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The Influence of a Major Disaster on Suicide Risk in the Population

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The authors investigated the relationship between the September 11, 2001 terrorist attacks and suicide risk in New York City from 1990 to 2006. The average monthly suicide rate over the study period was 0.56 per 100,000 people. The monthly rate after September 2001 was 0.11 per 100,000 people lower as compared to the rate in the period before. However, the rate of change in suicide was not significantly different before and after the disaster, and regression discontinuity analysis indicated no change at this date. There was no net change in the suicide rate in New York City attributable to this disaster, suggesting that factors other than exposure to traumatic events (e.g., cultural norms, availability of lethal methods) may be key drivers of suicide risk in this context.

Suicide is a major cause of disability-adjusted life years lost worldwide (Üstün, Ayuso-Mateos, & Chatterji, 2004) and annually accounts for approximately 30,000 deaths in the Unites States alone (Centers for Disease Control and Prevention, 2008). Several individual-level suicide risk factors have been identified (i.e., older age, male gender, psychopathology; Institute of Medicine, 2002; Maris, 2002; U.S. Department of Health and Human Services, 1999); however, most of the interpersonal variability in suicide risk remains unexplained and the evidence about the population-level drivers of suicide rates remains underexplored. Identifying factors that influence the population suicide rate, such as climate (Adjacic-Gross et al., 2006), seasonal allergens (Postolache et al., 2005), relative income inequality (Miller et al., 2005), and urbanicity (Qin, 2005) is critical for understanding the contextual elements of suicide risk (Hawton & van Heeringen, 2009; Institute of Medicine, 2002).

Disasters represent a natural experiment that may yield insight about the sources of population variability in suicide rates (Bromet & Dew, 1995). Given the well-documented acute increase in psychopathology after disasters (van Griensven et al., 2006), the suggestive evidence that such psychopathology may persist for years after the event (Adams, Boscarino, & Galea, 2006; Nandi et al., 2004), and the greater likelihood of suicide among persons with psychopathology (Bullman & Kang, 1994; Zivin et al., 2007), it may be expected that suicide rates may also rise after such events. Indeed, several studies have shown an increase in suicide risk after traumatic events, such as the Bali, Indonesia bombings (Suryani et al., 2008). Overall, however, the evidence concerning

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the association between disasters and suicide is mixed (Bromet & Dew, 1995; Rezaeian, 2008). In corrected analyses, Krug et al. (1999) reported no change in the suicide rate in U.S. counties affected by natural disasters, as did Bourque, Siegel, and Shoaf (2001) and Shoaf, Sauter, Bourque, Giangreco, and Weiss (2004) in their analyses of communities affected by the 1994 Northridge, California earthquake.

The September 11, 2001 terrorist attacks were the largest single day manmade loss of life in the United States since the Civil War, hence representing a unique opportunity for an assessment of suicide rates after a large disaster. However, existing investigations of the influence of this disaster on suicide risk have all examined populations that were not directly affected by the event, and these studies reported conflicting results. Salib (2003) reported a significant decrease in completed suicides in England and Wales during the weeks after the attacks, whereas de Lange and Neeleman (2004) reported an increase in completed suicide and deliberate self-harm in the Netherlands during a comparable period after the event.

It has been demonstrated that methodological choices can substantially influence the findings of changes after a disaster event (Norris, 2006), and an important limitation of extant studies of the September 11, 2001 disaster is that they have generally failed to account for variation in long- and short-term trends in suicide rates in their analytic approach (Dominici, Levy, & Louis, 2005; Lin, 2008). Traditional pre- and postdisaster comparisons may not control for time-varying factors that may be coincident with the study timeframe or explicitly test the causal nature of the exposure-outcome relationship (Kerlinger & Lee, 2000). Consequently, the utilization of multiple analytic approaches to investigate the association between a disaster event and suicide is critical when the relationship among the independent and dependent variables is not well understood, as well as to assess the robustness of findings.

Therefore, aiming to further our understanding of population suicide rates after a singular disaster we used multiple analytic approaches to assess the short-term (yearly) and the long-term (over 203 months) trends in monthly suicide rates, before and after the September 11, 2001 terrorist attacks, in the five boroughs of New York City (NYC) from 1990 to 2006. We used multiple methodological approaches to assess the robustness of our results. We predicted an increase in the suicide rate following the disaster based on the parallel findings that exposure to mass trauma is associated with severe psychological distress, and that such psychological distress, particularly depression and posttraumatic stress disorder, is associated with elevated suicide risk. In particular, there is evidence that severe and persistent psychological distress developed in New York City residents not directly exposed to the event (DiGrande et al., 2008; Galea & Resnick, 2005; Marshall et al., 2007), and that the event was characterized by substantial disruptions in domains of finances, work, and interpersonal relationships that influence psychopathology after a disaster event (Galea, Tracy, Norris, & Coffey, 2008; Nandi et al., 2004). Therefore, the extant evidence suggests that this disaster had the potential to influence suicide risk among the population overall.

METHOD

This study was reviewed and approved by the institutional review boards at the New York Academy of Medicine and the New York City Department of Health and Mental Hygiene.

Data Source

Data on all completed suicides between 1990 and 2006, inclusive, were collected from the Office of the Chief Medical Examiner of New York City. Trained assistants abstracted information on all causes of death and demographics as reported in the medical histories and autopsy reports in the Office of the Chief Medical Examiner. All deaths suspected to have occurred unnaturally within the five boroughs of NYC are reviewed by the Medical Examiner, and the manner of death is determined based on medical records, autopsy, forensic evidence, and investigative interviews. Medical Examiner surveillance data has been shown to have over 90% sensitivity and specificity for suicide deaths when compared to standardized assessment tools (Comstock, Mallonee, & Jordan, 2005).

Measures

Our dependent variable was the monthly suicide rate per 100,000 persons in NYC from 1990 to 2006. To estimate this rate we divided the count of suicides per month by the yearly NYC population estimates. The suicide counts (the numerator) included only suicides of NYC residents that occurred in the five boroughs of New York City. The NYC population counts (the denominator) were the midyear population estimates published by the United States Census (New York State Data Center, 2008), an approach used by many previous studies of disasters and suicide rates (e.g., de Lange et al., 2004; Liaw et al., 2008; Shoaf et al., 2004). To investigate variation in the association between this event and suicide, we also calculated gender- and race-specific monthly suicide rates (Hawton & van Heeringen, 2009; Institute of Medicine, 2002).

Our primary independent variable of interest was the date September 11, 2001. Other independent variables we included were various time components that accounted for short- and long-term trends in suicide rates; the format of all our independent variables depended on the type of modeling performed. We included a variable to account for short-term trends in suicide to adjust for a possible association between monthly suicide rates and warmer months. Where possible, we included a variable to account for long-term trends in suicide rates to adjust for patterns of monthly suicide rates from 1990–2006 unrelated to the September 11, 2001 terrorist attacks.

Data Analysis

To examine the robustness of the relationship between the September 11, 2001 attacks and monthly suicide rates we used multiple analytic approaches: chi-squared comparisons and simple linear regression, generalized additive models, piecewise-linear regression, and regression discontinuity analyses. For all models we eliminated one month of data (December 1999) for a total of 203 observations. Due to altering study management, the data collection methods in this one month may not have been comparable to the other months. We conducted a sensitivity analysis to determine the impact of excluding this data point, and inclusion of this month did not appreciably change our results or the inferences drawn (data not shown).

Initially, we sought to replicate the analytic strategies of previous studies of the influence of the September 11, 2001 disaster on suicide risk, namely chi-squared tests and linear modeling, to generate directly comparable results (de Lange & Neeleman, 2004; Salib, 2003). For these initial analyses we first compared the suicide counts for the years before and after 2001 to those in 2001 to see if the suicide risk in 2001 was different from these surrounding years. Second, to account for seasonable variation in suicide risk, we then compared suicide counts in September 2001 to the same month in previous and subsequent years. Finally, we compared the suicide rate of September 2001 to the other months in the same calendar year.

Generalized linear model. To account for short-term (e.g., yearly) trends in suicide rates, we fit a generalized linear model predicting the monthly suicide rate per 100,000. In this model, we created a binary dummy variable in which all months prior to September 2001 were coded as zero and September 2001 and all subsequent months were coded as one. We included a quadratic indicator variable for month (range: −6 to +6 centered on June) to account for the short-term trends. To determine whether this model specification was correctly identifying a change at September 2001, we also fit this model with another placebo month/year pairing as the zero/one switch point to examine whether the results were specific to September 2001. The model specification (the results of which are illustrated by Models 1 and 2 from Table 2) is below.

$$\text{E(Suicide Rate)} = \alpha + \beta(\text{Month/Year pairing}) + \beta(\text{Month}) + \beta(\text{Month})^2 + \epsilon \quad (1)$$

Generalized additive model. Previous research has indicated that suicide rates change over time in a nonlinear (e.g., logarithmic, quadratic, etc.) manner; thus, to accurately identify whether the September 2001 disaster influenced the monthly suicide rate it is critical to take into account these long-term trends. We fit a generalized additive model (GAM), which is a data-driven analytic method appropriate for identifying nonlinear changes over long periods of time that can complement traditional parametric analyses such as linear regression (Austin, 2007; Benedetti & Abrahamowicz, 2004) and has been previously used for time-series analyses (Dominici, 2004; Hastie & Tibshirani, 1990). We used generalized additive modeling to identify whether there were long-term (over 203 months from 1990 to 2006) nonlinear trends in the monthly suicide rate from 1990 to 2006 after accounting for the short-term (i.e., yearly) trends. This modeling approach accounts for nonlinear relationships by replacing the linear parameter with a smoothing function (indicated by s^1 and s^2 in the equation below). Consistent with other GAM applications to longitudinal data we used a univariate cubic spline basis and the generalized cross-validation method to determine the appropriate smoothness of these functions (Wood, 2006). To estimate the short-term trends we used the same indicator variable specification as in the generalized linear model described above (Month in the equation below, range: −6 to +6 centered on June) for each year. To estimate the long-term trends we used a variable sequentially numbered from January 1990 to December 2006 (Time in the equation below, range: 1 to 203). We fit this model to the entire sample, as well as to each gender and racial subgroup to examine any differences between the groups (there were none, and thus only the full-sample results are presented below). We estimated GAMs using both Poisson and Gaussian distributions for the monthly suicide rate and found no significant difference; therefore, for ease of comparison between model methods, all GAMs were fit with the Gaussian distribution with a canonical link. We used the results of GAMs to examine the short- and long-term trends in the monthly suicide rate and to determine the inflection points which delineated the piecewise regression analysis (described below). The GAM specification is below.

$$\text{E(Suicide Rate)} = \alpha + s^1(\text{Time, degrees of freedom}) + s^2(\text{Month, degrees of freedom}) + \epsilon \quad (2)$$

Piecewise linear regression. After identifying the nonlinear elements of long-term trends in suicide from inspecting the plots generated by the generalized additive model, we then built a piecewise generalized linear regression model to examine the association between September 11, 2001 and monthly suicide rates accounting for these nonlinear elements. To estimate the piecewise model, we marked specific dates where the monthly suicide rate appeared to change slope by visually inspecting the plot results generated by the GAM. Between these time points, we created four straight linear segments. We split the line segment containing September 2001 (which spanned January 1, 2000 to June 31, 2004), resulting in two sections surrounding September 2001 (January 1, 2000 to August 31, 2001 and September 1, 2001 to June 31, 2004) for a total of five line segments. We then compared the monthly suicide rate across these two to determine whether the rates changed at this date. As with the simple generalized linear model described above,

we included a quadratic term for the indicator variable for month to account for short-term trends. We evaluated the influence of age in stratified models (data not shown), but there was no evidence of effect modification and thus only the overall sample estimates are presented here. The model specification (Model 3 from Table 2) is below.

$$\begin{aligned} \text{E(Suicide Rate)} = {} & \alpha + \beta(\text{Month}) + \beta(\text{Month})^2 \\ & + \beta(\text{Segment 1: Jan 1990–Feb 1998}) \\ & + \beta(\text{Segment 2: Mar 1998–Nov 1999}) \\ & + \beta(\text{Segment 3: Jan 2000–Aug 2001}) \\ & + \beta(\text{Segment 4: Sept 2001–June 2004}) \\ & + \beta(\text{Segment 5: July 2004–Dec 2006}) \\ & + \epsilon \end{aligned} \quad (3)$$

Regression discontinuity analysis. To confirm the findings from the piecewise regression, we performed regression discontinuity analysis (Kerlinger & Lee, 2000; Trochim, 1990), which is an appropriate method for identifying changes due to an exposure using observational data. This analysis examined whether there was a significant change in the monthly suicide rates directly before and after September 11, 2001 using smoothed kernel functions with bootstrap standard errors (replications: 50).

All generalized additive modeling was carried out using R 2.7.2 and the generalized linear models were carried out using SAS 9.1. The regression discontinuity analysis was carried out in STATA 10. Statistical significance was set a priori as $p < .05$ and all p values refer to two-tailed tests.

RESULTS

There were 8,058 suicides among New York City residents from 1990 to 2006 (Table 1). For all years, the majority of suicides

Table 1. Average Annual and Monthly Suicide Rates for New York City per 100,000 Population, 1990–2006

	Suicides counts (*N*)				Annual suicide rates per 100,000
Year	Total	Male	White	Black	
1990	596	419	308	123	8.14
1991	612	458	333	103	8.30
1992	574	428	292	104	7.73
1993	511	386	286	105	6.81
1994	550	401	298	72	7.27
1995	516	377	259	105	6.76
1996	480	362	257	77	6.24
1997	458	319	245	82	5.89
1998	459	356	231	85	5.84
1999	371	277	198	64	4.67
2000	386	298	188	74	4.82
2001	400	296	227	77	4.96
2002	447	326	243	78	5.52
2003	424	297	233	64	5.22
2004	439	327	221	84	5.37
2005	419	313	222	71	5.10
2006	416	308	200	67	5.04
All years	8058				
Total number of months = 203	*M*	*SD*	Min	Max	
Monthly suicide rate	0.51	0.13	0.22	0.93	
Male monthly suicide rate	0.38	0.10	0.19	0.69	
Female monthly suicide rate	0.13	0.05	0.01	0.26	
Black monthly suicide rate	0.09	0.04	0.01	0.25	
White monthly suicide rate	0.27	0.08	0.10	0.45	
Other monthly suicide rate	0.15	0.06	0.02	0.34	

Table 2. Results From General Linear and Piecewise Regression Analyses Predicting Monthly Suicide Rate in New York City, 1990–2006

	Average expected change in monthly suicide rates					
	Model 1		Model 2		Model 3	
	B	*SE*	*B*	*SE*	*B*	*SE*
General linear model						
Month	0.001	0.003	0.001	0.002	0.002	0.002
Month^2	−0.002	0.0008*	−0.002	0.0008*	−0.002	0.0006**
September 2001 (ref. all months prior)	−0.109	0.02**				
Placebo date						
September 2000 (ref. all months prior)			−0.128	0.016**		
Piecewise regression						
Segment 1 (Jan 1990–Feb 1998)					−0.002	0.0002**
Segment 2 (Mar 1998–Nov 1999)					−0.005	0.002
Segment 3 (Jan 2000–Aug 2001)					0.002	0.003*
Segment 4 (Sep 2001–Jun 2004)					0.001	0.003
Segment 5 (Jul 2004–Dec 2006)					−0.002	0.002
Adjusted R^2	.170		.246		.534	

*$p < .05$. **$p < .01$.

were committed by White men. Over the 17-year (203-month) period, the average monthly suicide rate was 0.51 per 100,000 ($SD = 0.13$). As with the aggregated annual estimates, the majority of these monthly suicides were among men and non-Hispanic Whites.

In preliminary analyses we observed that the number of suicides in 2001 (4.96 per 100,000) was significantly lower compared to the average number of suicides for all previous years, 6.55 per 100,000 people from 1990 to 2000, χ^2 (1, $N = 901$) = 19.10, $p < .01$, and the average for all subsequent years, 6.58 per 100,000 people from 2002 to 2006, $\chi^2(1, N = 829) = 24.76$, $p < .01$. The monthly suicide rate in September 2001 (0.35 per 100,000) was not significantly different from the average September rate of previous years, 0.53 per 100,000, χ^2 (1, $N = 68$) = 3.43, $p = .06$, or subsequent years, 0.43 per 100,000, $\chi^2(1, N = 63) = 3.42$, $p = .06$. Finally, the suicide rate in September 2001 was not significantly different from that in the other months of 2001, χ^2 (11, $N = 62$) = 15.64, $p = .16$.

Table 2 shows the results from the simple generalized linear models (Model 1 and 2) and the piecewise regression (Model 3). Although Model 1 indicates that the mean monthly suicide rate is lower after September 11, 2001, Model 2 (which evaluates whether this finding is also true of placebo dates) demonstrates that this is also the case for September 2000. Model 3 demonstrates that after adjusting for long-term and short-term monthly suicide trends, the rate of change of monthly suicides was not significantly different after September 11, 2001 as compared to the prior period.

Figure 1 is a plot of the observed monthly suicide rates against the predicted trend from the GAM and depicts the nonlinear short-term (12 month) trends and nonlinear long-term (203-month) trend in suicide rates over the study period. The predicted slopes of the straight-line segments from the piecewise linear regression (Model 3 from Table 2) are also illustrated by this plot. Figure 1 indicates that the significant nonlinear short-term trend in monthly suicide rates has a quadratic shape and corresponds to higher suicide rates in the warmer (summer) months ($p < .01$). In addition, the significant long-term trend in suicide rates decreased from 1990 to 1999 and increased from 1999 onwards ($p < .01$).

As shown in Model 3 and depicted in Figure 1, the piecewise regression demonstrates that the trend of the monthly suicide rates was not significantly different after September 2001 as compared to the segment before September, 2001 ($p = .79$). These findings suggest that there is no direct evidence that the September 11, 2001 terrorist attacks affected the suicide rates in New York City. We found similar results when we ran both the generalized additive model and a piecewise regression using weekly suicide rates as opposed to monthly suicide rates (data not shown). Finally, there was no statistically significant change in the monthly suicide rates at September 11, 2001 in the regression discontinuity analysis using a kernel smoother (coefficient −0.01, $p = .84$).

DISCUSSION

In New York City during the 17-year period from 1990 through 2006 there was a significant decrease in the number of completed

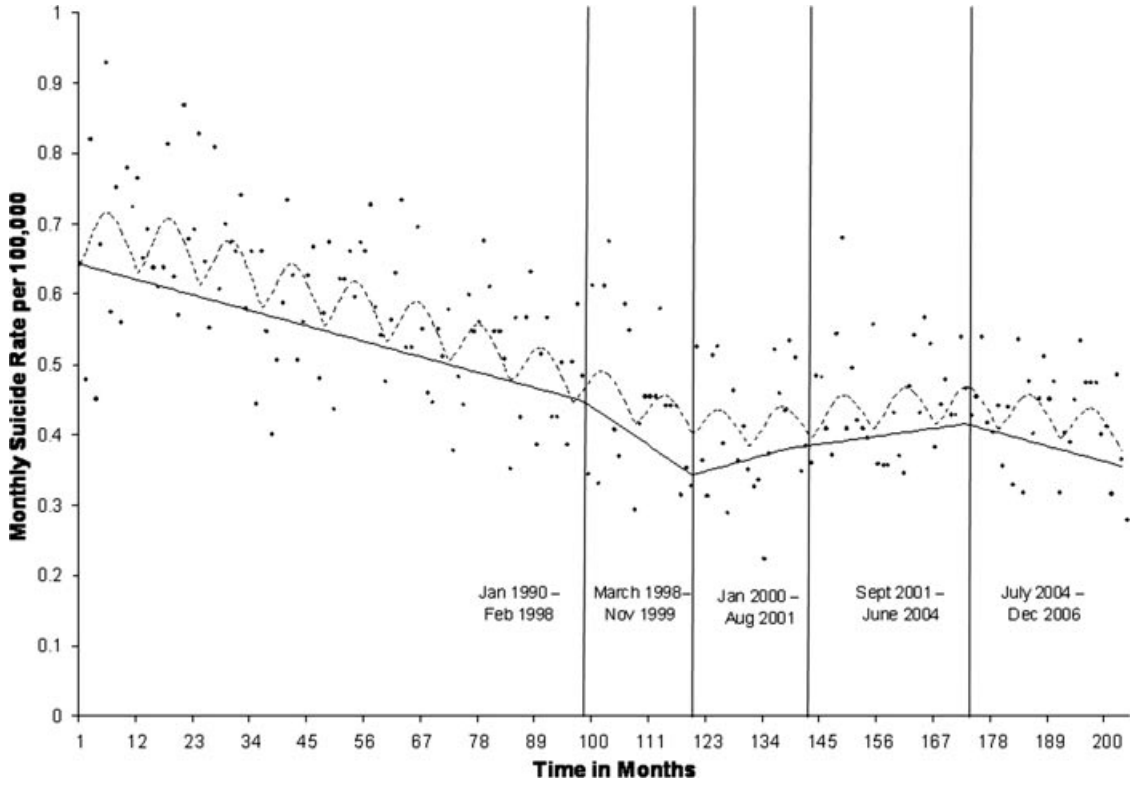

Figure 1. Monthly suicide rate per 100,000 people in New York City, 1990–2006. This plot displays the short-term (yearly) and long-term (over 203-month) trend in suicide rates predicted by the generalized additive model (GAM). The grey scalloped line represents each annual (12-month) trend in suicide and highlights the nonlinear distribution of suicides over the calendar year. The solid lines represent the piecewise segments that were predicted by visual inspection of the GAM plot and were used in the piecewise regression model described in Model 3, Table 2.

suicides, a trend that was not affected by the September 11, 2001 disaster. We used multiple analytic approaches that account for long-term trends in suicide risk in this population to evaluate the robustness of our findings. These findings are contrary to our initial hypothesis that the widespread distress and psychopathology documented in this population subsequent to this disaster would result in an increase in the population suicide rate. These results suggest that even in this setting—where there was a documented increase in psychological distress in the general population after the disaster—there was no association between a mass traumatic event and suicide rate. This suggests that factors other than exposure to traumatic events and psychopathology (e.g., cultural norms, socioeconomic characteristics, availability of lethal methods) may be key drivers of suicide rates in this context.

Our results contrast with the two previous studies of this relationship (de Lange & Neeleman, 2004; Salib, 2003), and there are several potential reasons why our findings differ from these earlier reports. First, prior studies only examined relatively brief (<3 months) periods after the September 11 disaster, whereas we were able to account for 5 years after the event. Second, these reports relied on methods that did not account for temporal variation in the base rate over time. Finally, they examined populations in Europe that were not directly affected by the event. This latter point is particularly important in light of evidence that sociocultural contexts influence the relationship between a disaster event and suicide risk (Bromet & Dew, 1995). For example, in contrast to the mixed evidence of a relationship between mass trauma and suicide in the United States, there is more consistent evidence that disaster events (both natural and human-made) in Eastern Europe and Southeast Asia tend to be associated with changes in suicide rates (Nishio et al., 2009; Rahu et al., 1997; Suryani et al., 2008; Yang, Xirasagar, Chung, Huang, & Lin, 2005), which suggests that our findings may not extend to non-Western settings.

Emile Durkheim (1951) argued that causes of suicide should be understood from a sociological framework because though each suicide is a singular event, the number of suicides in a given society is affected by societal factors, such as social connectedness and political institutions rather than individual characteristics such as psychological distress. In terms of the relationship between specific events and suicide, Durkheim argued that, "The most varied and

even the most contradictory events of life may equally serve as pretexts for suicide. This suggests that none of them is the specific cause" (p. 298). Our analysis is consistent with this view of the causes of suicide, i.e., that a single event—even one as horrific as the September 11, 2001 terrorist attacks—may have a complicated influence on, and potentially no net change in, population suicide risk.

These findings should be interpreted in light of study limitations. Foremost, we had information only on completed suicide, not attempts or ideation, and previous studies have suggested that persons who complete suicide have different demographic characteristics (e.g., women are more likely to attempt than men) than either of these groups (Mann, 2002). Although a key strength of our study is that we examined a population directly affected by this disaster, our analyses would have benefited from including a comparison city to examine whether the trends we observed in NYC over the study period were comparable to other urban areas. Also, although our dataset provides comprehensive information on suicides committed within the catchment area of the OCME, we do not have information on New York City residents who may have committed suicide outside this area. Although we used the midyear population estimate for the denominator for each year, these estimates may not fully capture the in- and out-migration from the NYC area over the study period. However, research suggests there was only limited out-migration from lower Manhattan after the attacks (DiGrande et al., 2008), and as long as such migration was nondifferential based on suicide risk, such imprecision would not have substantially biased our results. Another limitation of this study is the lack of data on the degree of exposure to the events of September 11, 2001 among the suicide cases. However, previous research has indicated that even individuals not directly affected by the attacks developed psychological distress (Galea & Resnick, 2005); thus, the lack of information on degree of exposure does not preclude our hypothesis that this event could have influenced the population as a whole. Our subgroup analyses based on age, sex, and race failed to reveal any differences in the influence of the disaster on suicide rates; however, this does not preclude that there are other subgroups that may have been particularly affected by this event (i.e., those with a prior history of psychopathology, or those who experienced personal loss due to the event). Unfortunately, our data source does not provide information on these individual characteristics to define such groups. Finally, given the source of the data, the results may not be generalizable to rural or suburban settings.

In summary, this analysis of the influence of a large disaster on suicide risk in New York City indicates there was no change in the population rate of suicide attributable to this event. Notwithstanding the well-documented relation between psychopathology and increased risk of individual suicide, our findings suggest that the determinants of suicide in the population are complex even in contexts where there is ample evidence of an increase in psychopathology, such as the aftermath of a disaster. We believe this study contributes three significant elements to research on the associations between disasters and suicide risk: (a) It demonstrates the utility of methods that account for temporal changes that may have occurred contemporaneously with the disaster that could have influenced suicide risk and thus produced a spurious association (Dominici et al., 2005); (b) this analysis directly responds to previous reports of an association between this disaster and suicide and, using more robust methods, challenges these findings; and (c) within the context of the robust finding that psychopathology is associated with suicide risk, and the links between traumatic exposure and psychopathology, we document no demonstrable link between exposure to mass traumatic events and suicide rates in the general population. This finding strongly suggests that a full understanding of the determinants of suicide rates in populations needs to account for a range of factors, both contextual and ecological, that operate to determine suicide rates in populations. The systematic study of natural experiments such as disasters can thus provide clues about the determinants of suicide risk in the general population, particularly in those instances where the results are contrary to expectations as in this case.

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