The fundamental causes of social inequalities in suicide

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Background: Suicide accounts for approximately 37,000 deaths in the United States every year, but recent research suggest that Selective Serotonin Reuptake Inhibitor (SSRI) use may reduce suicidal impulses and behaviors. Fundamental cause theory (FCT) posits that following an innovation in treatment or prevention, individuals with more resources, including education, money, power, prestige, and beneficial social connections, preferentially gain access to those advances.

Methods: We analyze county-level age, sex, and race-specific death-certificate and population data collated by the National Centers for Health Statistics from 1968 to 2009. We use data from the U.S. decennial census to estimate socioeconomic status at the county level and include those age 25 and over. We use negative binomial regression and change-point models on 40 years of population-level suicide death data.

Results: We find that suicide rates have been declining since the introduction of SSRIs in 1988. We also find that since 1988, social inequalities in suicide have been rising.

Conclusions: SSRIs appear to be an effective prevention for suicide at the population level. However, as predicted by FCT, after SSRIs were introduced, there was a substantial rise in socioeconomic inequalities in suicide.

Approximately 37,000 deaths each year in the United States (U.S.) are due to suicide. It is the tenth most prominent cause of death and in 2009, killing three times as many people as homicide, and accounts for around 1.5% of all-cause mortality among individuals over ten years old.[1] The likelihood of committing suicide is distributed differently across sex, age, and race/ethnicity, with higher rates of suicide mortality being reported among men, older individuals, and Whites. It is not, reliably related to social and economic inequalities: though some studies have shown that income and occupational disparities between racial groups may predict higher suicide rates in lower SES groups,[2] stressful life factors like occupational, financial, and/or familial strife often act as triggers and thus suicide is often seen as particularly likely among higher socioeconomic status individuals.[3]

At the individual level, 90% of all suicides are associated with psychiatric illnesses, particularly depression.[4,5] Selective Serotonin Reuptake Inhibitors (SSRIs) have been shown to to reduce both the number and severity of depressive symptoms, thereby preventing the likelihood of experiencing suicidal thoughts as well as subsequent action.. A recent systematic review suggests that SSRIs are likely to provide a strong benefit in the prevention of suicide and may reduce the risk of suicide by as much as 40% for individuals over 25 years of age,[6,7] There has been a substantial push towards using SSRIs to treat depression, which has been accompanied by steadily declining suicide rates since SSRIs became available.[8]

Fundamental cause theory (FCT) posits that socioeconomic inequalities in health arise, in part, because individuals and groups marshal all of the resources at their disposal, including knowledge, money, power, prestige, and beneficial social connections, to secure access to effective preventive and therapeutic advances.[9-18] Suicide prevention is most effective when it is appropriately targeted and is efficiently distributed, and socioeconomic inequalities in suicide could arise when individuals seek different kinds of care, at different rates, and at different times.[19] In particular, timeliness is important. For 24% of suicides, the time spent between suicidal thoughts and attempts is less than 5 minutes; for 70%, it is less than one hour.[20] However,

diagnosis and treatment of underlying psychiatric disorders or symptomology is also an essential pathway through which suicide can be prevented. Only 64% of suicidal individuals seek help.[21] Furthermore, many of those who seek help do so from institutions and healthcare providers that cannot adequately address their underlying mental health problems or provide efficacious treatment regimens.[22,23] Because of difficulties like these, research suggests among those exhibiting signs of mental health problems, treatment with SSRIs is more likely among people with higher SES.[24]

This research leads us to the following hypotheses. First, due to a change in the preventability of suicide through the use of SSRIs, there should be a change in the suicide rate starting in 1988 with the introduction of SSRIs. Second, FCT hypothesizes that there will be an increase in the impact of SES on suicide following the advent of SSRIs, with rates of suicide decreasing most dramatically among high SES individuals who possess the resources to purposefully gain access to the most recent and efficacious medical advances in the treatment of depression.

Data

We combined 40 years of death certificate data for the entire U.S. that was obtained from the National Center for Health Statistics with age-, sex-, race-, and county-specific population estimates originally generated by the Census Bureau. [25] Deaths are attributed to place of residence as opposed to place of occurrence; non-resident U.S. citizens were excluded from analysis. We include information on 99% of counties in the continental U.S. and Hawaii. We exclude counties whose borders were substantially altered over the time period under study for a final n = 3,110 counties between 1968 and 2009 (124,400 county-years). We identified those who died from suicide using International Classification of Disease (ICD) categories indicating death due to intentional self-harm (ICD-8 & 9: E950-E959; 10: X60-X84 & Y87.0). We limit our sample to adults aged 25 and over because the use of SSRIs has been linked to an increased likelihood of suicide for children and adolescents.[26] We include information for Blacks and Whites, but we

exclude data from 'other' races due to relatively small cell sizes for the outcome of interest and a lack of comparability in racial categories over time. Using census data, we created an aggregate measure of SES for all counties in the U.S. based on five distinct variables: the proportion of individuals in each county with fewer than nine years of education, more than twelve years of education, white-collar occupations, indoor plumbing, or telephone access in their homes.[27] We combined these measures using factor analysis, which yielded a single factor solution, and linearly interpolated data for inter-censal years. To adjust for secular change potentially impacting suicide mortality, we include yearly measures of urbanicity, which measures the proportion of the county that is rural, and a dichotomous indicator for years with stock market crashes. To provide trends in SSRI use, we used data from the National Health and Nutrition Examination Survey (NHANES) from 1988-2010. All analysis is done using Stata 11/IC.

Methods

We expect that suicide rates will decrease substantially following the introduction of SSRIs in January 1988 (t_{pk}). This change in rates of change over time can be specified using "change-point" models, which suggests using a piecewise-linear modification variable to model change in suicide rates following a particular point in time (e.g. the introduction of SSRIs in 1988).[28] The change covariate therefore equals 0 for years prior to 1988 and then enumerates years after; a significant change variable suggests that the yearly change differs from the yearly rate of change prior to this point.

For descriptive analyses, we calculate age-adjusted suicide rates, weighted to the World Health Organization's (WHO) standard population. We provide analysis in the entire sample and separate analysis into three groups cut at one standard deviation above and below population average. We then use ordinary least squares regression to overlay a piecewise-linear rate of change in suicide mortality.

For multivariate analyses, we rely on negative binomial regression to predict suicide mortality rates at the county-level. Negative binomial regression is preferable to Poisson

regression when data are over-dispersed, which occurs when the variance is larger than the mean.[29] Midyear age-, sex-, and race-specific population counts for each county are used to capture the population at risk of exposure to death by suicide. We use Huber-White standard errors and adjust for clustering at the county level. For ease of interpretation, we provide mortality rate ratios (MRRs). An MRR of 1.05 suggests that one unit of change in the variable of interest is associated with a 5% increase in suicide.

We provide a baseline model (Model 1) adjusting for race, sex, urbanicity, and age. We then include a second model that adjusts solely for SES (Model 2). Finally, we incorporate an interaction between SES and time after 1988 (Model 3). To compare models we use the relative likelihood (R_L), estimated using the Aikake information criteria (AIC), to compare negative binomial models. R_L estimates the probability that the model fits the data significantly better than a comparison model: a relative likelihoods less than 0.05 suggests that the new model significantly improves on the earlier model.[30]

Results

Figure 1 provides the U.S. average age-, sex-, and race-adjusted suicide rate across the 41-year time period of interest for those aged 25 and over. We find that suicide rates were decreasing at a rate of -0.005 suicides per 10,000 in the years prior to 1988 ($p = 0.167$). Following 1988, suicide rates declined at a rate of -0.012 per 10,000 ($p < 0.05$).

[Figure 1]

To model the change in suicide in a multivariate context, we use these change points in a negative binomial regression analysis to predict county-specific suicide rates (Table 1). All models adjust for age, sex, race, stock market crashes, and urbancity. Model 1 shows that there is a significant change in suicide rates over time, with pre-SSRI rates decreasing on average at a rate of 0.6% per year, and post-SSRI change estimated to decrease at a rate of (1 - 0.994*0.998 =) 0.8% per year, a significant increase ($p = 0.017$). Similarly, SES appears to be generally

protective: a one standard deviation increase in SES results in a reduction in the risk of suicide (MRR = 0.966, $p = 0.009$).

Next we incorporate an interaction between SES and years after the introduction of SSRIs (Model 2), resulting in a model that the AIC suggests is preferable to model 1 ($R_L < 0.05$). Modeling the influence of SES over time (Model 2), we find that SES has lost its significant influence on mortality rates ($p = 0.786$). The average post-SSRI change has reduced, but remains significant ($p = 0.023$). However, following the introduction of SSRIs in 1988, the protective influence of SES on suicide mortality increases by (1-0.995*100% =) 0.5% per year after 1988. By the end of the study period, living in areas with one standard deviation higher SES is associated with a 10.0% reduction in suicide.

[Table 1]

Finally, we use the results shown in Table 1 Model 2 above to provide population averages estimating the relative impact of SES following the introduction of SSRIs. In the post-SSRI era, there is an average decrease in suicide mortality; however, this decrease is pronounced amongst those living in counties with higher SES. Specifically, suicide risk diverges substantially depending on SES following the introduction of SSRIs.

[Figure 2]

Sensitivity analyses

We analyzed the relevance of using a "random" change point at the state level, which uses an optimization routine to specify a different change point for each state. This type of analysis assumes that SES works in part through selective diffusion and implementation of new innovations across geographical areas.[31] Doing so results in a better model ($R_L < 0.05$), which also shows an increase in the impact of SES on post-SSRI change in suicide (MRR = 0.991, $p < 0.001$).

Limitations

We rely on aggregate data at the county level to conduct the analyses presented here. Therefore, we cannot examine individual-specific behaviors, such as alcohol intake or the use of psychotherapy, that might also impact suicide; nor can we causally link SSRI use to the broad reduction in suicide. At the same time our results document that circa 1988 a characteristic of the environmental milieu changed for residents of high SES counties that was correlated with significantly lower suicide rates.

Our data include mortality counts generated from death certificates, which are susceptible to misclassification of suicide deaths. In particular, studies have shown that among Catholics, whose faith has traditionally characterized suicide as a sin, there is an underreporting of suicide and a concomitant over-reporting of accidental deaths. Insofar as bias arises from misidentification of suicide, it is likely to be stable over time unless values are changing; if traditional values may have waned over time, under-reporting of suicides might also have diminished and we would expect a rise in suicides, an empirical result that we did not observe.[3]

Finally, consistent with past research, our results show that prior to the to the late 1980s SES was not predictive of suicide. But following 1988 and coincident with the widespread distribution of the SSRIs, which have been shown to reduce suicide,[4] inequalities were produced. We cannot causally identify which individuals were taking SSRIs, nor can we assess the relevance of SSRI use to suicide mortality. However, we are not aware of any other explanation for such a clear dive in the suicide rate in combination with an increase in the role of SES in suicide mortality in this particular time frame.

Discussion

We sought to examine how social inequalities in suicide change in relation to the timing of the introduction and dissemination of SSRIs to treat depression and prevent suicide. We first replicated earlier results showing a decline in suicide following the introduction of SSRI use. We also found that socioeconomic inequalities played an increasingly important role in suicide reductions. In 2009, residents of high SES counties (1 standard deviation above the mean) faced a

10% lower risk of suicide mortality than residents of low SES counties (1 standard deviation below the mean). In addition, we provide evidence that these inequalities arose following the diffusion of SSRI treatments for depression, which began in 1988. Our results suggest that understanding the unique history of suicide is important when trying to understand why health disparities fluctuate over time.

Depression, a driving force behind suicidal behaviors, is a psychiatric disorder that remains under-diagnosed and under-treated in the United States.[19] This condition is typically treated using a variety of approaches including psychotherapy and pharmacologic treatment with SSRIs.[32] Recent research has highlighted the effectiveness of SSRIs use in preventing suicide by reducing suicidal thoughts and the risk of suicidal behaviors.[6-8] Our results demonstrate that following the widespread introduction of SSRIs to treat depression in 1988, suicide mortality among adults 25 years and older decreased annually by 0.6% per year.

Since Durkheim's seminal thesis, social scientists have primarily focused on the role of social integration in influencing an individual's risk of suicide. While suicide is more or less prevalent among certain demographic groups, up until very recently, the suicide rate has been generally stable.[33] Our results suggest that future analyses may find increasingly substantial social inequalities in suicide mortality, as some individuals access SSRI treatments while others do not. If SSRI treatment is important to reducing the risk of suicide, as we have suggested it is, future analysis, especially at the individual level, may help us to more efficiently target treatments, shape policy responses, and identify people facing a greater risk of suicide.

FCT posits that when effective preventions are found, individuals and groups with greater resources will secure access to those preventions in a timelier and more effective manner than those with fewer resources. FCT therefore implies that cause-specific mortality is *particularly susceptible* to social inequality when individuals can use resources to effectively impact health, disease, and death.[9,17,18,31] Indeed, when seeking help for depression, individuals with more resources tend to access psychiatric professionals over primary care physicians or social

workers.[19] Psychiatric professionals are trained to identify depression, a main predictor of suicide,[4,5] and to treat it using SSRIs. Our results lend further support to the FCT. Namely, that socioeconomic inequalities in suicide, though not existent in our data or in the literature prior to the 1980s, emerged following the introduction of SSRIs in 1988,[3] when suicide rates substantially declined in high SES locales.

To assess acceleration in the rate of change in suicide mortality over time, we employed innovative methods to discern trends throughout a 40-year time period using population-level data. In particular, we found that using a "change-point" model (also called a "join-point" model[34]) aided in our ability to understand how social inequalities fluctuate over time, especially in reference to a transition in capacity to prevent mortality. Change-point models fit a piece-wise linear acceleration curve and were originally conceptualized to understand differences in the rapidity of aging prior to disease diagnosis or death.[28,35] We found that the change-point was situated in 1988, the year that widespread introduction of SSRIs into the U.S. population as a treatment for depression. However, sensitivity analyses showed that this date depended somewhat on state-level contexts: this change point was located later in some states (e.g. Arkansas) than others (e.g. California). Prior research has suggested that diffusion of innovations may differ by state-level policy in a way that affects distribution of new preventative information;[31] however, cultural and contextual factors relating to the speed of diffusion may be equally important.

Suicide prevention is most effective when it is appropriately targeted. One problem that hampers efforts to reduce suicide mortality is the timing of initiation. For 24% of suicides, the time spent between decision and attempted to take one's own life is less than 5 minutes; for 70% it is less than an hour.[20] Given such a short interval between the decision to commit suicide and when an individual takes action, an effective prevention strategy must reduce suicidal ideation and/or behaviors in a timely manner and must be provided to those at risk of developing suicidal behaviors. While most people who commit suicide do seek professional help in the month leading up to their death, few who are seen by healthcare professionals and physicians tend to be

prescribed SSRIs or are referred to mental health professionals for treatment.[22,23] For instance, in the Netherlands where health insurance is universal, 64% of those with mood disorders sought help, but of these, only 34% received help from mental health practitioners while 54% were seen by primary care physicians.[21] Preventing suicide requires quick identification of individuals at risk of suicide followed by immediate treatment.

Conclusion

Social inequalities in all-cause mortality are increasing.[36] FCT is alone in positing that social inequalities arise because individuals and groups actively use their resources to influence survival when effective preventions and treatments are available. Suicide has recently become more preventable with the advent of SSRIs for the treatment of mood disorders, including depression. We used FCT to show that socioeconomic inequalities in suicide, which did not exist prior to the advent of SSRIs, are rapidly increasing. This is in line with prior research on FCT across a wide range of health outcomes that explicitly demonstrates social inequalities do not arise *despite* but rather *because of* human intervention.

Figure 1. Yearly average -, sex-, and race-adjusted suicide rates (circles) overlaid with marginal estimates of trends in suicide rates using regression on mean age-, sex-, and race-adjusted suicide rates with national trends in SSRI use, NCHS 1968-2009 & NHANES 1987-2009

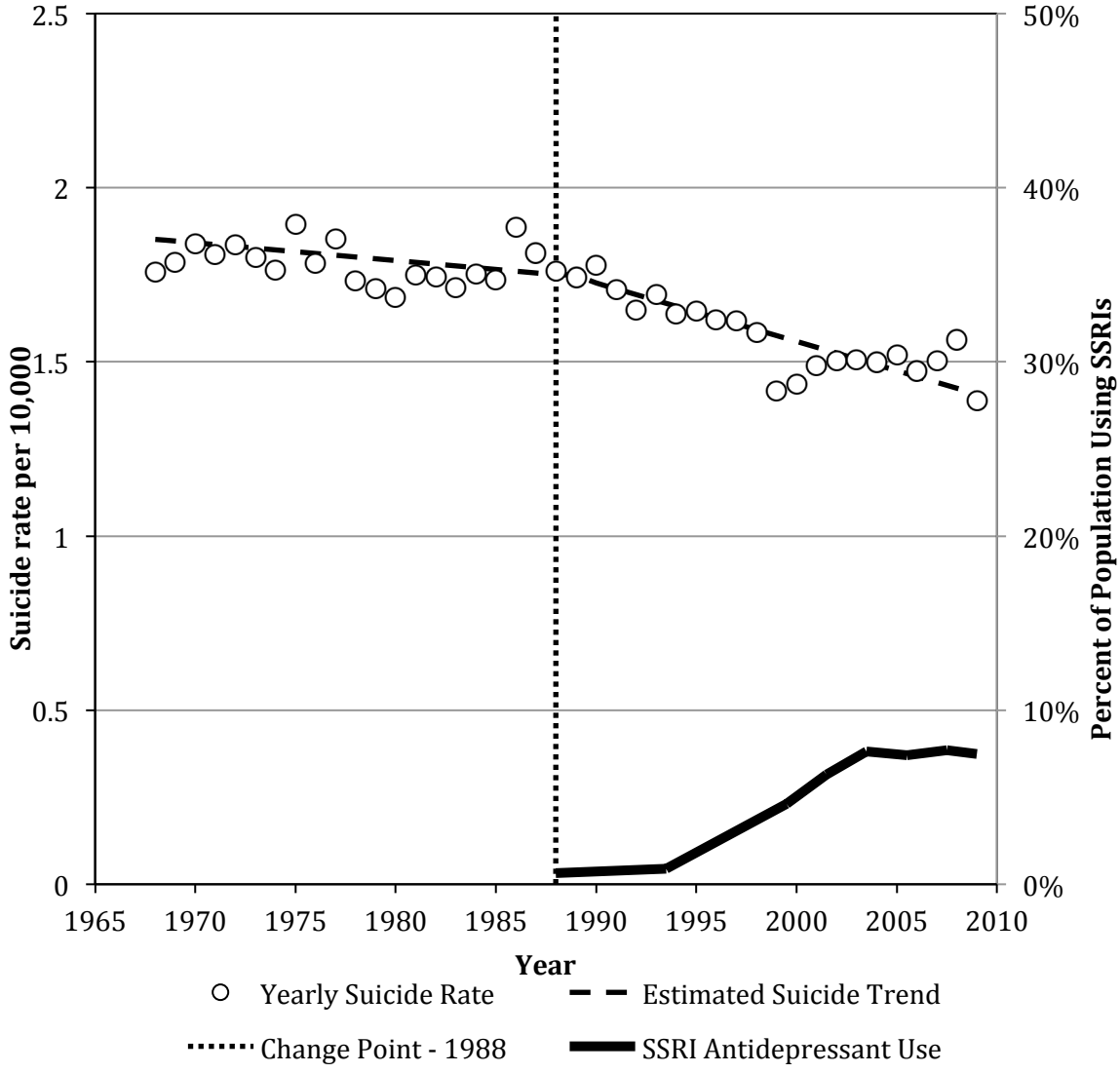

Figure 2. Estimated mortality rates for those living in counties one standard deviation above and below national average, NCHS 1968-2009.

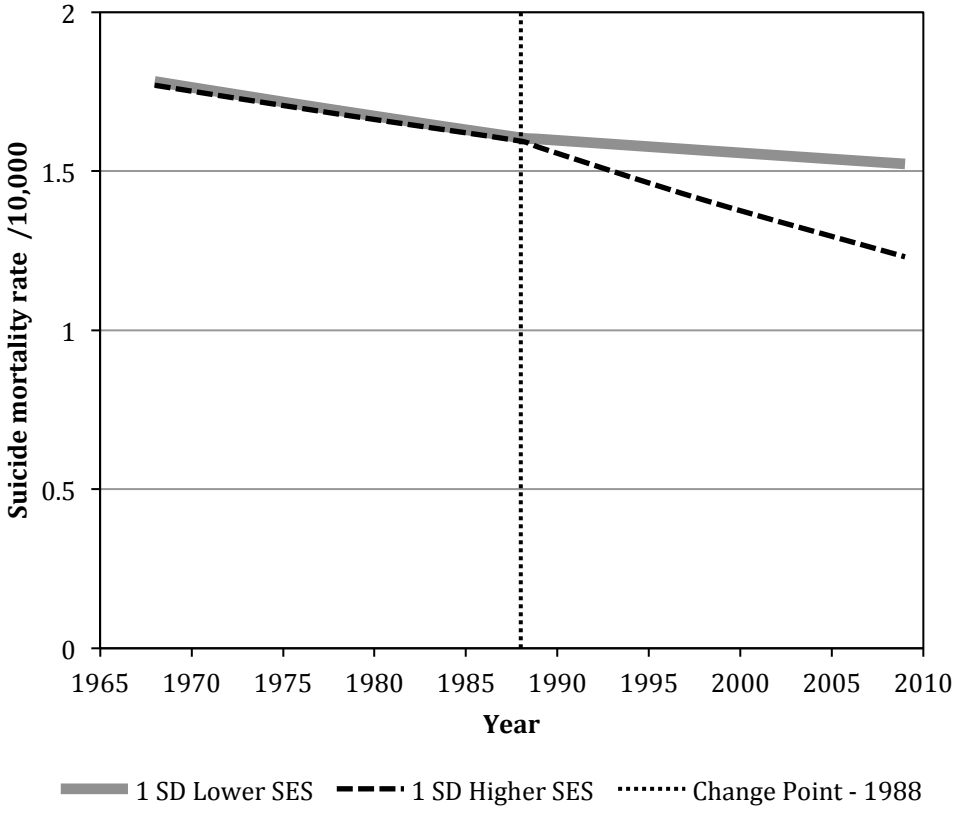

Table 1. Negative binomial estimates of the relationship between rates of suicide post-prevention and socioeconomic status, NCHS 1968-2009

	Model 1			Model 2		
	MRR	95% C.I.	p-value	MRR	95% C.I.	p-value
Year	0.994	(0.993, 0.996)	<0.001	0.995	(0.993, 0.996)	<0.001
Post-SSRI Change	0.996	(0.994, 0.997)	<0.001	0.998	(0.996, 1.000)	0.023
Socioeconomic Status	0.966	(0.942, 0.991)	0.009	0.997	(0.973, 1.021)	0.786
Post-SSRI Change x Socioeconomic Status				0.995	(0.994, 0.996)	<0.001
Log Pseudo-Likelihood			-1159930.5			-1159644.5
AIC			2319891			2319321
Observations			3149651			3149651
Degrees of Freedom			14			15

NB: All models adjust for urbanicity, race, sex, and age. We model exposure using mid-year population. We use Huber-White robust standard errors and adjust for county-level clustering.

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