorgi et al., 2020). We are testing whether the social influences at work on individual behavior extend to suicide.

We do not believe that a mentally healthy person will suddenly become suicidal because of the suicide of a coworker. But we do consider the possibility that past exposure to a coworker's suicide may be a highly memorable event that may play a role in future periods of poor mental health or tragic life events. Exposed individuals, in their desperation, may interpret the suicide they observed as a perceived solution to their current problems.

We set up a quasi-experimental research design and examine whether individuals who have been exposed to the suicide of a coworker have a different propensity to commit suicide themselves. A keystone of our research design is a match between the *Austrian*

[1]The term originates from the character Papageno in Mozart's opera The "*Magic Flute*." In the opera, Papageno, overwhelmed by despair, contemplates ending his life. However, he is dissuaded by others who offer him alternative solutions and emotional support, ultimately helping him find hope and choose life.

Death Register, and the *Austrian Social Security Database*, a matched employer-employee dataset covering the universe of workers since the 1980s. The former data source contains information on the universe of all deaths along with information on the cause of death. To construct our sample, we proceed in two main steps. First, we start with the workers who actually died by suicide ("deceased workers") and find observationally identical workers who did not die by suicide ("non-deceased workers"). To identify our non-deceased workers, we follow a matching procedure similar to (Jäger and Heining, 2019). Second, once we have matched these pairs, we can naturally use their co-workers at the time of the event as "exposed workers" and "non-exposed workers".

We then use an event study approach to compare our approximately 150,000 exposed workers with non-exposed workers. An important check to show that our research design is credible is the analysis of pre-trends. We show that our matched exposed and non-exposed workers follow the same trend in labor market and health outcomes before the event. The same holds true for their firm characteristics, such as pre-event firm size and firm growth. To our knowledge, we are the first to test the suicide contagion hypothesis in the workplace context with a design-based approach.

We find that exposure to a coworker's suicide has a statistically significant positive effect on one's own suicide. Over a post-event period of 20 years, we find a cumulative treatment effect of plus 0.04 percentage points, or 33.3 percent. The effect is quite stable over the entire post-event period. What strongly supports a causal interpretation of our results is that the exposed individuals who also die by suicide are more likely to choose the same suicide method as their deceased co-worker.

As an important placebo check, we repeat our analysis for workers who left the firm shortly before the suicide event. These workers were exposed to a very comparable (unobserved) firm environment, but were not exposed to the suicide event. We compare this group to workers who left a control firm early. We find a zero effect for these placebo-exposed workers. This supports the notion that our results are not driven by unobserved firm heterogeneity. In a second placebo check, we consider individuals who were exposed to the fatal car accident of a co-worker. While this is also a tragic event, we do not expect any effect on one's suicidal behaviour due to contagion. Again, we find no treatment effects. In terms of heterogeneity, we find that the effect is driven by young and male individuals.

Our work contributes most directly to the empirical literature on suicide contagion. To the best of our knowledge, there is no design-based study testing the suicide contagion hypothesis in the workplace. We are aware of one observational study (Hedström et al., 2008), which uses administrative data to systematically estimate correlations between the exposure to suicide and own suicide.[2]

[2]Hedström et al. (2008) have access to a panel data set covering all individuals who ever lived in Stockholm during the 1990s. The authors use logistic regressions to estimate the association between

A large number of empirical studies across academic disciplines have examined the relationship between media coverage and subsequent suicide risk. The most recent meta-analysis concludes that media coverage of celebrity deaths by suicide increases the risk of suicide, while general coverage of suicide has no effect (Niederkrotenthaler et al., 2020).[3] Many countries now have media guidelines for reporting on suicide (Pirkis et al., 2006) to prevent or reduce this contagion (in the sense of imitation).

While it is certainly important to understand the impact of media coverage on suicide, there are contexts other than media coverage in which suicide contagion may occur. In more private settings, within a peer group, there is likely to be a much closer (emotional) connection between a suicide victim and potential imitators. This relates to the second most studied context of suicide contagion, the spread of suicide among youth in a school (or other community) setting. Methodologically, this literature either uses survey data or works more qualitatively in case studies on so-called space-time suicide clusters. A typical finding in this quantitative and qualitative studies is, that suicides in schools typically lead to elevated rates of suicide and suicide attempts. Based on these methods it seems hard to establish causality.[4]

This literature justifies its focus on adolescents by the fact that this group is at a more vulnerable life course stage for suicidality and typically in socially bounded spaces (such as high schools) that may be more susceptible to suicide contagion. "*Contagion effects are far less evident for adults and the elderly, suggesting that social interactions are less important for these groups.*" (Cutler et al., 2001, page 224). Our findings challenge this assessment and show that more research on suicide contagion among adults may be warranted. It is possible that so far limited data on existing networks among adults, rather than a lack of interest, may account for the focus of the contagion literature on media and youth suicide.

Our paper also contributes to the broader empirical literature on determinants of suicide. There is no monocausal explanation for suicide. Scholars in different academic disciplines focus on different explanation, and offer theories within their domain. Medical specialists consider suicide to be the result of depression and other psychiatric disorders (Mann et al., 2005). In this line of reasoning, suicide is an irrational act resulting from

suicide exposure within the family, and at work and own suicide. Workplace exposure is statistically significant only for men. After accounting for other risk factors, men exposed to suicide at work have a 3.5 times higher likelihood of dying by suicide compared to non-exposed men. In this study, the association within the family domain is statistically significant for both sexes and comparatively higher at about 9. Overall, however, workplace exposure is more important than the family domain because individuals are more often exposed to suicides of coworkers than of family members. However, due to the observational nature of the study, it is unclear whether this association can be interpreted as causal.

[3]The risk of suicide increased by 13% in the period following media coverage of a celebrity's death by suicide. When the method of suicide used by the celebrity was reported, there was an associated 30% increase in deaths by the same method (Niederkrotenthaler et al., 2020).

[4]A related strand of literature studies suicidal ideation among teenagers (see, for instance, Wang, 2016). There is causal evidence for the US that in-person schooling *per se* causes youth suicide (Hansen et al., 2024).

mental illness. In strak contrast to this view, economists typically model suicide as a forward-looking utility-maximizing behavior (Hamermesh and Soss, 1974; Cutler et al., 2001; Becker and Posner, 2004). There is indeed robust empirical evidence that suicide rates respond to economic circumstances, illness, family disruption and other variables that affect the utility from living (Chen et al., 2010).[5] Campaniello et al. (2017) identify a similar behavioral mechanism among inmates in Italian prisons, demonstrating that suicide decisions are influenced by inmates' expectations regarding the length of their sentences. The influence of life circumstances on suicide decisions is also explored in the "deaths of despair" literature (Case and Deaton, 2015, 2017, 2020). This body of work attributes the rising midlife mortality rates in the United States—driven by drug overdoses, alcohol-related deaths, and suicides — to deteriorating economic conditions, particularly among less-educated, middle-aged white Americans.[6] Sociological explanations of suicide are heavily influenced by the work of Durkheim, who argues that the causes must be sought in the relationship of the individual to society. He suggests that suicide is inversely related to the degree of social cohesion and provides some supporting descriptive statistics.[7]

Finally, our results contribute to the literature on peer effects. Existing research demonstrates peer effects (or in some cases peer correlations) for a number of health-related behaviors (Montgomery et al., 2020), such as alcohol consumption (Kremer and Levy, 2008), smoking (Alexander et al., 2001) and overweight (Trogdon et al., 2008). Our findings highlight an additional dimension of health-related behavior where peer effects warrant further consideration.

The remainder of this paper is organized as follows. Section 2 presents our data sources. Section 3 discusses several stylized facts about suicide in Austria, which are also observed in many other countries. Section 4 outlines our research design. First, we explain the matching process for deceased and non-deceased workers, which naturally defines our sample of exposed and non-exposed co-workers. Second, we demonstrate that matched exposed and non-exposed workers exhibit similar trends in health and labor market outcomes prior to the event. Third, we describe our estimation models for assessing the spillover effects of a workplace suicide. Section 5 presents our estimation results. First, we provide baseline estimates for the overall sample. Second, we conduct placebo checks. Third, we examine treatment effect heterogeneity across several dimensions. Section 6 concludes the paper.

[5]For instance, Ruhm (2000) shows that US suicide rates are, in contrast to total mortality, countercyclical. A one percentage point increase in state unemployment rates is associated with an increase in suicides by 1.3 percent.

[6]Ruhm (2022, 2024) offers a critical assessment of the "deaths of despair" hypothesis, challenging its strong causal implications.

[7]Becker and Woessmann (2018) identify social cohesion as an important mechanism by which Protestants have higher suicide rates than Catholics.

2 Data sources

We combine two sources of administrative data: First, we use the *Austrian Death Register*. This covers the universe of deaths since 1980. It provides information on the cause of death based on the *International Classification of Diseases* (ICD) and whether an autopsy was performed. In the case of a suicide, information on the method of suicide is also included. The registry also includes basic socioeconomic information, such as sex, age, marital status, citizenship, and religious denomination.

The reliability and validity of suicide data from the *Austrian Death Register* are high by international standards. Regular training and standardized procedures for recording the cause of death according to ICD by physicians and *Statistics Austria* contribute to the reliability of the data over time. While the specificity (probability that a certified suicide was actually a suicide) can be assumed to be close to 100%, the sensitivity (proportion of correctly classified suicides out of all possible suicides) is certainly lower (Rockett and Thomas, 1999).[8] It is worth noting that drug overdose deaths can be difficult to classify, as intent may be unclear in certain cases. However, Austria has a relatively low number of drug overdose deaths.[9] There are no reliable statistics on suicide attempts, as they are often not recognized or documented as such (BMSGPK, 2024).

Second, we use the *Austrian Social Security Database* (ASSD). These data are administrative records to verify pension claims and are structured as a matched employer-employee dataset. For each private-sector employee, we can observe daily data on his or her workplace and co-workers. We also observe socio-economic characteristics, such as age, education, broad occupation, experience, tenure, and earnings, the latter provided per year and per employer. We can also construct firm-level variables such as firm size (growth). The limitations of the data are the top-coded wages and the lack of information on working hours (Zweimüller et al., 2009). While the ASSD records the date of death, it does not specify the cause of death.

To address this, we match individuals who died between 1980 and 2010 in the *Austrian Death Register* with the ASSD. Since no administrative identifier is available, the matching relies on a combination of sex, birth date, marriage date (if applicable), and death date. These variables enable a unique match for 1,696,724 individuals, which account for approximately 67% of individuals in the *Austrian Death Register*. Non-matched individuals in the ASSD (column 3) either have never been employed or cannot be uniquely identified. The match rate is higher for younger birth cohorts, which can be attributed

[8] Autopsies are an important contributor to the quality of cause-of-death statistics. In Austria, they have declined from 34.7% in 1984 to 19.6% in 2007. This reflects the international trend away from autopsies. Nevertheless, the Austrian autopsy rate is still among the highest in Europe (Kapusta et al., 2011). Appendix Table A.2 shows, based on individual-level data from the *Austrian Death Register*, that there is a small negative correlation between suicide (as final cause of death) and autopsy.

[9] In 2023, the United States recorded 316 drug overdose deaths per one million people, whereas the rate in Austria was only 27.

to their smaller cohort sizes. Consequently, younger cohorts, whose deaths occurred on average in more recent years, are overrepresented in our matched sample (see Appendix Table A.1).

3 Facts about suicide in Austria

In this section we present some basic facts about suicide using data from the *Austrian Death Register*. This is helpful in setting the stage for our later analysis. Most of the patterns described below are not unique to Austria. They can also be observed in other high-income countries.

Figure 1: Leading cause of death for selected age groups, 1970-2010

Rank	10-14	15-24	25-34	35-44	45-54	55-64	All Ages
1	Injuries and Poisoning (1.589)	Injuries and Poisoning (20.624)	Injuries and Poisoning (14.013)	Malignant Neoplasms (16.175)	Malignant Neoplasms (51.618)	Malignant Neoplasms (115.583)	Diseases of the Circulatory System (1.759.050)
2	Malignant Neoplasms (564)	Suicide (5.974)	Suicide (8.057)	Injuries and Poisoning (13.226)	Diseases of the Circulatory System (40.022)	Diseases of the Circulatory System (106.244)	Malignant Neoplasms (783.918)
3	Diseases of the Nervous System (327)	Malignant Neoplasms (2.145)	Malignant Neoplasms (4.921)	Diseases of the Circulatory System (12.913)	Diseases of the Digestive System (18.080)	Diseases of the Digestive System (28.319)	Diseases of the Respiratory System (187.861)
4	Congenital Malformations, Deformities and Chromosomal Anomalies (200)	Diseases of the Circulatory System (1.438)	Diseases of the Circulatory System (3.657)	Suicide (9.878)	Injuries and Poisoning (14.116)	Injuries and Poisoning (14.210)	Diseases of the Digestive System (178.151)
5	Diseases of the Circulatory System (194)	Diseases of the Nervous System (1.164)	Diseases of the Digestive System (1.736)	Diseases of the Digestive System (7.421)	Suicide (10.798)	Diseases of the Respiratory System (11.072)	Injuries and Poisoning (168.185)
6	Suicide (164)	Mental Diseases (888)	Mental Diseases (1.216)	Mental Diseases (1.759)	Diseases of the Respiratory System (4.166)	Suicide (9.388)	Endocrine, Nutritional & Metabolic Diseases (86.635)
7	Diseases of the Respiratory System (163)	Diseases of the Respiratory System (645)	Diseases of the Nervous System (1.082)	Diseases of the Respiratory System (1.693)	Endocrine, Nutritional and Metabolic Diseases (2.867)	Endocrine, Nutritional and Metabolic Diseases (7.977)	Suicide (70.465)
8	Endocrine, Nutritional and Metabolic Diseases (86)	Congenital Malformations, Deformities and Chromosomal Anomalies (466)	Infectious and Parasitic Diseases (802)	Diseases of the Nervous System (1.622)	Diseases of the Nervous System (2.616)	Diseases of the Nervous System (4.373)	Diseases of the Nervous System (54.095)

Notes: Calculations are based on the *Austrian Death Register*.

Suicide occurs across the lifespan. For younger people, who generally have a lower risk of death, suicide is a leading cause of death (see Figure 1). It is the second leading cause of death for those aged 15-34, and the fourth leading cause of death for those aged 34-44. It is at least the seventh leading cause of death in all other age groups. Panel (a) of Figure 2 shows suicide rates by sex and age, based on data for the period from 1970 to 2010. Two important patterns emerge. First, as in every region of the world, we observe a consistently higher rate of suicide among men than among women. Second, suicide is more relevant to older people in absolute terms. For both sexes, we observe most cases among those aged 75 and over.

Panel (b) of Figure 2 shows suicide rates by sex over time. In each year, male suicides outnumber female suicides. For men, suicides had increased between 1970 and 1984. In the year 1984 we observe a turning point. Since then, we observe a decreasing trend, which coincides with the introduction of media guidelines for suicide reporting. For women, we also observe a turning point around the same time. Their rate has been stable and declining since about 1984.[10] Panels (c) and (d) of Figure 2 show sex-specific suicide rates by age over time. We see that the increasing trend in overall male suicide rates between 1970 and 1984, was predominantly driven by those aged 75 and over, and those aged 25-44. Since the late 1980s, we see a declining trend across all age groups. The same holds true for female suicide rates. As in most other countries (Yu et al., 2020), we observe seasonal patterns in suicide, with counts peaking in spring and reaching a low in winter (not shown).

In terms of method of suicide, the majority of cases are by suffocation. Among men, this method accounted for about half of the cases between 1970 and 2010. For women, the proportion is about one-third. There is a gender difference in the second most common method. Men use more firearms (about 20 percent of all male cases), while women use more poisoning (about 25 percent of all female cases). The other most common methods are jumping and drowning. With the exception of a decline in poisoning since the mid-1980s, the distribution of suicide methods is fairly stable over time (see Appendix Figure A.2).

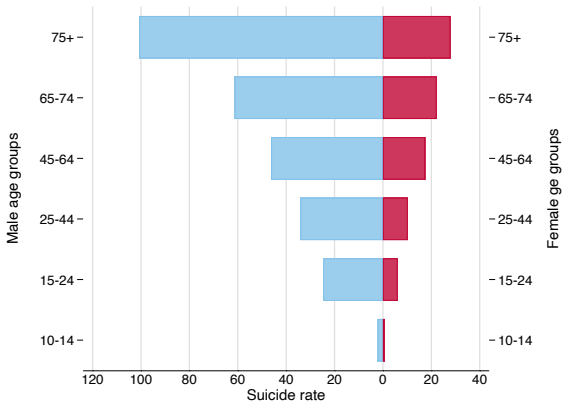
4 Research design

For our estimation, we need to draw samples of two types of workers. The first type has experienced the suicide of a co-worker ("exposed worker"). The second type has not experienced this event ("non-exposed worker"). Apart from this difference, these two types of workers should be indistinguishable. Put differently, we need to obtain a matched sample in which the event "suicide of a co-worker" is exogenous. To construct this sample, we start with the co-worker who has actually committed suicide ("deceased worker"), and match each of these observations with an observationally identical worker who did not committed suicide ("non-deceased worker"). Once we have matched these pairs, we can naturally use their co-workers at the time of the event as exposed and non-exposed workers. Co-workers who left the firm before shortly before the event are used for a placebo test (see Section 5.2).

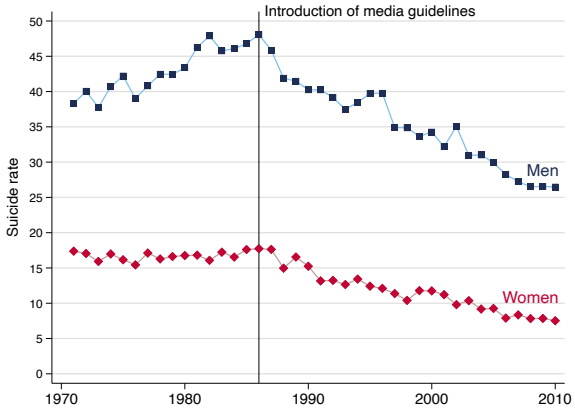
In the remainder of this section, we first describe this multistage matching procedure in more detail. We then present our estimation strategies applied to this sample. Finally,

[10] Whether the introduction caused the decline is an interesting question, but beyond the scope of this paper. For a simple time-series analysis see Niederkrotenthaler and Sonneck (2007). It is worth noting that Austria introduced selective serotonin reuptake inhibitors (SSRIs) in 1985. These antidepressants have been linked to a reduction in suicide rates (Ludwig et al., 2009).

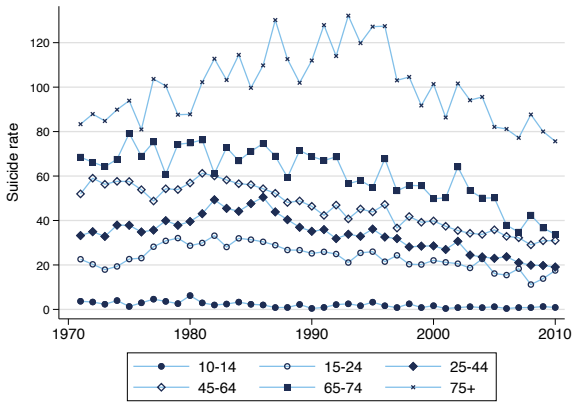
Figure 2: Suicide rates in Austria



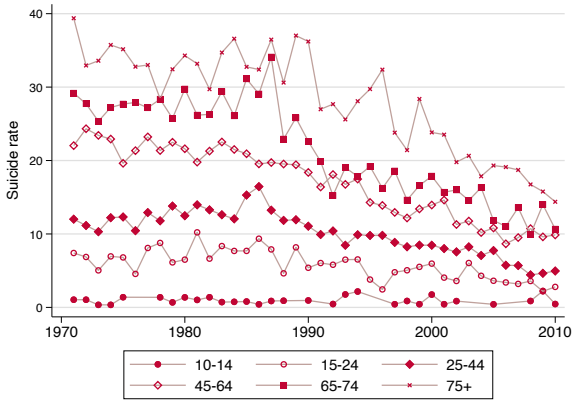
(a) Suicide rates by sex and age, 1970-2010



(b) Suicide rates by sex, 1970-2010



(c) Male suicide rates by age, 1970-2010



(d) Female suicide rates by age, 1970-2010

Notes: Calculations are based on the *Austrian Death Register*. Suicide rates are defined as the number of persons who died by suicide per 100,000 population. The sex- and age-specific population for the years 1972 to 1980 is based on a linear interpolation between the years 1971 and 1981. Panel (a) is based on the population 10 year and older. Panel (b) is based on data from 1970 to 2010. Bars on the left are for men. Bars on the right are for women. Note that panels (c) and (d) have differently scaled vertical axes. Appendix Figure A.1 provides the figures in Panel (b) by decade.

we present evidence that exposed and non-exposed workers share common pre-trends in key labor market and health outcomes.

4.1 Matching of deceased and non-deceased workers

In a first step, we identify all individuals aged 15 to 65 who committed suicide between 1980 and 2010 in the *Austrian Death Register*. We then match these individuals to the *Austrian Social Security Database*. From this sample, we exclude individuals who were not employed at the time of death, who were not employed 4 years prior to the death date, who were employed in very large firms ($>$ 1,000), and who had a long-term sickness spell in the year prior to death. We introduce these restrictions to obtain a sample of workers consistent with our research question and design. We are then left with 1,259

deceased workers (see first column in Table 1). The majority of this group are men (about 84 percent) and the average age is 36 years. In terms of socio-economic characteristics, the average worker has a medium education and is employed in a blue collar job (about 66 percent).

In our second step, we identify all worker-firm pairs which did not experience a suicide. For each quarter, we sample a comparable non-deceased worker from the set of worker-firm pairs in firms which did not experience a suicide in a given quarter q. This given quarter determines the respective placebo event date. As in the case of the deceased workers, we disregard observations based on age, firm-size, employment and long sick leave restrictions. For the remaining non-deceased worker, we obtain their labor market outcomes up to 4 years prior to the placebo event date.

In our third step, we follow a matching procedure similar as Jäger and Heining (2019). For each worker in the treatment group and event time q, we sample worker-firm pairs from the control group with the same sex, age, education and earnings percentile four years prior to death, as well as same firm-size percentile four years prior to death. If multiple control individuals can be found, we do the following. We obtain a propensity score for each worker based on a linear regression using covariates measured four years prior to the (placebo) event.[11] We then use the control individuals with the propensity score closest to the treated individual. The second column in Table 1 describes our 1,259 matched non-deceased workers.[12] As expected, these workers have exactly the same means for the matched variables as the deceased workers. But also for the non-matched variables we find very comparable means.

4.2 Exposed and non-exposed co-workers

For each worker-firm pair in the deceased and the non-deceased group, we determine all co-workers in the same firm at the (placebo) event date t. We refer to these as the sample of exposed and non-exposed workers, respectively. These two groups of co-workers constitute our main estimation sample. For each worker in this sample, we capture their past and future labor market outcomes. We also determine whether the worker died until the end of our observation period and the cause of death.

Columns three and four in Table 1 describe our sample of exposed and non-exposed workers, which constitutes our estimation sample. In total, we have 151,373 workers exposed to a co-worker's suicide, and 171,830 workers who were not exposed. Although we do not include any workers from these groups in our initial matching process, the characteristics of workers exposed to a co-worker's suicide are very similar to those of workers

[11] The covariates are sex, age, age-squared, education, daily wage and firm tenure in t_{-4}, firm's size and mean wage in t_{-4}, and NACE-08 1-digit industry dummies.

[12] Of the 1,259 treated firm observations, 1,066 appear once, 151 twice, and 42 three or more times. In instances where a firm was subject to multiple treatments, we treat these firms as separate observations.

Table 1: Balancing checks of individual characteristics

	"Matched"		"Non-matched"	
	Deceased	Non-deceased	Exposed	Non-Exposed
Outcome				
Suicide	1.000	0.000‖	0.0013	0.0010
Matching variables				
Age	35.48	35.48	35.82	35.84
	(9.43)‡	(9.43)	(10.96)	(10.99)
Female	16.36	16.36	33.41	37.19
High education	10.33	10.33	23.15	21.86
Medium education	52.10	52.10	45.27	43.54
Non-matched covariates				
Tenure in firm (quarters)†	19.48	22.73	19.99	19.80
	(21.30)	(21.19)	(21.64)	(21.34)
Any long-term sickness spell†	0.00‖	0.00‖	7.46	6.89
Long-term sickness days†	0.00‖	0.00‖	2.93	2.78
			(17.68)	(17.54)
Blue collar worker†	66.32	60.84	54.36	51.91
Daily wage (in Euros)†	84.94	83.12	86.09	83.21
	(62.16)	(32.48)	(34.27)	(32.72)
Individuals	1,259	1,259	151,373	171,830

Notes: The first two columns show summary statistics of individual background characteristics of individuals who died by suicides (deceased) and the matched control individuals (non-deceased) respectively. Columns four and five show summary background information for individuals exposed to the suicide of a co-worker and controls (non-exposed) respectively. All variables are measured on year prior to treatment. To obtain non-deceased control individuals, for each quarter q a deceased worker, a non-deceased worker firm pair with the same sex, age, education, earnings percentile, and firm-size percentile as the deceased worker, measured four years prior to the death date is matched. In case multiple control matches are available, first a propensity score from a linear model, accounting for sex, age, age-squared, education, daily wage, firm tenure, as well as firm size, firm mean wage, and NACE-08 1-digit industry dummies measured four years prior, is obtained. Then, the control individuals with the closest propensity score to the deceased is selected. Exposed (non-exposed) individuals are then all co-workers of a deceases (non-deceased) worker at the respective firm. † These variables are not included in the matching procedure. ‡ Standard deviations are only shown for non-binary variables. ‖ These means are zero by sample construction.

who were not exposed in the past. The same holds true for firm-level characteristics such as sector, size, and average wage (see Table 2).

4.3 Pre-trends in important labor market and health outcomes

Given that suicide is an absorbing state, it is impossible to check whether our matched exposed and non-exposed workers follow the same trend in the outcome variable before

Table 2: Balancing checks of firm characteristics

	Firms of	
	Deceased & exposed workers	Non-deceased & non-exposed workers
Matching variables		
Sector		
Production	28.99	29.39
Construction	14.46	14.61
Service	16.60	16.76
Others	39.95	39.24
Non-matched covariates		
Total number of employees†	172.93	193.70
	(228.89)‡	(231.35)
Firm mean wage (in Euros)†	63.13	64.21
	(22.49)	(22.40)
Firm age (in years)†	17.83	19.11
	(9.92)	(9.81)
Firms	1,259	1,259

Notes: The table shows firms' summary statistics of deceased/exposed individual in the first column and non-deceased/non-exposed individuals in the second column; see also notes to Table 1. All variables are measured on year prior to the (placebo) event date. † These variables are not included in the matching procedure. ‡ Standard deviations are only shown for non-binary variables.

the event. However, we can perform an alternative assessment, based on important labor market and health outcomes, such as days employed, log daily wages, and long-term sickness spells. Small and insignificant differences prior to the actual exposure suggest an absence of differential trends in labor and health outcomes that could otherwise explain post-treatment differences unrelated to the event itself. They would also give reassurance that there was little anticipation of the exposure.

To assess whether labor market and health outcomes differed between exposed and non-exposed individuals already prior to exposure, we estimate an event-study specification on our matched sample of exposed and non-exposed workers.

$$y_{i,r} = \alpha_i + \sum_{\substack{r=-4 \\ r\neq -1}}^{5} \beta_r \cdot \mathbb{1}(r = t - s) + \sum_{\substack{r=-4 \\ r\neq -1}}^{5} \delta_r \cdot \mathbb{1}(r = t - s) \cdot S_i + u_{i,r} \tag{1}$$

Here, S_i is a binary indicator, taking a value of one if an individual i was exposed to a suicide and $y_{i,r}$ denotes the outcome of the worker in year $r = t - s$, relative to the base year s. We also include worker fixed effects and leads and lags around the even

time $\mathbb{1}(r = t - s)$.[13] The coefficients of interest are δ_r. Our difference-in-differences (DiD) design, with a perfectly balanced panel, resembles a stacked design similar to that of Cengiz et al. (2019). Consequently, it does not suffer from potential issues related to heterogeneous treatment effects in difference-in-differences and event study settings (de Chaisemartin and D'Haultfoeuille, 2020; Callaway and Sant'Anna, 2021; Sun and Abraham, 2021).[14] When investigating the impact on long-term sick leaves, we also include tenure at the current firm as well as firm size dummies in Equation (1). These additional covariates account for the fact that eligibility for and duration of sick leave benefits in Austria depend primarily on the size of the employer and worker's tenure at the current firm.[15] In general, none of our event study estimates are sensitive to the inclusion of covariates.

Figure 3 plots the coefficients δ_s from our event-study. We do not find evidence that exposed workers differ in important labor market and health outcomes from non-exposed workers prior to a co-workers suicide. Panel (a) and (b) of Figure 3 show that exposed workers had similar employment and waged trends prior to the exposure. There is also no evidence that exposed workers are more likely to enter long-term sick leave spells (panel c) or that they have sick leave spells, if they do enter long term sick leave (panel d). When testing the joint significance of the pre-exposure coefficients, we fail to reject the null hypothesis for all considered outcomes. Overall, the results in this section show that exposed and non-exposed workers had similar labor market trajectories before the treatment.[16] The same holds for event studies in the subsamples of white- and blue-collar workers (see Appendix Figures A.7 and A.8). It also holds for firm characteristics. Appendix Figure A.6 shows that firms of exposed workers followed the same trends as those of non-exposed workers prior to the suicide event, in key dimensions such as employment size and pay structure.

[13] We consider individuals sampled at different years and different firms as different workers, similar as in Jäger and Heining (2019). For example, if an individual i was sampled in 1995 in firm A and in 2001 in firm B, we include two separate fixed effects in our estimation. In that sense, α_i accounts for unobserved heterogeneity across workers within the same firm. Notice that we do not include calendar time fixed effects, as calendar time is balanced between the exposed and non-exposed group through our matching procedure. Including calendar time effects leaves the point estimates unchanged and has only minor effects on the estimated standard errors.

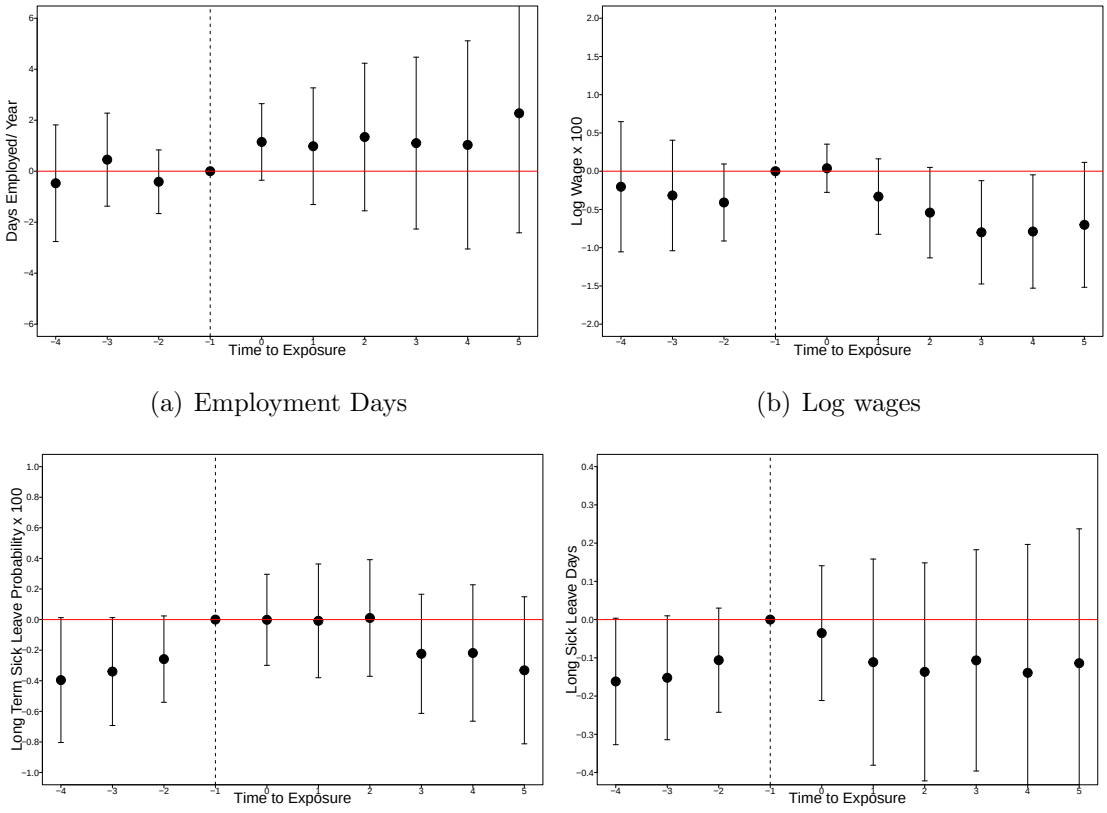
[14] An alternative to our matched DiD design would, in principle, be the synthetic control difference-in-differences (SCDD) estimator proposed by Arkhangelsky et al. (2021), which combines synthetic control methods with a DiD framework. The SCDD estimator reweights and matches on pre-exposure trends in the outcome in a data-driven manner, while also allowing for individual fixed effects, resembling a type of weighted two-way fixed effects regression. However, since our outcome is absorbing, there are no observable pre-trends, and thus we cannot implement the SCDD approach.

[15] In Austria, sick workers receive their full salary for a pre-defined period of time from the employer only. After this period, firms only need to pay a share of the salary and the remainder is topped up by public sickness benefits, in which case we observe the long-term sickness spell in our data. The exact duration depends on various factors, mainly on the size of the employer and tenure at the current firm, see Halla et al. (2015) for a detailed explanation.

[16] Our results do not crucially depend on including workers with sick leaves. Excluding all individuals with a sick leave spell in the year prior to (non-) exposure gives virtually identical results.

It is important to notice that we do not assume that health and labor market outcomes *after* treatment are unaffected by the exposure to a co-worker's suicide. For example, being exposed to a co-worker's suicide may lower mental health, which can lead to worse labor market outcomes after the exposure. Such an observation does not invalidate our research design. We find that wages of exposed workers decrease relative to non-exposed ones *after* exposure to a co-worker's suicide.

Figure 3: Impact of exposure to co-worker suicide on labor market outcomes: Event-study results



(a) Employment Days

(b) Log wages

(c) Long sick leave (0/1)

(d) Long sick leave days

Notes: The figure plots the estimated coefficients δ_r from the event-study specification in Equation 1. Vertical lines show 95% confidence intervals, obtained from standard errors clustered at the firm level. Panel (a) and (b) use yearly employment days and log daily wages as outcome respectively. Panel (c) uses a binary indicator whether the individual had a long-term sick leave spell in a given year. Panel (d) plot the estimated coefficients when using the total long-term sick leave days as outcome. The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 151,373 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 171,830 workers. See Section 4 for details on the exact matching procedure. P-values from a Wald test for joint significant of pre-expousre coefficients are as follows: employment days ($p = 0.17$); log wages ($p = 0.29$); long sick leave (0/1) ($p = 0.22$); and long sick leave days ($p = 0.25$).

4.4 Estimation strategies

We employ three different estimation models, each designed to capture different dynamics to fully capture the spillover effects of workplace suicides. First, we use a standard linear probability model that captures the cumulative treatment effect over our entire time horizon of 20 years. Next, we relax the assumption of time-invariant linear effects and employ a nonparametric model to estimate the dynamic cumulative treatment effect. Finally, we use a period-by-period specification to identify time-varying hazard ratios.

4.4.1 Cumulative treatment effect based on linear model

Our first approach is the standard linear probability model. Our outcome variable is D_i, a binary indicator that takes a value of one if an individual i has died by suicide by the end of our observation period, and zero otherwise. Similarly, our treatment variable S_i is defined as a binary indicator that takes a value of one if an individual i was exposed to a suicide by a co-worker, and zero otherwise. In our estimation model

$$D_i = \alpha + \gamma S_i + \beta x_i' + \epsilon_i, \tag{2}$$

we include a vector of covariates x_i which accounts for individual and firm characteristics. To control for workers' socio-economic background we include age, age squared, gender, education, occupation, as well as the wage and tenure at the relevant firm. All these variables are measured at baseline. To capture workers' health we also include a long-term sickness indicator.[17] Firm characteristics include firm size, average wage within the firm, firm age as well as a set of sector dummies. In addition, we include event year fixed effects in all our regressions.

Under the assumption that, after controlling for individual and firm characteristics, there are no systematic differences between the matched exposed and non-exposed individuals, the so-called conditional independence assumption (CIA), the parameter γ in Equation (2) captures the causal cumulative spillover effects of exposure to a co-worker's suicide. Although the CIA is not directly testable, it is likely to hold in our setting. As shown in the previous section, we find no significant difference in key outcomes between the matched exposed and non-exposed groups, either in terms of personal characteristics or firm variables. More reassuringly, even when we look at outcomes not included in our matching procedure, such as tenure and wages, we still observe balance. Our pre-trend estimates further suggest that individuals exposed to suicides are unlikely to follow a different career path than those not exposed to such an event before the actual exposure. Therefore, it is unlikely that unobserved shocks, such as to individuals' labor market careers, can explain our results.

[17] Our results are identical when excluding the sick leave dummy from our models or when excluding all individuals with a sick leave spell in the year prior to (non-) exposure.

4.4.2 Cumulative treatment effect based on nonparametric model

Our linear model in equation (2) informs us about the cumulative and time-aggregated effects of suicide spillover. However, for the design of suicide prevention strategies, it may be important to identify specific sensitive time periods. To capture such potential dynamic effects, we also consider a nonparametric model which provides estimates of the cumulative treatment effects at different points in time (relative to the event time).

To define our nonparametric model, we introduce some additional notation. Let S be the indicator whether a worker was exposed to a suicide of a co-worker, as described earlier. Let $Y(1)$ be the potential time until death by suicide when exposed, and $Y(0)$ be the potential time when not exposed. Denote by $\mathbb{1}\{A\}$ the indicator function which takes a value of 1 if the argument A is true. The dynamic impacts of exposure on workers' cumulative risk of dying by suicide is

$$\Delta(t) = E[\mathbb{1}\{Y(1) \leq t\} - \mathbb{1}\{Y(0) \leq t\}] \tag{3}$$

Identification of $\Delta(t)$ is complicated by censoring, as we only observe Y only until 2010. The outcome of workers who died after 2010 is unobserved. Due to censoring, instead of observing Y directly, we only observe

$$D = SD(1) + (1 - B)S(0)$$

where $D(1) = min\{Y(1), C\}$ and $D(0) = min\{Y(0), C\}$, with C are censoring times. We also observe a censoring indicator

$$\xi = B\xi(1) + (1 - B)\xi(0)$$

where $\xi(1) = \mathbf{1}\{Y(1) \leq C\}$ and $\xi(0) = \mathbf{1}\{Y(0) \leq C\}$.[18]

To estimate $\Delta(t)$ under these challenges we follow the approach suggested in Sant'Anna (2016) and Schmidpeter (2024) (see also Browning and Heinesen, 2012). They show that under the CIA, we can estimate $\Delta(t)$ from the data using a propensity score weighted Kaplan-Meier estimator. Specifically, we first estimate weights ω as

$$\omega = \frac{S}{\pi(X)} + \frac{(1 - S)}{1 - \pi(X)},$$

where $\pi = P(S = 1)$ and $\pi(X) = P(S = 1|X)$ are the propensity scores, which we estimate by means of a logistic regression, including the same set of covariates as in our

[18] Implicit in our notation is the assumption that there is no selective drop out from our sample, neither in the exposed nor in the non-exposed group; see the discussion in Sant'Anna (2016) and Schmidpeter (2024). Since our analysis is based on administrative data and every person is followed until death, this is not of a concern in our analysis.

linear probability model. In a second step, these weights are used to obtain propensity score weighted hazard rates and a propensity score weighted Kaplan-Meier estimator. At time t_j and for member i of group $S = s$, the propensity score weighted hazard rate is given by

$$h_{t_j}^{S=s} = \frac{\sum_{i:D_i=t_j} \omega_i \xi_i \mathbf{1}\{S_i = s\}}{\sum_{i:D_i \geq t_j} \omega_i \mathbf{1}\{S_i = s\}}. \tag{4}$$

The survival function for group $S = s$ is then given by the propensity score weighted Kaplan-Meier estimator

$$\delta_t^{S=s} = \prod_{t_j \leq t} \left(1 - h_{t_j}^{S=s}\right). \tag{5}$$

We use the estimates of δ_t to obtain the main treatment effect as the difference of the weighted Kaplan-Meier estimates between the non-exposed and exposed groups,

$$\gamma_t^{WKME} = \delta_t^0 - \delta_t^1, \tag{6}$$

where γ_t^{WKME} reflects the risk difference of dying by suicide between the exposed and non-exposed group.[19] A positive effect therefore indicates that exposure to a co-worker's suicide increases the own risk of dying by suicide. We also report period-by-period hazard ratios

$$R_p = \frac{h_p^1}{h_p^0} \tag{7}$$

where we pool different time periods together to ensure stable estimates. We make this explicit by using the subscript p instead of t. Since death by suicide is a relatively rare event, this pooling is necessary to prevent periodically small or large hazard rates to influences the estimated spillover effects. In practice, we do so using estimated hazard rates aggregated over one and two years respectively.

Our estimates of γ_t^{WKME} capture at each elapsed time t the difference in the cumulative suicide spillover effect under the CIA. Likewise, under the CIA, R_p gives the relative suicide spillover effect at a specific point in time p.The advantage of γ_t^{WKME} over the total cumulative effect γ is that we are able to capture any possible underlying dynamics without imposing strong restrictions on how the treatment effect evolves over time. This is particularly important in settings, such as in ours, where one does not have a strong prior about treatment effect dynamics.

Expressing the effect of interest as in Equation (6) also avoids difficulties in interpret-

[19]Note that δ_t^S is an estimate of the survival function $P(Y(S) > t)$. Thus, $\Delta(t) = \left(1 - \delta_t^1\right) - \left(1 - \delta_t^0\right) = \delta_t^0 - \delta_t^1$.

ing the treatment effect, for example, compared to hazard ratios. While hazard ratios, as express in Equation (7) are very popular in applied work and have an intuitive interpretation as reflecting relative risks, they can also suffer from a "built-in" selection bias, even under initially perfect randomization (Hernán, 2010; Schmidpeter, 2024). The challenges of hazard ratios arises from the inclusion of individuals at time p who have not yet experienced the event until that time for calculation. If the treatment negatively interacts with unobserved factors, such as initial health conditions, this can lead to a negative dynamic selection over time, *even when* the share of individuals with such unobserved health problems is balanced at baseline. Consequently, the proportion of individuals with unobserved health problems in the treatment group decreases initially faster relative to the share in the control group. But this implies that more individuals with unobserved problems remain in the control group over a longer time period, leading to dynamic selection. Therefore, estimated hazard ratios could decrease, not because there is not a long-run impact of our treatment but because of selection.[20]

Despite these considerations and for comparability, we also report hazard ratios R_p. The hazard ratios allow us to obtain a snapshot at a specific time period how exposure to suicide affects individuals at a specific time period, keeping in mind the dynamic selection issue. Overall, our three estimation approaches allow us to obtain a complete picture of the possible spillover effects of suicide and their dynamics over time.

5 Estimation results

In this section, we begin by presenting our baseline estimates for the overall sample. Next, we report the results of our placebo checks. First, we replicate our analysis for workers who left the firm shortly before the suicide event. Second, we examine individuals who were exposed to a co-worker's fatal car accident. Finally, we explore treatment effect heterogeneity across several dimensions.

5.1 Baseline estimates

Table 3 summarizes the cumulative treatment effect γ over the entire 20-year post-event period, estimated using our linear model (see equation 2). We present three specifications: in column (1), we include only year fixed effects; in column (2), we add individual-level covariates; and in column (3), we further incorporate firm-level covariates. The individual-level covariates include age, sex, education, occupation type (blue- vs. white-collar), wage, and history of extended sick leave. Firm-level covariates include firm size, sector, average

[20]This argument is similar to the one often made in duration models, where one explicitly models unobserved heterogeneity to account for selection (e.g. Abbring and Van den Berg, 2003; Frimmel et al., 2022). Our approach, however, imposes less structure on the underlying heterogeneity and is therefore more flexible.

Table 3: Average treatment effect over 20 years

	(1)	(2)	(3)
Exposed to co-worker's suicide	0.0004*** (0.001)	0.0004*** (0.001)	0.0004*** (0.0001)
%-change	33.33	33.33	33.33
Individual-level covariates			
Age (squared)	No	Yes	Yes
Education	No	Yes	Yes
Sex	No	Yes	Yes
Tenure	No	Yes	Yes
Wage	No	Yes	Yes
Collar	No	Yes	Yes
Long sick leaves	No	Yes	Yes
Firm-level covariates			
Sector	No	No	Yes
Size	No	No	Yes
Av. employee wage	No	No	Yes
Av. employee age	No	No	Yes
Year FE	Yes	Yes	Yes
Mean of dep var	0.0012	0.0012	0.0012
Observations	323,203	323,203	323,203

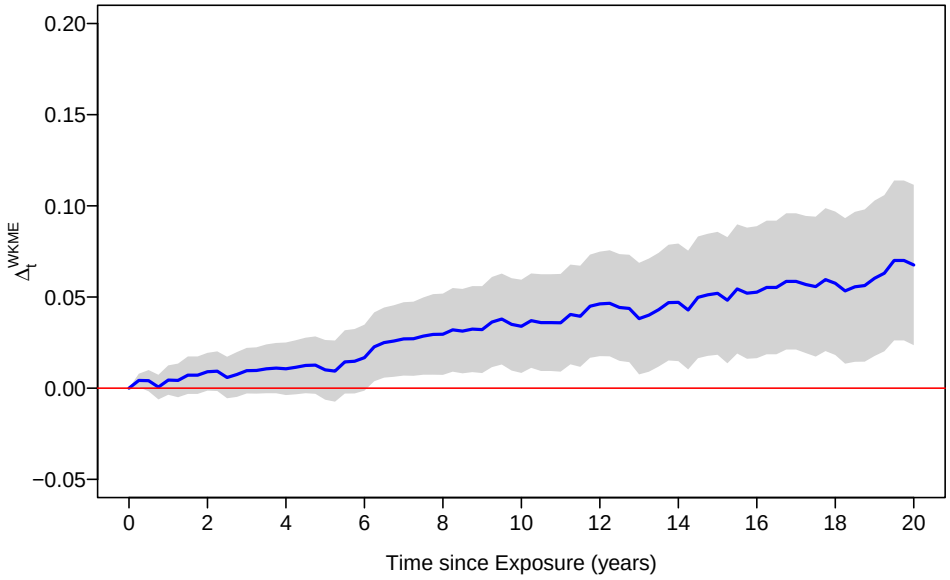
Notes: The table shows the coefficient γ from Equation (2), the average effect of dying by suicide when exposed to the suicide of a co-worker. %-change is calculated relative to mean of the dependent variable. Each column presents the results when a set of additional control variables are added. The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 151,373 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 171,830 workers. See Section 4 for details on the matching procedure, and notes to Table 1 for details on the covariates.

employee wage, and average employee age. Across all specifications, we consistently find that exposure to a co-worker's suicide increases the cumulative likelihood of an individual's own suicide by 0.04 percentage points, equivalent to an increase of approximately 33 percent. The fact that adding covariates does not alter the estimated effect size supports the notion that our research design provides exogenous variation in the treatment.

Figure 4 shows the cumulative treatment effect based on our nonparametric model (see equation 6). This approach allows us to flexibly trace the dynamics of the treatment effect over time. The plotted weighted Kaplan-Meier estimator, γ_t^{WKME}, captures the cumulative effect of exposure to a co-worker's suicide on the probability of dying by

suicide at each elapsed time t. The slope of the estimated function is remarkably constant over time. This suggests that the treatment effect is fairly stable over the entire 20-year post-event period. This is confirmed by our estimation of period-by-period hazard ratios, R_p (see equation 7). Appendix Figure A.3 presents two different specifications based on one-year and two-year intervals. While the estimates show some variation across periods, they generally support the idea of a relatively stable treatment effect over time. Therefore, our parsimonious linear model likely captures the true dynamics with a high degree of accuracy.

Figure 4: Impact of exposure to co-worker suicide on suffering from suicide: Weighted Kaplan-Meier Estimates



Notes: The figure plots the estimated coefficients Δ_t^{WKME} from the weighted Kaplan-Meier Estimator in Equation 6. The depicted effect shows the *cumulative* effect of being exposed to a co-worker's suicide on the probability of dying from suicide. Shaded areas correspond to 95% confidence intervals, obtained from a non-parametric bootstrap with 1,000 replications. The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 151,373 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 171,830 workers. See Section 4 for details on the exact matching procedure.

Substantially, this estimation suggests that the emotional and psychological effects of exposure to a co-worker's suicide are not short-lived but instead persist over the long term. Such exposure may lead to enduring emotional or cognitive changes that continue to influence an individual's behavior over time. One potential mechanism behind this finding is the combination of a stressor later in life with an active memory of the past exposure. Psychiatrists argue that some suicides are impulsive acts (Gvion et al., 2015) triggered by

stressful life events (Fjeldsted et al., 2017) such as financial difficulties or divorces. It is possible that individuals previously exposed to suicide may react differently when faced with a crisis later in life. These individuals may be more likely to perceive suicide as a solution to their problems, while those without such exposure may seek alternative coping mechanisms.

5.1.1 Predictive power of exposed suicide method

If one is exposed to a co-worker's suicide, they likely learn something about the circumstances. One detail that may become common knowledge among co-workers is the suicide method, and this information is documented in our data (see Appendix Figure A.2). We use this information to compare suicide methods between initially deceased workers and exposed workers who later died by suicide. Table 4 provides a contingency table showing the joint relative frequencies for each method, with absolute marginal frequencies in the 8^{th} column and row, respectively. With the exception of firearms, there is strong alignment across all suicide methods. Specifically, the relative frequencies in the diagonal cells are consistently higher than those in the off-diagonal cells. Thus, exposed individuals who also die by suicide are more likely to select the same suicide method as their deceased co-worker.[21]

Figure 5 provides another way to illustrate this connection. The top dark blue bars show the conditional probability that an exposed worker adopts a specific suicide method, given that their initially deceased co-worker used the same method. These probabilities correspond to the relative frequencies found in the diagonal cells of Table 4. In contrast, the second and third bars represent the unconditional probabilities of each suicide method for initially deceased and exposed workers, respectively. These unconditional probabilities align with the marginal distributions of our contingency Table 4, reflecting the average shares for each group. The clear difference between the conditional and unconditional probabilities — where the conditional probabilities are consistently higher — highlights the predictive power of the initially deceased co-worker's suicide method. This observation strongly supports a causal interpretation of our findings.

5.2 Placebo tests

We now conduct two placebo checks. First, we repeat our analysis for workers who left the firm shortly before the suicide event ("early leavers"). These workers were exposed to the same (or a very similar) firm environment but were not present during the suicide event itself. A zero placebo effect would support our previous interpretation. We define

[21] The original literature on the "*Werther effect*", which links media reporting on suicides to subsequent suicide cases, also highlights a correlation between the methods used in the reported case and those in the subsequent cases (Domaradzki, 2021).

Table 4: Contingency table of suicide methods among initially deceased and exposed workers

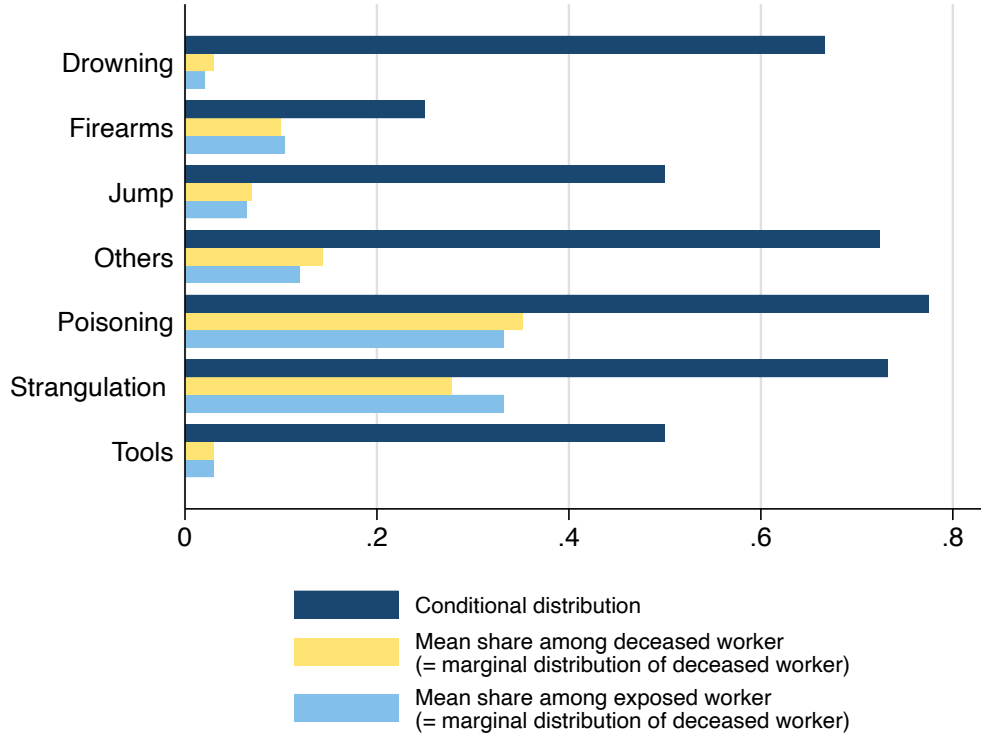
	Suicide method of deceased worker							
	(1) Pois -oning	(2) Strang -ulation	(3) Jump	(4) Fire- -arms	(5) Tools	(6) Drown -ing	(7) Others	Obs.
(1) Poisoning	**77.46**	7.14	14.29	5.00	16.67	0.00	13.79	67
(2) Strangulation	12.68	**73.21**	21.43	**45.00**	33.33	16.67	6.90	67
(3) Jump	0.00	3.57	**50.00**	10.00	0.00	0.00	6.90	13
(4) Firearms	8.45	12.50	14.29	25.00	0.00	16.67	0.00	21
(5) Tools	0.00	3.57	0.00	5.00	**50.00**	0.00	0.00	6
(6) Drowning	0.00	0.00	0.00	0.00	0.00	**66.67**	0.00	4
(7) Others	1.41	0.00	0.00	10.00	0.00	0.00	**72.41**	24
Observations	71	56	14	20	6	6	29	

Notes: This contingency table presents suicide methods among initially deceased workers and those exposed workers who later died by suicide. It displays the joint relative frequencies for each method, with absolute marginal frequencies provided in the 8^{th} column and row, respectively. The columns represent the causes of suicide among initially deceased workers, while the rows indicate causes among exposed workers. Bold entries highlight the highest value in each column.

the placebo exposed group as those employed at the treated firms, but left up to one year before the exposure quarter, resulting in a sample of 20,524 placebo-exposed workers. We also apply this approach to firms with non-deceased workers. The placebo non-exposed group consists of workers who left a non-deceased (=control) firm up to one year prior to the non-exposure quarter, yielding 24,685 placebo non-exposed workers. Thus, in our placebo check, we compare workers who left a treated firm early to those who left a control firm early. These two groups exhibit highly comparable pre-exposure characteristics (see Appendix Table A.3) and appear somewhat negatively selected compared to individuals in the baseline sample. We then apply the estimation analysis from above to this new sample. As expected, we find no effect for these placebo-exposed workers. This result holds across both our linear model estimates (see Table 5) and our nonparametric model (see Panel a of Figure 6). This supports the idea that our results are not driven by an unobserved poor firm environment.

Second, we repeat our analysis for workers whose co-worker died in a traffic accident. Although a traffic accident is a tragic event, we do not expect it to influence an individual's own risk of suicide. Thus, observing no effect here would further support our previous interpretation. We identify all cases of fatal traffic accidents (ICD-9 codes V800-V848) classifying the co-workers of these deceased individuals as placebo-exposed. To create a comparable placebo non-exposed group, we first match each deceased worker with a

Figure 5: Conditional and marginal distribution of suicide method among initially deceased and exposed workers



Notes: The top dark blue bars represent the conditional probabilities that an exposed worker adopts a specific suicide method, given that their initially deceased co-worker used this same method. These probabilities correspond to the relative frequencies in the diagonal cells of Table 4. The second and third bars depict the unconditional probabilities of selecting each suicide method for initially deceased and exposed workers, respectively. These values correspond to the two marginal distributions of our contingency Table 4, representing the relative frequencies for each group.

non-deceased counterpart, then assign the co-workers of these non-deceased individuals to the placebo non-exposed group, following the same algorithm as described in Section 4. This approach results in a sample of 67,792 placebo-exposed workers (those with a co-worker who died in a traffic accident) and 82,295 placebo non-exposed workers (with no co-worker who died in a traffic accident). We then apply our estimation analysis to this new sample. For both our estimation approaches we find zero effects (see Table 5 and Panel b of Figure 6).

In summary, both placebo tests support the causal interpretation of our estimates discussed in Section 5.1.

5.3 Treatment effect heterogeneity

Finally, we present three sets of tests to examine treatment heterogeneity. First, we split the sample by the exposed workers' sex, age, occupation, and joint tenure with

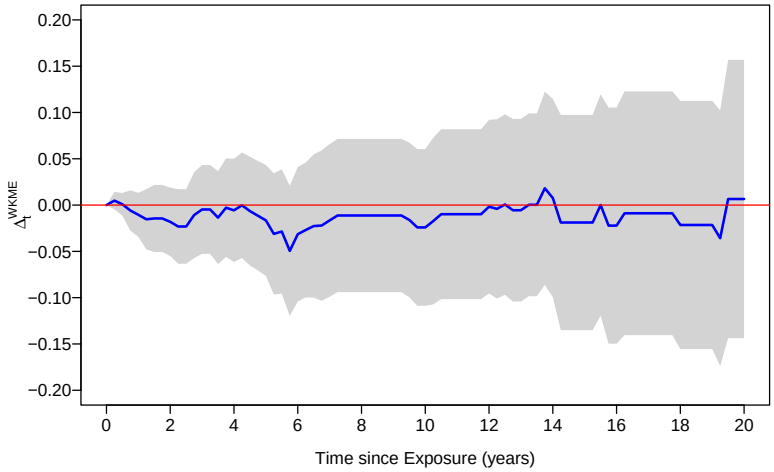
Table 5: Placebo checks with "early leavers" and workers exposed to co-worker with fatal traffic accident

	(1) **Early leavers**	(2) **Traffic accidents**
Placebo treatment	0.0000	0.0001
	(0.0004)	(0.0002)
%-change	1.11	9.09
Individual-level covariates		
Age (squared)	Yes	Yes
Education	Yes	Yes
Sex	Yes	Yes
Tenure	Yes	Yes
Wage	Yes	Yes
Collar	Yes	Yes
Long sick leaves	Yes	Yes
Firm-level covariates		
Sector	Yes	Yes
Size	Yes	Yes
Av. employee wage	Yes	Yes
Av. employee age	Yes	Yes
Year FE	Yes	Yes
Mean of dep var	0.0018	0.0011
Observations	45,209	150,087

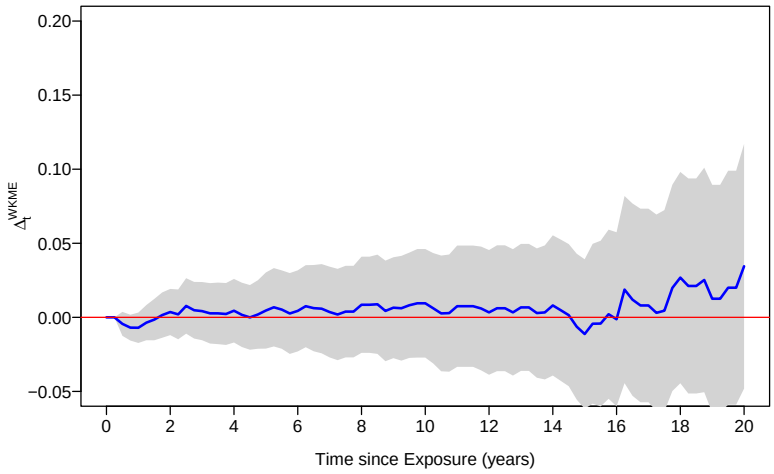
Notes: The table shows the coefficient γ from Equation (2) for two placebo-treatments. %-change is calculated relative to the mean of the dependent variable. The first placebo treatment in Column (1) is being an "early leaver." The "early leaver" group includes only individuals who left the firm up to one year before the placebo exposure (placebo non-exposure); in total 45,209 early leavers, with 20,524 individuals in the placebo exposed group and 24,685 observation in the placebo non-exposed group. The second placebo treatment in Column (2) is exposure to the death of a co-worker in a traffic accident. The construction of the "traffic accidents" group follows the same sampling steps as outlined in Section 4 but defines exposure as the fatal traffic accident of a co-worker. This results in a total of 150,087 observations, comprising 67,792 individuals in the placebo exposed group and 82,295 observations in the placebo non-exposed group.

the deceased worker. Table 6 summarizes the estimated coefficients for these subsamples based on our linear model. We observe positive treatment effects across all subsamples. In terms of sex, the effects are statistically significant for males but not for females. Although the percentage effect is larger for females, we conclude that the overall effect is primarily driven by male workers. For age, the effects are mainly driven by younger workers (below 35 years of age), for whom the estimated treatment effect is both statistically significant

Figure 6: Placebo checks with "early leavers" and exposure to co-worker with fatal traffic accidents: Weighted Kaplan-Meier Estimates



(a) Early Leavers



(b) Exposure to fatal traffic accident

Notes: The figure plots the estimated coefficients Δ_t^{WKME} from the weighted Kaplan-Meier Estimator in equation 6 for placebo treatment groups. The depicted effect shows the *cumulative* effect of being exposed to a co-worker's suicide on the probability of dying from suicide. Shaded areas correspond to 95% confidence intervals, obtained from a non-parametric bootstrap with 1,000 replications. Panel (a) includes only individuals who left the firm up to one year before the exposure (non-exposure); in total 45,209 early leavers with individuals 20,524 in the placebo-exposed group and 24,685 observation in the placebo-non exposed group. Panel (b) follows the same sample construction steps as outlined in Section 4, but uses as exposure a fatal traffic accident of the co-worker. This results in a total of 150,087 observations, comprising 67,792 individuals in the placebo exposed group and 82,295 observations in the placebo non-exposed group.

and larger in magnitude. With respect to occupation, we observe statistically significant effects for both blue-collar and white-collar workers. However, since white-collar workers have a lower baseline probability, the effect is larger when expressed as a percentage change. Finally, we differentiate between individuals who worked with the deceased for two years or less and those who worked with them for more than two years at the same

firm. One might speculate that a longer joint tenure with the deceased could result in larger treatment effects. However, we observe statistically significant effects for both tenure groups, with nearly identical relative effect sizes when expressed as percentage changes. Figure 7 illustrates the estimated cumulative treatment effects for the splits by sex and age, based on our nonparametric model. These results confirm our interpretation that the effects are primarily driven by young male workers.

Table 6: Heterogeneous average treatment effect over 20 years

	Group of (non-)exposed workers included in sample							
	Sex/Gender		**Age**		**Occupation type**		**Joint tenure**	
	Male	Female	Young (< 35 yrs)	Old (≥ 35 yrs)	Blue collar	White collar	Short (≤ 2 yrs)	Long (> 2 yrs)
Exposed to co-worker's suicide	0.0004** (0.0002)	0.0002 (0.0002)	0.0006*** (0.0002)	0.0001 (0.0002)	0.0004** (0.0002)	0.0004** (0.0002)	0.0005** (0.0002)	0.0003** (0.0002)
%-change	26.67	36.11	42.86	11.99	26.67	50.00	35.71	33.33
Individual-level covariates								
Age (squared)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sex	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Tenure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Wage	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Collar	Yes	Yes	Yes	Yes	No	No	No	No
Long sick leaves	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Firm-level covariates								
Firm size	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Av firm's wage	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Av firm's age	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sector	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mean of dep var	0.0015	0.0006	0.0014	0.0009	0.0015	0.0008	0.0014	0.0009
Observations	208,724	114,479	163,939	159,264	171,478	151,383	162,174	161,029

Notes: The table shows the coefficient γ from Equation (2), the average effect of dying by suicide when exposed to the suicide of a co-worker. %-change is calculated relative to the mean of the dependent variable. Each column corresponds to a different subgroup. Subgroups are categorized by sex/gender (first and second columns), age (third and fourth columns), occupation (fifth and sixth columns), and the length of joint tenure with the deceased coworker at the firm. The short (long) joint tenure group comprises individuals who worked with the deceased for two years or less (more than two years) at the same firm. See Section 4 and notes to Table 1 for details.

Second, we test the hypothesis that exposed workers may respond more strongly (or exclusively) to the suicide of a co-worker of the same type. We approximate this by the type of their collar (blue vs. white). Specifically, we define "same-type exposure" as a suicide by a co-worker of the same collar type, and replicate our analysis for this new exposure classification. Table 7 presents, for comparison, our baseline effect in the first column. The second column shows the effect of collar-specific exposure. The third and fourth columns display the results from the collar-specific exposure in the subsamples of blue-collar and white-collar workers, respectively. A comparison of the estimated treatment effects across collar types reveals that all the estimated effects are quite similar. This finding does not support the idea that the type of deceased co-worker influences one's own suicide behavior. The equivalent nonparametric estimates (see Appendix Figure A.4) support this conclusion.

Third, we investigate whether the impact of exposure to suicide varies by firm size. One might hypothesize that smaller firms foster greater camaraderie, which could result in heightened emotional involvement following exposure to a suicide. To test this hypothesis, we re-estimate our model using sub-samples defined by firm size. Appendix Figure A.5 presents the estimated coefficients. The results suggest that the treatment effect remains relatively constant across firm sizes. However, in the sub-sample of smaller firms (with fewer employees), we cannot rule out that the lower statistical power may obscure potential differences.

6 Conclusions

Suicide is a leading cause of death and a critical public health issue. While much research has examined suicide contagion in media and youth settings, our study sheds light on its occurrence in workplace environments. Using high-quality administrative data from Austria, we demonstrate that exposure to a coworker's suicide significantly increases an individual's suicide risk. Our event study approach compares approximately 150,000 workers exposed to a coworker's suicide with a matched group exposed to a "placebo suicide". Importantly, we demonstrate that exposed and non-exposed workers exhibit parallel trends in labor market and health outcomes before the event, as do their firms' pre-event characteristics, such as size and growth.

We find a statistically significant positive effect of coworker suicide exposure on subsequent suicide risk. Over a 20-year post-event period, the cumulative treatment effect is an increase of 0.04 percentage points, or 33.3 percent. The exposed individuals who also die by suicide are more likely to adopt the same suicide method as their deceased coworker, providing strong evidence of a causal link.

To validate our findings, we conduct two placebo checks. First, we analyze workers who left the firm shortly before the suicide event and find no effect on their suicide risk.

Table 7: Heterogeneous average treatment effect over 20 years with collar-specific treatment

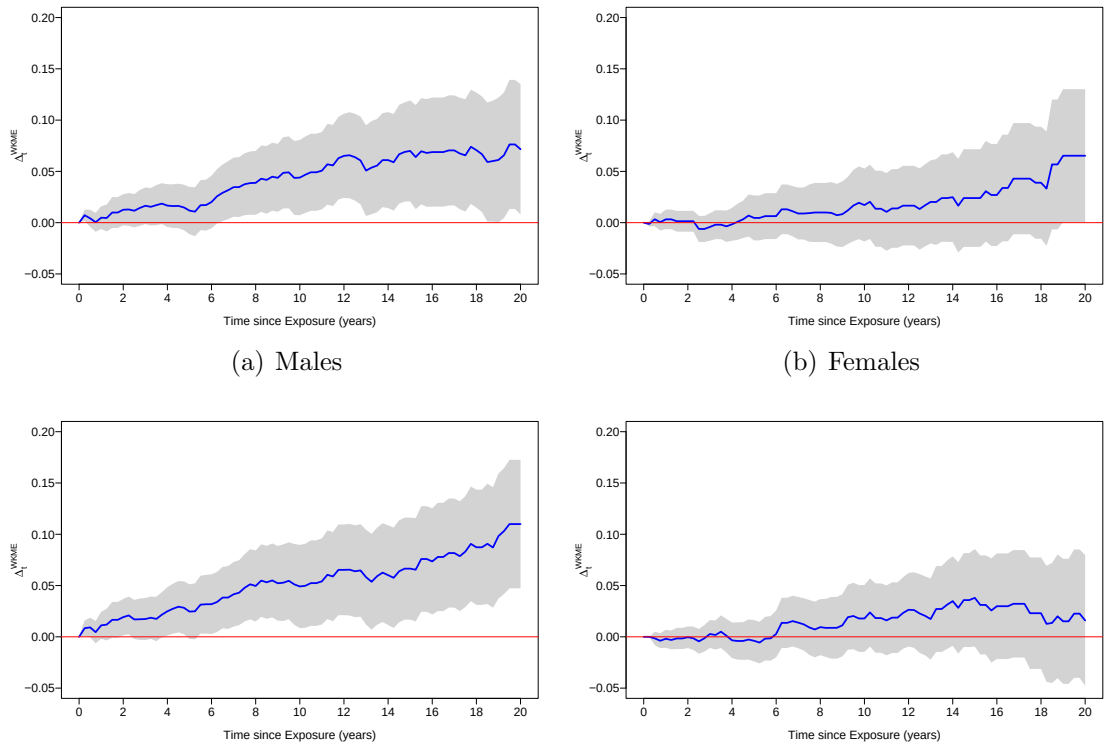
	(1)	(2)	(3)	(4)
	Baseline	*Only treated by deceased workers with same collar* (others cases excluded)		
	All	**Collares pooled**	**Blue collar**	**White collar**
Exposed to co-worker's suicide	0.0004***	0.0004**	0.0004*	0.0003
	(0.0001)	(0.0002)	(0.0002)	(0.0002)
%-change	33.33	30.77	28.57	37.50
Individual-level covariates				
Age (squared)	Yes	Yes	Yes	Yes
Education, Sex	Yes	Yes	Yes	Yes
Tenure	Yes	Yes	Yes	Yes
Wage	Yes	Yes	Yes	Yes
Collar	Yes	Yes	No	No
Long sick leaves	Yes	Yes	Yes	Yes
Firm-level covariates				
Firm size	Yes	Yes	Yes	Yes
Av. firm's wage	Yes	Yes	Yes	Yes
Av. firm's age	Yes	Yes	Yes	Yes
Sector	Yes	Yes	Yes	Yes
Location FE.	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Mean of dep var	0.0012	0.0013	0.0016	0.0008
No. of observations	323,203	236,978	135,471	101,507

Notes: The table shows the coefficient γ from equation (2), the average effect of dying by suicide when exposed to the suicide of a co-worker. %-change is calculated relative to the mean of the dependent variable. Column (1) shows the baseline specification from Table 3. Column (2) shows the results when considering only deceased and exposed workers with the same collar. Columns (3) and (4) do the same, but separately for blue and white collar workers respectively. See Section 4 and notes to Table 1 for details.

Second, we examine individuals exposed to a coworker's fatal car accident and again find no effect on their suicidal behavior. These results underscore the specificity of suicide contagion in workplace settings.

These findings underline the importance of extending mental health support programs beyond the family to include workplace environments. Interventions such as peer counseling, employee assistance programs, and targeted suicide prevention training could play a crucial role in mitigating the risk of contagion after a workplace suicide. Further re-

Figure 7: Heterogeneous impacts of exposure to co-worker suicide on suffering from suicide: Weighted Kaplan-Meier Estimates



(a) Males

(b) Females

(c) Young: Below 35 yrs. at exposure

(d) Old: Above 35 yrs. at exposure

Notes: The figure plots the estimated coefficients Δ_t^{WKME} from the weighted Kaplan-Meier Estimator in equation 6 for different subgroups. The depicted effect shows the *cumulative* effect of being exposed to a co-worker's suicide on the probability of dying from suicide. Shaded areas correspond to 95% confidence intervals, obtained from a non-parametric bootstrap with 1,000 replications. Panels (a) and (b) present results broken down by gender. Similarly, Panels (c) and (d) show results based on age groups, distinguishing between younger workers (aged below 35) and older workers (aged 35 and above) at the time of exposure.

search is needed to evaluate the effectiveness of such programs and to better understand the mechanisms of workplace suicide contagion. Qualitative studies could examine the roles of shared grief, emotional proximity, and discussions surrounding the coworker's suicide in influencing exposed individuals. By addressing these gaps, future studies can contribute to more effective strategies for reducing the public health burden of suicide in both workplace and broader social contexts.

References

Abbring, J. H. and G. J. Van den Berg (2003). The Nonparametric Identification of Treatment Effects in Duration Models. *Econometrica 71*(5), 1491–1517.

Alexander, C., M. Piazza, D. Mekos, and T. Valente (2001). Peers, Schools, and Adolescent Cigarette Smoking. *Journal of Adolescent Health 29*(1), 22–30.

Arkhangelsky, D., S. Athey, D. A. Hirshberg, G. W. Imbens, and S. Wager (2021). Synthetic Difference-in-Difference. *American Economic Review 111*(12), 4088–4118.

Becker, G. S. and R. A. Posner (2004). Suicide: An Economic Approach. Unpublished manuscript, University of Chicago, Chicago, IL.

Becker, S. O. and L. Woessmann (2018). Social Cohesion, Religious Beliefs, and the Effect of Protestantism on Suicide. *The Review of Economics and Statistics C*(3), 377–392.

BMSGPK (2024). Suizid und Suizidprävention in Österreich. Technical report, Bundesministerium für Soziales, Gesundheit, Pflege und Konsumentenschutz, Wien.

Browning, M. and E. Heinesen (2012). Effect of Job Loss due to Plant Closure on Mortality and Hospitalization. *Journal of Health Economics 31*, 599–616.

Callaway, B. and P. H. Sant'Anna (2021). Difference-in-Differences with Multiple Time Periods. *Journal of Econometrics 225*(2), 200–230.

Campaniello, N., T. M. Diasakos, and G. Mastrobuoni (2017). Rationalizable Suicides: Evidence from Changes in Inmates' Expected Length of Sentence. *Journal of the European Economic Association 15*(2), 388–428.

Case, A. and A. Deaton (2015). Rising Morbidity and Mortality in Midlife among White non-Hispanic Americans in the 21st Century. *Proceedings of the National Academy of Sciences 112*(49), 15078–15083.

Case, A. and A. Deaton (2017). Mortality and Morbidity in the 21st Century. *Brookings Papers on Economic Activity*.

Case, A. and A. Deaton (2020). *Deaths of Despair and the Future of Capitalism*. Princeton, NJ: Princeton University Press.

Cengiz, D., A. Dube, A. Lindner, and B. Zipperer (2019). The Effect of Minimum Wages on Low-Wage Jobs. *Quarterly Journal of Economics 134*(3), 1405–1454.

Chen, J., Y. J. Choi, K. Mor, Y. Sawada, and S. Sugano (2010). Socio-Economic Studies on Suicide: A Survey. *Journal of Economic Surveys 26*(2), 271–306.

Cutler, D. M., E. L. Glaeser, and K. E. Norberg (2001). Explaining the Rise in Youth Suicide. In J. Gruber (Ed.), *Risky Behavior among Youths: An Economic Analysis*, pp. 219–270. University of Chicago Press.

Dahl, G. B., K. V. Løken, and M. Mogstad (2014). Peer Effects in Program Participation. *American Economic Review 104*(7), 2049–2074.

de Chaisemartin, C. and X. D'Haultfoeuille (2020). Two-way Fixed Effects Estimators with Heterogeneous Treatment Effects. *American Economic Review 110*(9), 2964–2996.

De Giorgi, G., A. Frederiksen, and L. Pistaferri (2020). Consumption Network Effects. *Review of Economic Studies 87*(1), 130–163.

Domaradzki, J. (2021). The Werther Effect, the Papageno Effect or No Effect? A Literature Review. *International Journal of Environmental Research and Public Health 18*(5), 2396.

Duflo, E. and E. Saez (2003). The Role of Information and Social Interactions in Retirement Plan Decisions: Evidence from a Randomized Experiment. *Quarterly Journal of Economics 118*(3), 815–842.

Fjeldsted, R., T. W. Teasdale, M. Jensen, and A. Erlangsen (2017). Suicide in Relation to the Experience of Stressful Life Events: A Population-Based Study. *Archives of Suicide Research 21*, 544—555.

Frimmel, W., M. Halla, B. Schmidpeter, and R. Winter-Ebmer (2022). Grandmothers' Labor Supply. *Journal of Human Resources 57*(5), 1645–1689.

Gvion, Y., Y. Levi-Belz, G. Hadlaczky, and A. Apter (2015). On the Role of Impulsivity and Decision-making in Suicidal Behavior. *World Journal of Psychiatry 5*(3), 255–290.

Halla, M., S. Pech, and M. Zweimüller (2015). The Effect of Statutory Sick Pay Regulations on Workers' Health. IZA Discussion Paper 9091, Institute of Labor Economics, Bonn, Germany.

Hamermesh, D. S. and N. M. Soss (1974). An Economic Theory of Suicide. *Journal of Polticial Economy 82*(1), 83–98.

Hansen, B., J. J. Sabia, and J. Schaller (2024). In-Person Schooling and Youth Suicides: Evidence from School Calendars and Pandemic Era School Closings. *Journal of Human Resources 59*(S), S227–S255.

Hedström, P., K. Liu, and M. K. Nordvik (2008). Interaction Domains and Suicide: A Population-based Panel Study of Suicides in Stockholm, 1991–1999 . *Social Forces 87*(2), 713–740.

Hernán, M. A. (2010). The Hazards of Hazard Ratios. *Epidemiology 21*(1), 13–15.

Hvide, H. K. and P. Östberg (2015). Social Interaction at Work. *Journal of Financial Economics 117*(3), 628–652.

Jäger, S. and J. Heining (2019). How Substitutable Are Workers? Evidence from Worker Deaths. Unpublished manuscript, Massachusetts Institute of Technology, Boston, MA.

Kapusta, N. D., U. S. Tran, I. R. Rockett, D. De Leo, C. P. Naylor, T. Niederkrotenthaler, M. Voracek, E. Etzersdorfer, and G. Sonneck (2011). Declining Autopsy Rates and Suicide Misclassification: A Cross-national Analysis of 35 Countries. *Archives of General Psychiatry 68*(10), 1050–1057.

Kremer, M. and D. Levy (2008). Peer Effects and Alcohol Use among College Students. *Journal of Economic Perspectives 22*(3), 189–206.

Ludwig, J., D. E. Marcotte, and K. Norberg (2009). Anti-Depressants and Suicide. *Journal of Health Economics 28*, 659–676.

Mann, J., A. Apter, J. Bertolote, A. Beautrais, D. Currier, A. Haas, U. Hegerl, J. Lonnqvist, K. Malone, A. Marusic, L. Mehlum, G. Patton, M. Phillips, W. Rutz, Z. Rihmer, A. Schmidtke, D. Shaffer, M. Silverman, Y. Takahashi, A. Varnik, D. Wasserman, P. Yip, and H. Hendin (2005). Suicide Prevention Strategies: A Systematic Review. *Journal of the American Medical Association 294*(16), 2064–74.

Montgomery, S. C., M. Donnelly, P. Bhatnagar, A. Carlin, F. Kee, and R. F. Hunter (2020). Peer Social Network Processes and Adolescent Health Behaviors: A Systematic Review. *Preventive Medicine 130*, 105900.

Niederkrotenthaler, T., M. Braun, J. Pirkis, B. Till, S. Stack, M. Sinyor, U. S. Tran, M. Voracek, Q. Cheng, F. Arendt, S. Scherr, P. S. Yip, and M. J. Spittal (2020). Association between Suicide Reporting in the Media and Suicide: Systematic Review and Meta-analysis. *British Medical Journal 368*.

Niederkrotenthaler, T. and G. Sonneck (2007). Assessing the Impact of Media Guidelines for Reporting on Suicides in Austria: Interrupted Time Series Analysis. *Australian & New Zealand Journal of Psychiatry 41*, 419–428.

Niederkrotenthaler, T., M. Voracek, A. Herberth, B. Till, M. Strauss, E. Etzersdorfer, B. Eisenwort, and G. Sonneck (2010). Role of Media Reports in Completed and Prevented Suicide: Werther v. Papageno Effects. *British Journal of Psychiatry 197*(3), 234–243.

Phillips, D. P. (1974). The Influence of Suggestion on Suicide: Substantive and Theoretical Implications of the Werther Effect. *American Sociological Review 39*(3), 340–354.

Pink, S., T. Leopold, and H. Engelhardt (2014). Fertility and Social Interaction at the Workplace: Does Childbearing Spread among Colleagues? *Advances in Life Course Research 21*, 113–122.

Pirkis, J., R. Blood, A. Beautrais, P. Burgess, and J. Skehan (2006). Media Guidelines on the Reporting of Suicide. *Crisis 27*(2), 82–87.

Rockett, I. R. and B. Thomas (1999). Reliability and Sensitivity of Suicide Certification in Higher-Income Countries. *Suicide and Life-Threatening Behavior 29*(2), 141–149.

Ruhm, C. J. (2000). Are Recessions Good for Your Health? *The Quarterly Journal of Economics 115*(2), 617–650.

Ruhm, C. J. (2022). Living and Dying in America: An Essay on *Deaths of Despair and the Future of Capitalism*. *Journal of Economic Literature 60*(4), 1159–1187.

Ruhm, C. J. (2024). "Despair" and Death in the United States. NBER Working Paper 32978, National Bureau of Economic Research, Cambridge, MA.

Sant'Anna, P. H. (2016). Program Evaluation with Right Censored Data. *mimeo*.

Schmidpeter, B. (2024). Does Stress Shorten Your Life? Evidence from Parental Bereavement. *Oxford Bulletin of Economics and Statistics 86*(3), 485–518.

Sun, L. and S. Abraham (2021). Estimating Dynamic Treatment Effects in Event Studies with Heterogeneous Treatment Effects. *Journal of Econometrics 225*(2), 175–199.

Thorson, J. and P. Öberg (2003). Was There a Suicide Epidemic After Goethe's Werther? *Archives of Suicide Research* 7(1), 69–72.

Trogdon, J. G., J. Nonnemaker, and J. Pais (2008). Peer Effects in Adolescent Overweight. *Journal of Health Economics 27*(5), 1388–1399.

Wang, L. C. (2016). The Effect of High-stakes Testing on Suicidal Ideation of Teenagers with Reference-dependent Preferences. *Journal of Population Economics 29*(2), 345–364.

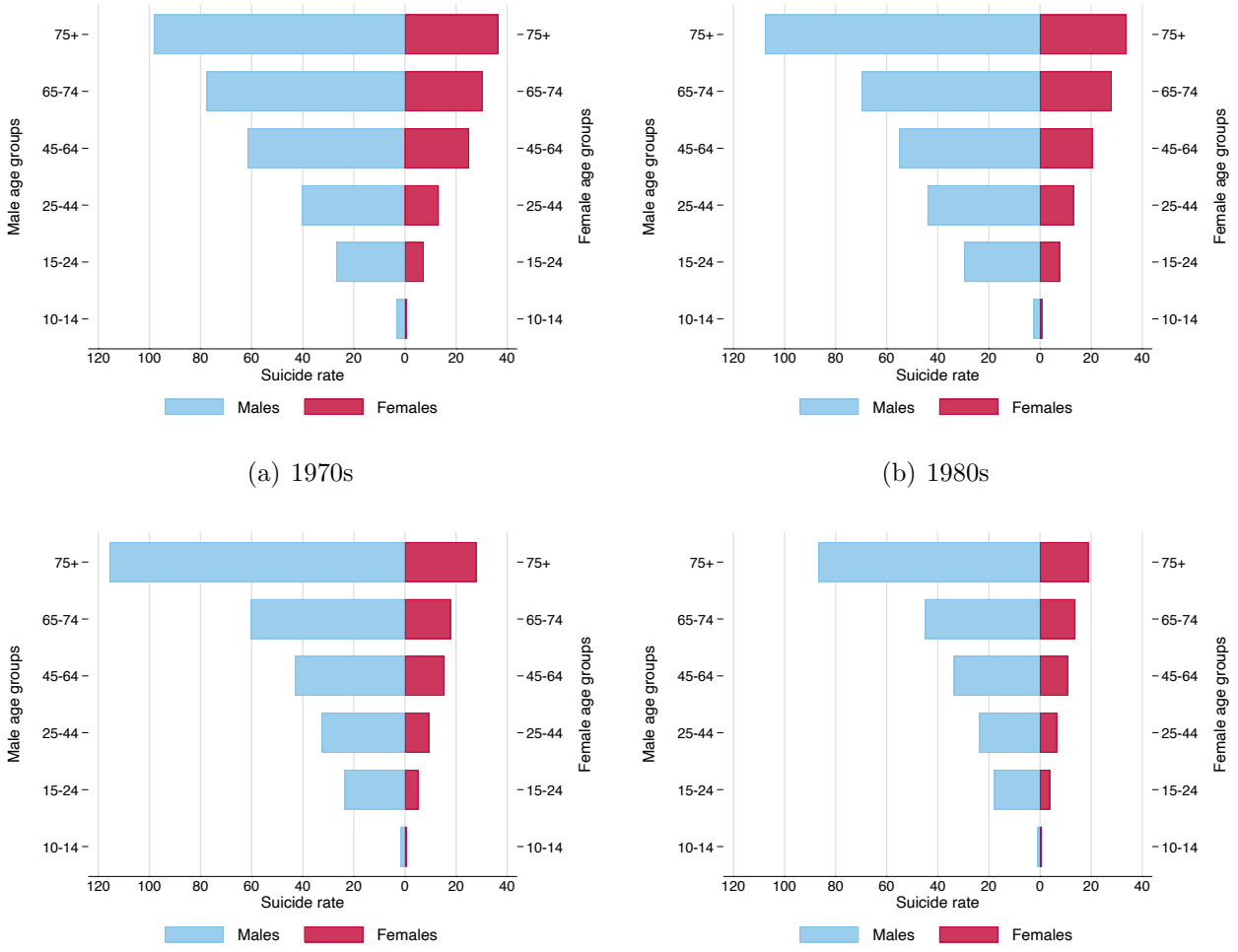
Yu, J., D. Yang, Y. Kim, M. Hashizume, A. Gasparrini, B. Armstrong, Y. Honda, A. Tobias, F. Sera, A. Vicedo-Cabrera, H. Kim, C. I. niguez, E. Lavigne, M. Ragettli, N. Scovronick, F. Acquaotta, B. Chen, Y. Guo, M. de Sousa Zanotti Stagliori Coelho, P. Saldiva, A. Zanobetti, J. Schwartz, M. Bell, M. Diaz, C. De la Cruz Valencia, I. Holobâcă, S. Fratianni, and Y. Chung. (2020). Seasonality of Suicide: A Multi-country Multicommunity Observational Study. *Epidemiology and Psychiatric Sciences 129*, e163.

Zweimüller, J., R. Winter-Ebmer, R. Lalive, A. Kuhn, J. Wuellrich, O. Ruf, and S. Büchi (2009). The Austrian Social Security Database (ASSD). Working Paper 0901, The Austrian Center for Labor Economics and the Analysis of the Welfare State, Linz, Austria.

Web appendix

This Web Appendix provides additional material discussed in the unpublished manuscript "Werther at Work: Intra-firm Spillovers of Suicides" by Martin Halla and Bernhard Schmidpeter.

Figure A.1: Sex and age-specific suicide rates by decades



(a) 1970s

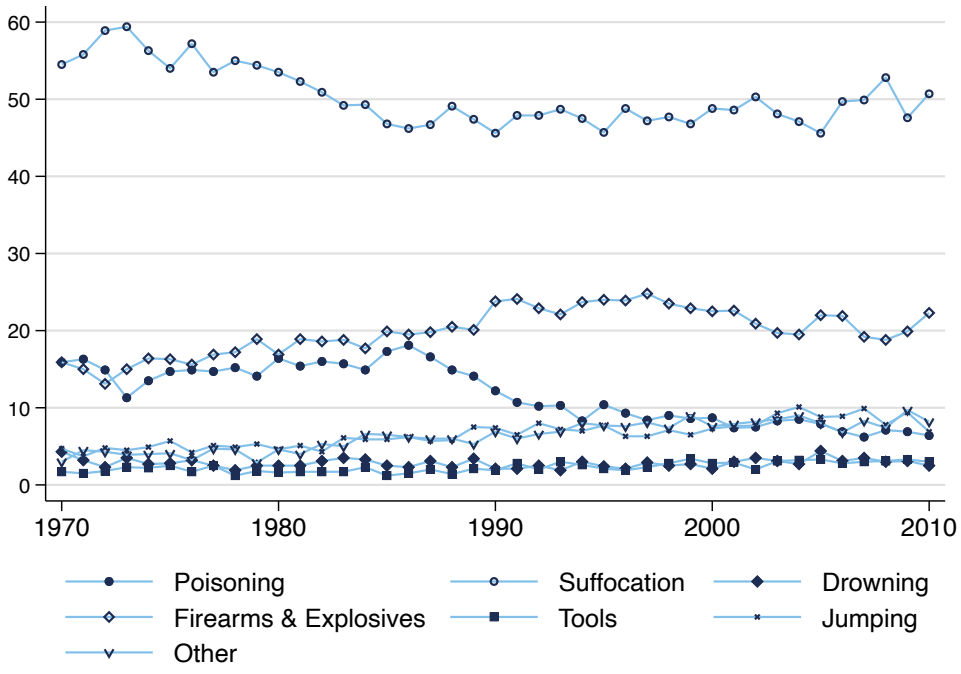
(b) 1980s

(c) 1990s

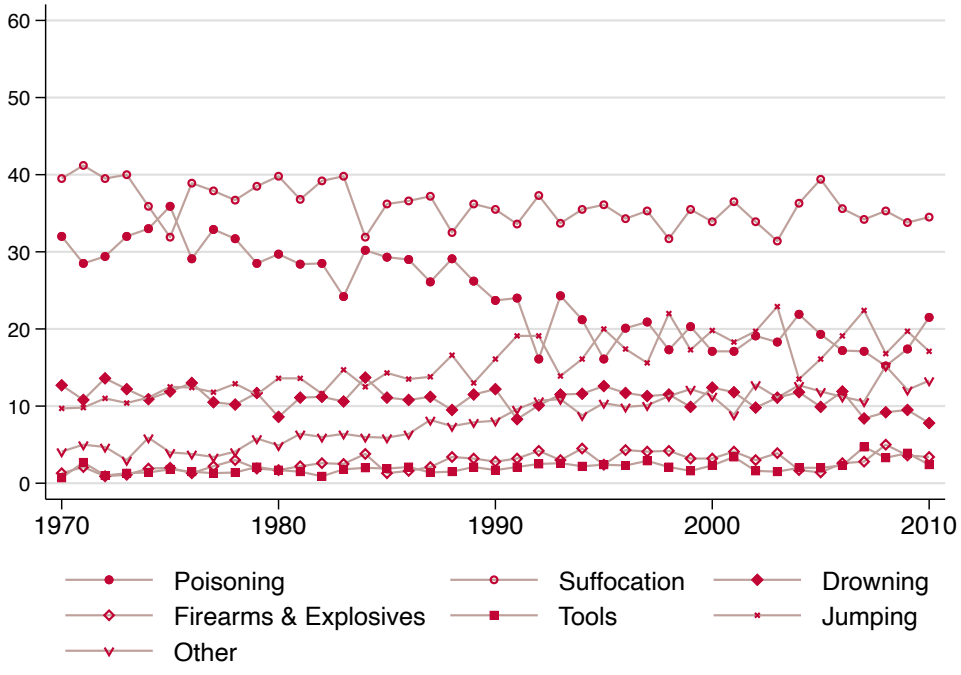
(d) 2000s

Notes: Calculations are based on the *Austrian Death Register*. Suicide rates are defined as the number of persons who died by suicide per 100,000 population. The sex- and age-specific population for the years 1972 to 1980 is based on a linear interpolation between the years 1971 and 1981. Panel (a) to Panel (d) show the sex- and age-specific suicide rates by decade. In each case, the bars on the left are for men and the bars on the right are for women.

Figure A.2: Distribution of suicide method, by sex



(a) Men



(b) Women

Notes: Calculations are based on the *Austrian Death Register*.

Table A.1: Matching between Austrian Death Register (ADR) and Austrian Social Security Database (ASSD)

	(1)	(2)	(3)	(4)
	Match between ADR → ASSD		**Match between ASSD → ADR**	
	Match rate = 66.95%		Match rate = 71.49%	
	Matched	Non-Matched	Matched	Non-Matched
Age at death	75.77	71.84	76.46	77.91
	(14.43)	(19.56)	(13.23)	(10.79)
Year of Death	1996.60	1989.98	1996.62	1993.77
	(8.14)	(8.99)	(8.13)	(9.91)
Female	52.81	54.37	53.13	50.64
Austrian	98.20	96.26	98.83	95.93
Catholic	78.15	81.20		
Individuals	1,696,724	837,753	1,672,255	666,884

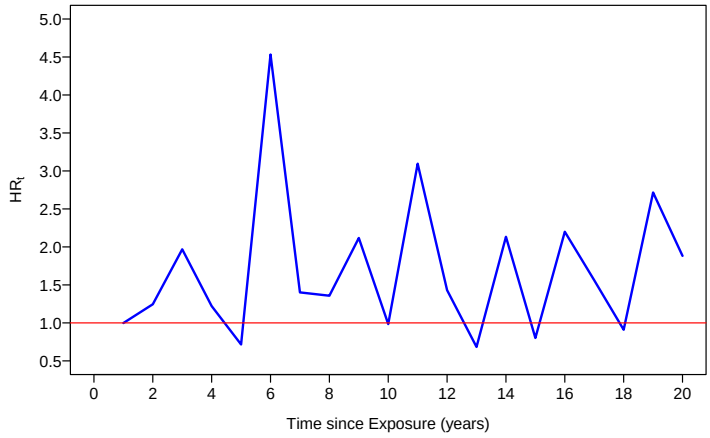
Notes: The table provides summary statistics for individuals. The first two columns pertain to all cases in the *Austrian Death Register* (ADR) from 1980 to 2010. Column (1) includes individuals successfully matched between the death register and the *Austrian Social Security Database* (ASSD), with a match rate of 66.95%. Column (2) covers individuals who could not be matched. These individuals were either not employed or could not be uniquely identified due to the absence of a unique identifier. The next two columns focus on all cases in the ASSD of individuals who died between 1980 and 2010. Column (3) includes individuals successfully matched between the ASSD and the ADR, with a match rate of 71.49%. Column (4) covers individuals who could not be matched. These individuals either emigrated from Austria and died abroad or could not be uniquely identified due to the absence of a unique identifier. † Notice that labor market outcomes are available for individuals in the ASSD only while reason of death is only available from the death register. Note that earnings of individuals who are alive, but not observed in the ASSD are recorded as zero. In case of that these individuals left Austria this assumption would be incorrect and underestimated their true earnings.

Table A.2: Correlation between suicide as a finl cause of death and autopsy

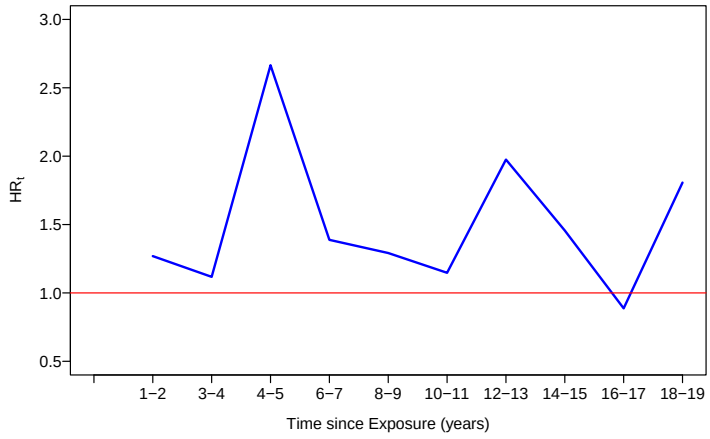
	(I)	(II)	(III)	(IV)
Autopsy	−0.019***	−0.020***	−0.020***	−0.020***
	(0.000)	(0.000)	(0.000)	(0.000)
Constant	0.065***	−0.080***	−0.086***	−0.087***
	(0.006)	(0.008)	(0.008)	(0.008)
Sex	Yes	Yes	Yes	Yes
Age at death	Yes	Yes	Yes	Yes
Year of death	Yes	Yes	Yes	Yes
No. of observations	1,921,736	1,921,736	1,921,736	1,921,736
Mean of outcome	0.021	0.021	0.021	0.021
R-squared	0.073	0.077	0.078	0.078

Notes: This table summarizes the results of a regression analysis using data from the *Austrian Death Register* between 1984 and 2007. The estimation method is OLS. The dependent variable is a binary indicator for suicide. The independet variable of primary interest is a binary indicator for autopsy. Robust standard errors are shown in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Figure A.3: Impact of exposure to co-worker suicide on suffering from suicide: Period-by-Period Hazard Rate



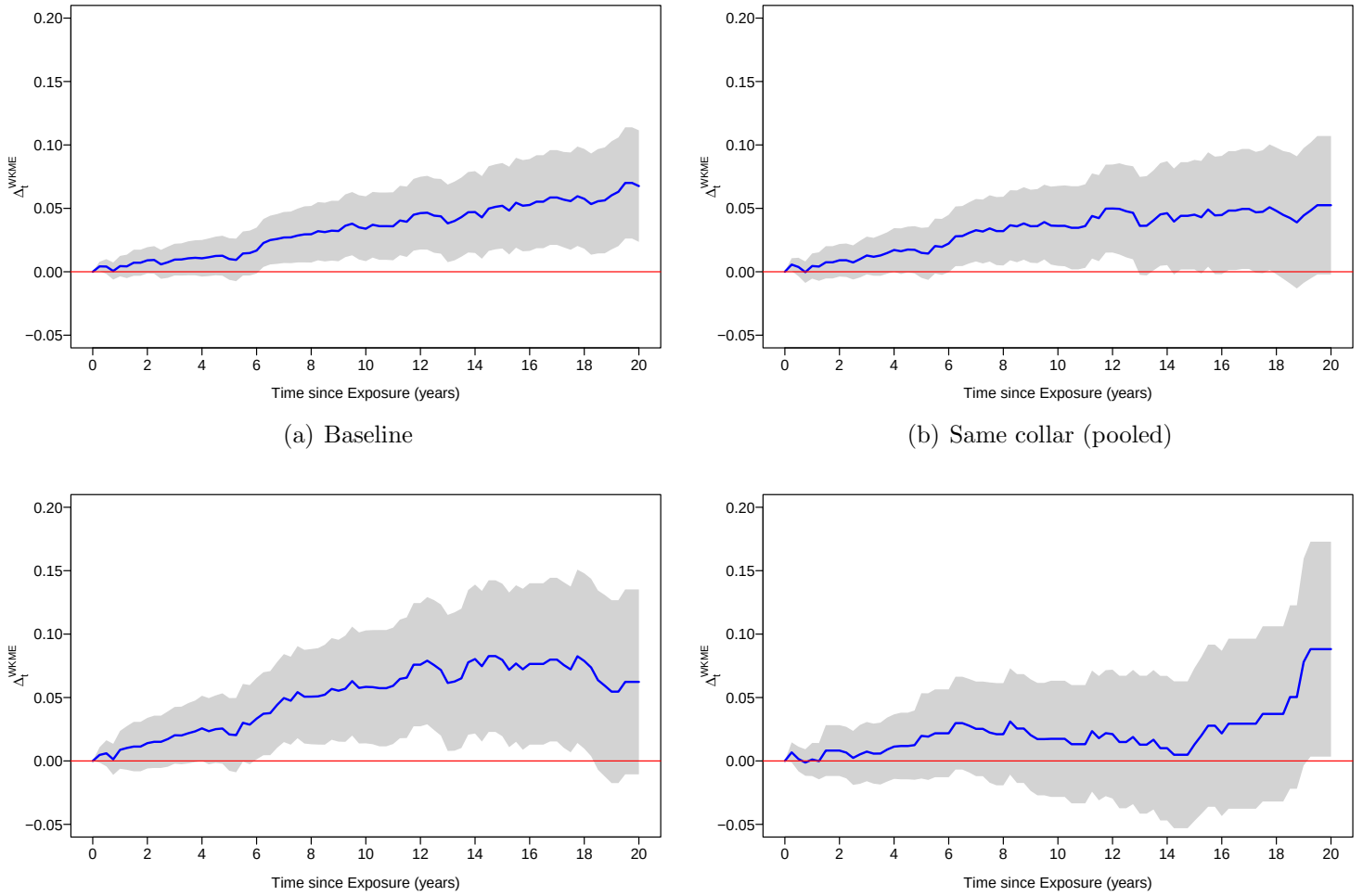
(a) 1 year interval



(b) 2 year interval

Notes: The figure plots the estimated period-by-period hazard ratios in Equation 7. To ensure stable estimates, different time periods are pooled together. Panel (a) shows the period-by-period hazard ratio when pooling effects over 1 year intervals and Panel (b) for a 2 year interval.The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 151,373 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 171,830 workers. See Section 4 for details on the exact matching procedure.

Figure A.4: Collar-specific impacts of exposure to co-worker suicide on suffering from suicide: Weighted Kaplan-Meier Estimates



(a) Baseline

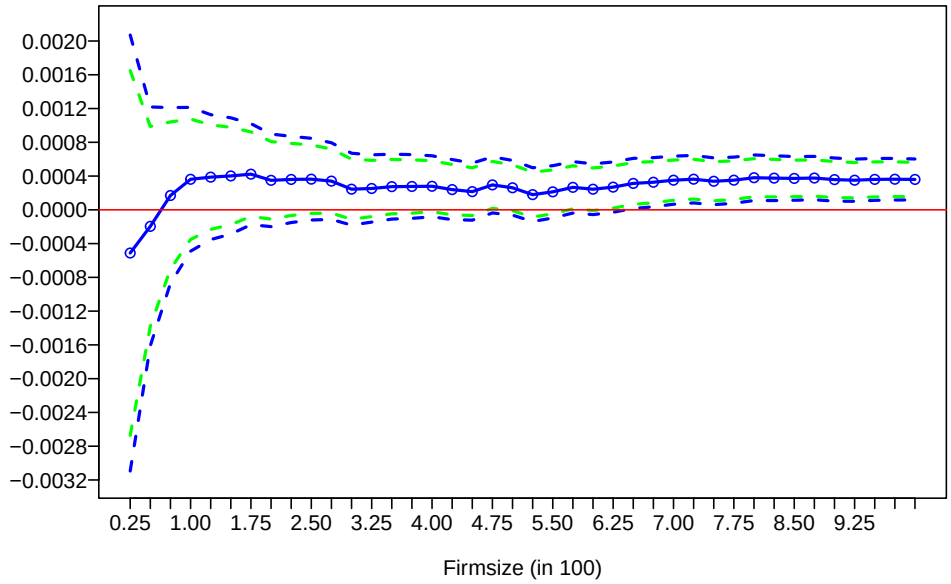
(b) Same collar (pooled)

(c) Blue collar

(d) White collar

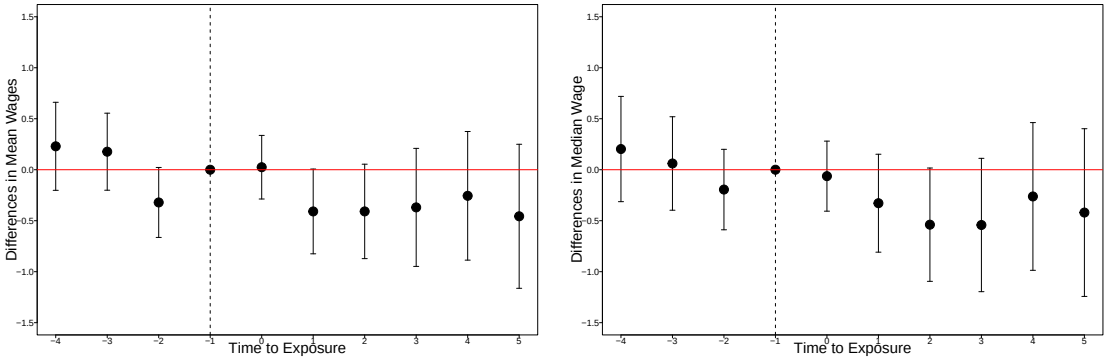
Notes: The figure plots the estimated coefficients Δ_t^{WKME} from the weighted Kaplan-Meier Estimator in equation 6 when exposed to a co-worker's suicide of the same collar. The depicted effect shows the *cumulative* effect of being exposed to a co-worker's suicide on the probability of dying from suicide. Shaded areas correspond to 95% confidence intervals, obtained from a non-parametric bootstrap with 1,000 replications. Panel (a) shows the baseline estimates. Panel (b) pools the collar estimates together. Likewise, results in Panel (c) and (d) are based on only blue collar and white collar workers respectively.

Figure A.5: Impact of exposure to co-worker suicide on suffering from suicide: Effect by firm-size



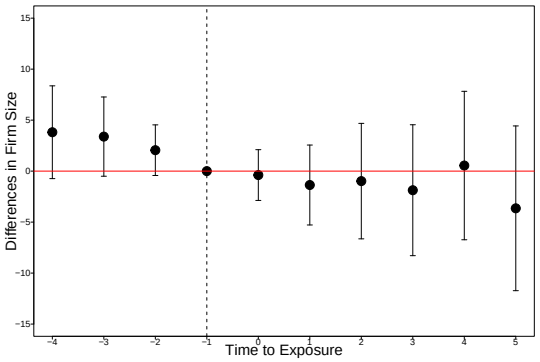
Notes: The figure plots the estimated coefficients γ from separate regressions based on equation 2, with the sample split by firm size using an interval length of 25. The blue outer dashed lines represent 95% confidence intervals and the inner green dashed lines 90% confidence intervals.The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 151,373 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 171,830 workers. See Section 4 for details on the exact matching procedure.

Figure A.6: Impact of exposure to co-worker suicide on firm outcomes: Firm-level event-study results



(a) Mean wages

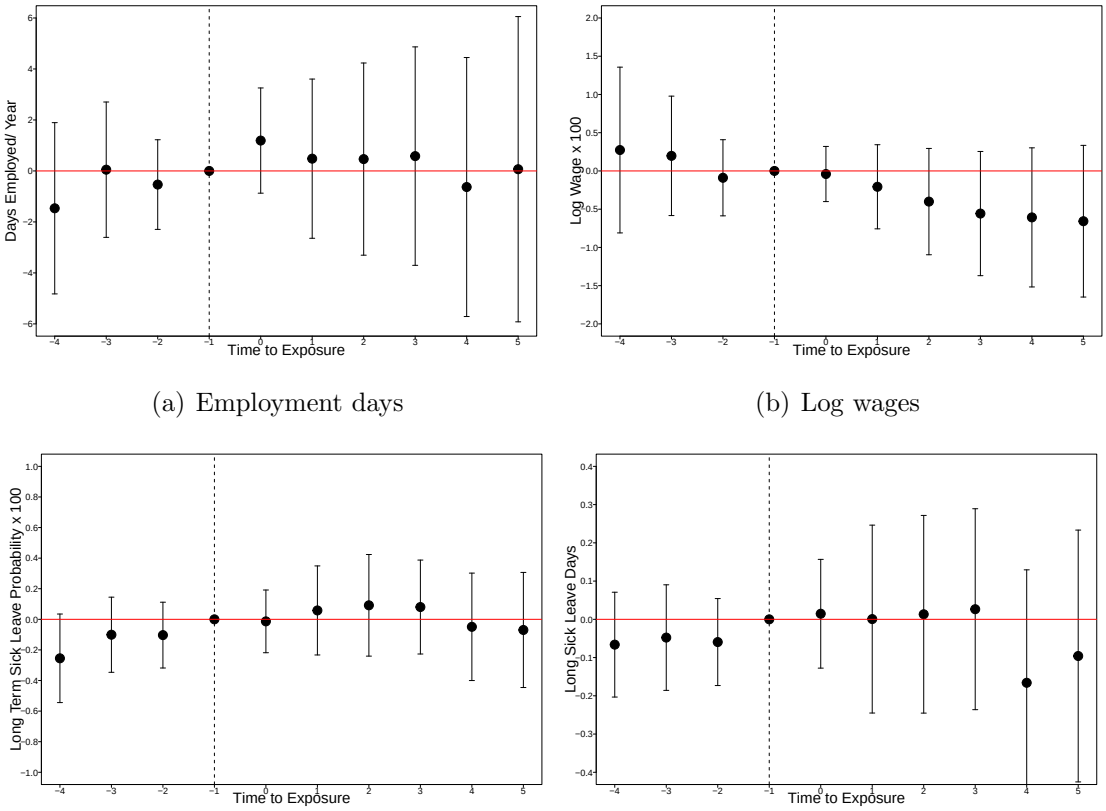
(b) Median wages



(c) Firm size

Notes: The figure plots the estimated coefficients δ_r from an event-study specification similar to Equation 1, applied to firm-level outcomes. Vertical lines show 95% confidence intervals, obtained from standard errors clustered at the firm level. Panel (a) and (b) use mean and median daily firm wages as outcome, respectively. Panel (d) plot the estimated coefficients using firm size where the bottom and top 5% ob observations are winsorized. The exposed sample of firms consists of all firms where at least one worker aged 15 to 65 was exposed to a suicide of a co-worker (treated); in total 1,259 firms. The non-exposed group of firms comprises a similar set of firms employing comparable workers, but who were not exposed to a suicide (control); in total 1,259 firms. See Section 4 for details on the exact matching procedure. P-values from a Wald test for joint significant of pre-expousre coefficients are as follows: mean wages ($p = 0.02$); median wages ($p = 0.36$); and firm size ($p = 0.32$).

Figure A.7: Impact of exposure to co-worker suicide on labor market outcomes: Event-study results for white-collar workers



(a) Employment days

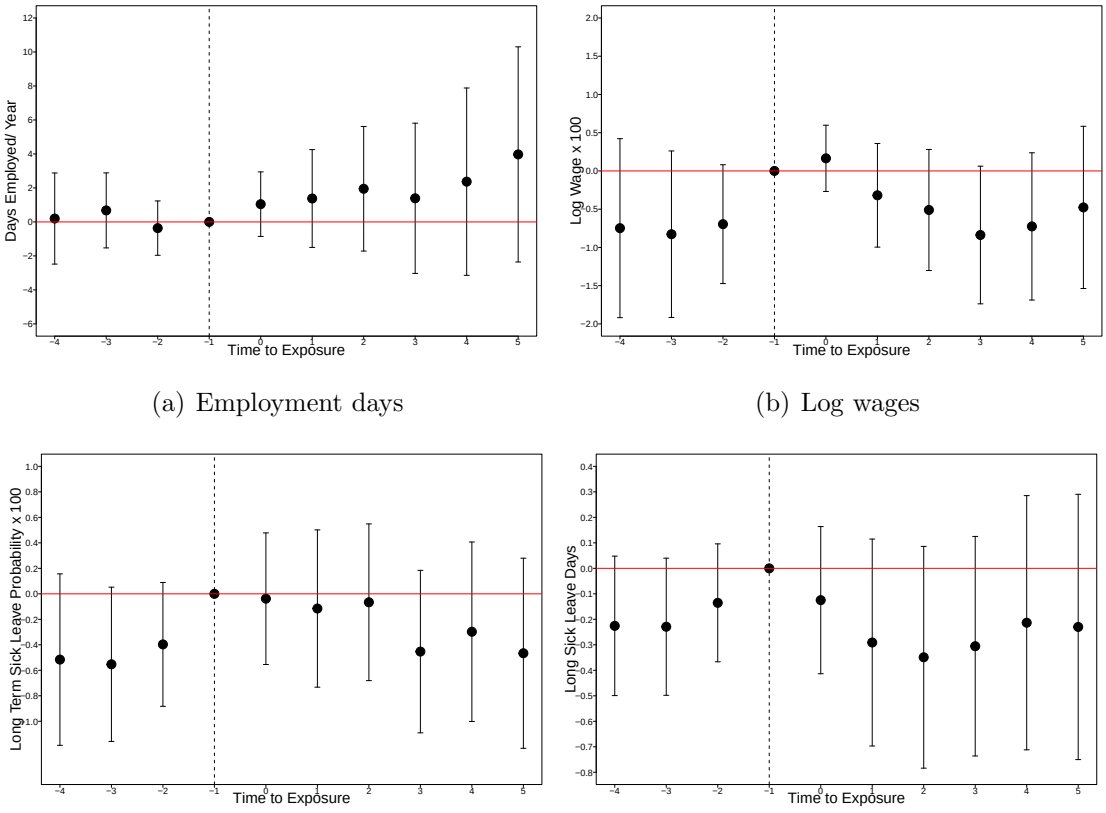
(b) Log wages

(c) Long sick leave (0/1)

(d) Long sick leave days

Notes: The figure plots the estimated coefficients δ_r from the event-study specification in Equation 1 for white collar workers only. Vertical lines show 95% confidence intervals, obtained from standard errors clustered at the firm level. Panel (a) and (b) use yearly employment days and log daily wages as outcome respectively. Panel (c) uses a binary indicator whether the individual had a long-term sick leave spell in a given year. Panel (d) plot the estimated coefficients when using the total long-term sick leave days as outcome. The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death as white-collar worker, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 68,966 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 82,417 workers. See Section 4 for details on the exact matching procedure. P-values from a Wald test for joint significant of pre-expousre coefficients are as follows: employment days ($p = 0.25$); log wages ($p = 0.59$); long sick leave (0/1) ($p = 0.36$); and long sick leave days ($p = 0.70$).

Figure A.8: Impact of exposure to co-worker suicide on labor market outcomes: Event-study results for blue-collar workers



(a) Employment days

(b) Log wages

(c) Long sick leave (0/1)

(d) Long sick leave days

Notes: The figure plots the estimated coefficients δ_r from the event-study specification in Equation 1 for white collar workers only. Vertical lines show 95% confidence intervals, obtained from standard errors clustered at the firm level. Panel (a) and (b) use yearly employment days and log daily wages as outcome respectively. Panel (c) uses a binary indicator whether the individual had a long-term sick leave spell in a given year. Panel (d) plot the estimated coefficients when using the total long-term sick leave days as outcome. The exposed sample includes all workers aged 15 to 65 who met the following criteria: they were employed at the time of the co-worker's death as blue-collar worker, were employed four years prior to the death date, worked in firms with no more than 1,000 employees, had no long-term sickness spells in the year preceding the death, and were exposed to the suicide of a co-worker (treated group). This sample comprises a total of 82,285 workers. The non-exposed group consists of matched workers with comparable characteristics who were employed in similar firms but were not exposed to a coworker's suicide (control group); totaling 89,193 workers. See Section 4 for details on the exact matching procedure. P-values from a Wald test for joint significant of pre-expousre coefficients are as follows: employment days ($p = 0.47$); log wages ($p = 0.37$); long sick leave (0/1) ($p = 0.32$); and long sick leave days ($p = 0.37$).

Table A.3: Balancing checks of individual characteristics: Early leavers

	Early Leavers	
	Placebo exposed	Placebo non-exposed
Age	31.97	33.03
	(12.23)	(12.45)
Female	27.09	30.59
High education	29.93	26.67
Medium education	38.66	39.19
Any long-term sickness spell	16.75	15.28
Long-term sickness days	8.95	8.15
Blue collar worker	77.49	75.11
Daily wage (in Euros)	72.33	69.79
	(43.33)	(31.79)
Individuals	20,524	24,685

Notes: The table shows summary background information for the placebo exposed group and the placebo non-exposed group. The first group consists of workers employed at the treated firms, but who left the firm between 1 and 4 quarters prior to the exposure quarter. The second group consists of workers, who left a non-deceased (=control) firm between 1 and 4 quarters prior to the placebo non-exposure quarter. Standard deviations are only shown for non-binary variables.