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ARTICLE *in* JOURNAL OF AFFECTIVE DISORDERS · AUGUST 2015

Impact Factor: 3.38 · DOI: 10.1016/j.jad.2015.07.001

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## Research report

## Contagion from peer suicidal behavior in a representative sample of American adolescents

Jason R. Randall<sup>a,b,\*</sup>, Nathan C. Nickel<sup>a,b</sup>, Ian Colman<sup>c</sup><sup>a</sup> Department of Community Health Sciences, University of Manitoba, Canada<sup>b</sup> Manitoba Centre for Health Policy, University of Manitoba, Canada<sup>c</sup> Department of Epidemiology and Community Medicine, University of Ottawa, Canada

## ARTICLE INFO

## Article history:

Received 1 April 2015

Received in revised form

29 June 2015

Accepted 3 July 2015

Available online 26 July 2015

## Keywords:

Attempted suicide

Child

Adolescent

Propensity score

Depression

Prospective

Self-injurious behavior

Risk factors

## ABSTRACT

**Background:** Assortative relating is a proposed explanation for the increased occurrence of suicidal behavior among those exposed to suicidal peers. This explanation proposes that high-risk individuals associate with each other, and shared risk factors explain the effect.

**Methods:** Data were obtained from the ADDhealth longitudinal survey waves I and II ( $n=4834$  school attending adolescents). People who reported peer suicidal behavior in the first wave were identified and classified as the exposure group. Potentially confounding variables were identified, and propensity scores were calculated for the exposure variable using logistic regression. Inverse-probability-of-treatment weighted regression estimated the effect of exposure on the risk for a suicide attempt during the first two waves.

**Results:** Weighted analysis showed that the group exposed to a friend's suicide attempt had a higher occurrence of suicide attempts in both waves. Exposure to peer suicide attempts was associated with increased suicide attempts at baseline ( $RR=1.93$ ;  $95\%CI=1.23-3.04$ ) and 1-year follow-up ( $RR=1.70$ ;  $95\%CI=1.12-2.60$ ).

**Limitation:** Only two consecutive years of data are provided. Misclassification and recall bias are possible due to the use of self-report. The outcome may be misclassified due to respondent misunderstanding of what constitutes a suicide attempts, versus non-suicidal self-injury. Non-response and trimming reduced the sample size significantly.

**Conclusions:** Assortative relating did not account for all the variance and is currently not sufficient to explain the increased risk after exposure to peer suicidal behavior. Clinicians should assess for exposure to suicidal behaviors in their patients.

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## 1. Introduction

Suicidal behavior amongst youth is a significant cause of morbidity and mortality (Hawton and van Heeringen, 2009; Nock et al., 2013, 2008). A range of psycho-social-environmental factors have been linked with suicidal behavior (Bolton and Robinson, 2010; Cavanagh et al., 2003; Cooper et al., 2005; Harris and Barraclough, 1997; Hawton and van Heeringen, 2009; Hawton et al., 2003; Nepon et al., 2010; Nock et al., 2013). However, there are still uncertainties about the causes of suicidal behavior, and factors that could be addressed to prevent suicidal behavior amongst

adolescents (Bohanna, 2013; Hawton and van Heeringen, 2009). One particular area of sustained controversy has been whether the occurrence of suicide clusters indicates the existence of 'suicide contagion' (Davidson and Gould, 1989; Gould et al., 1994; Joiner, 2003, 1999; McKenzie et al., 2005; Robbins and Conroy, 1983; Wasserman, 1984). The existence of a causal effect from exposure to suicidal peers is contentious (Joiner, 2003). Some researchers have argued that suicide clusters occur because suicidal individuals cluster together (Joiner, 2003). This competing hypothesis will be referred to as the 'assortative relating hypothesis'. It suggests those at high risk of suicidal behavior are more likely be friends with other high risk individuals. This hypothesis suggests that the increased risk among those exposed to suicidal behavior would be explainable by shared risk factors, and is merely an association and not a causal relationship.

Previous studies have not provided definitive evidence for, or against, assortative relating as the sole cause of increased risk after

Abbreviations: RR, risk ratio

\* Correspondence to: Community Health Sciences, Faculty of Medicine, 408–727 McDermot Ave. University of Manitoba Bannatyne Campus, Winnipeg, Canada MB R3E 3P5.

E-mail address: [Jason\\_Randall@cpe.umanitoba.ca](mailto:Jason_Randall@cpe.umanitoba.ca) (J.R. Randall).

<http://dx.doi.org/10.1016/j.jad.2015.07.001>

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exposure to suicidal peers (Brent et al., 1992; De Leo and Heller 2008; Gould et al., 1994; Haw et al., 2013). Recent research has examined the association of peer suicidal behavior and the risk of suicide attempts in adolescents, and found evidence for an increase in risk (Abrutyn and Mueller, 2014; Feigelman and Gorman, 2008; Nanayakkara et al., 2013; Swanson and Colman, 2013). However, these studies have generally adjusted for a small number of confounding factors, such as depression and substance use. In order to discount this alternate explanation as the sole reason for the association, a wide range of potential variables involved in assortative selection of peers need to be controlled. The primary hypothesis is that being exposed to peer's suicidal behaviors will increase the risk of suicide attempts, even after controlling for the variables suspected of being responsible for the association under the assortative relating hypothesis. A secondary hypothesis is that this effect, if it exists, will be concentrated in those with pre-existing risk factors (e.g. depression, substances abuse, stressful environment). This article will test these two competing theories of the association between exposure to suicidal behavior and suicide attempts: assortative relating, and a true effect of exposure (i.e. contagion). This article will examine whether assortative relating can entirely explain the association.

## 2. Methods

### 2.1. Sample

This study analyzed data in the public use dataset from the ADDhealth survey. This survey began in 1994 and currently contains four waves worth of data. The survey is longitudinal and surveys the same participants over multiple years. We limited analysis to the first two waves of data, referred to as 'Year I' and 'Year II' in this article, since they provided consecutive years of assessment. The third and fourth waves occur with considerable lags in time, preventing the use of a cross-lagged design. Also, the failure to find an association in the later waves could be due to the multi-year gaps rather than the absence of any effect (e.g. increased risk could be time limited). These waves also occur outside of adolescence, when exposure to peer suicide could be a strong risk factor.

Year I and Year II were undertaken in consecutive years in 1994/5 and 1995/6. The participants were aged 11–20. Most respondents were between 13 and 17 years of age. In the public use dataset, the first wave of data sampled 6504 people. The second year surveyed 4834 of the original sample in the following year. People in the first year were dropped during the second year for two reasons; they were in grade 12 during the first year, or they were enrolled as part of two specific sub-samples. This study

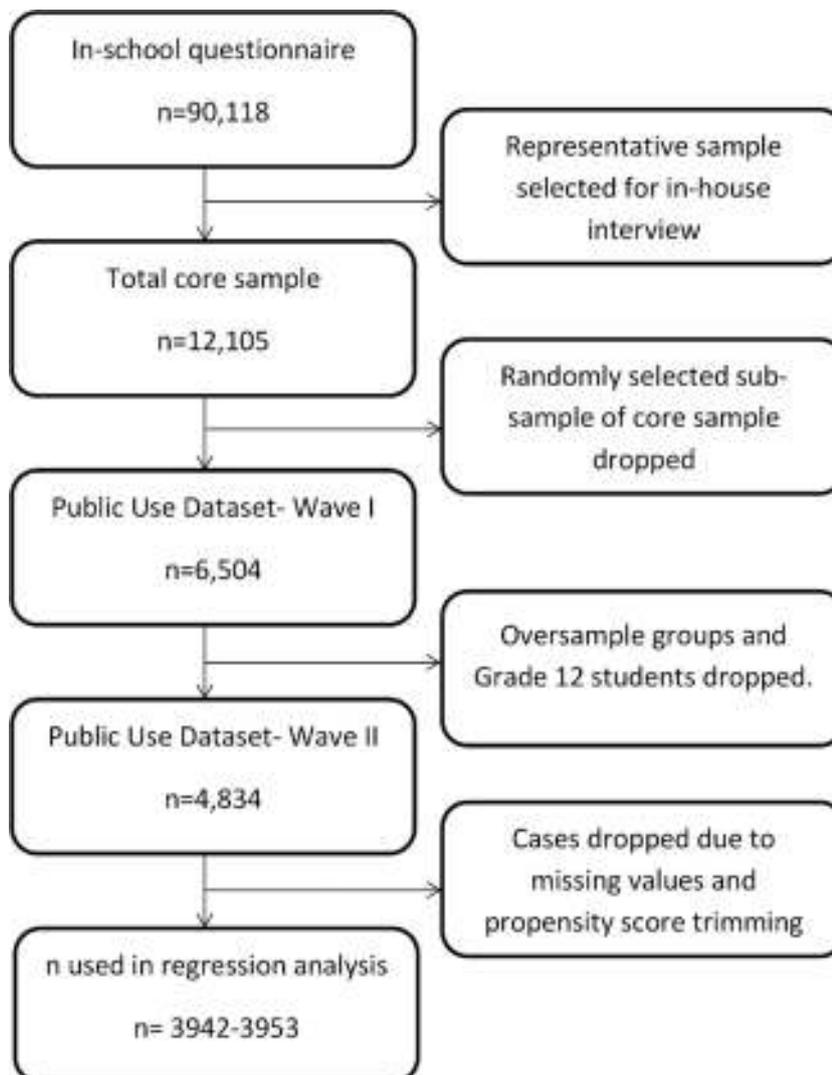


Fig. 1. Sampling flowchart.

focused on those with data from both years of enrollment ( $n=4834$ ). Details of the sampling are depicted in Fig. 1.

A cluster sampling method design was used to obtain a representative sample of school-attending adolescents in the United States. The primary sampling unit was schools. Schools were selected and individuals were weighted to ensure a representative sample of US schools in regards to region of country, urbanicity, school size, school type, and ethnicity. A total of 80 high schools and 52 middle schools were selected. An in-school questionnaire was administered to more than 90,000 students which asked questions about social and demographic characteristics, education and occupation of parents, household structure, risk behaviors, expectations for the future and several other factors. A second in-home questionnaire was administered to the students who were registered at the schools sampled during the in-school questionnaire. A total of 12,105 adolescents were sampled for the core sample of the study. Selection into the core sample was stratified to ensure that information was available for various demographic groups identified in by the in-school questionnaire. Survey weights were developed to ensure that the sample was still representative. The interview was conducted by a trained interviewer. However, for sensitive questions the students listened to pre-recorded questions and recorded their answers themselves in order to reduce response bias. The public use dataset is a randomly selected subset of the 12,105 student core sample with adjusted survey weights. Full information on the sampling methods used are available at the ADDhealth website (<http://www.cpc.unc.edu/projects/addhealth/design>; Harris et al., 2009). Although the data was collected two decades ago, it is unlikely that the age of the data would have any effect on the occurrence of suicide contagion or the hypotheses examined. This data was used because it provided a large representative sample with a large selection of potential confounders. It also provided the possibility for both cross-sectional and longitudinal analysis, and contained reasonably well assessed exposure and outcome variables.

## 2.2. Measures

The exposure variable for this study was reporting that a friend had made a suicide attempt in the previous year. The survey question used to determine exposure-status was; 'Have any of your friends tried to kill themselves during the past 12 months?' Those who answered 'yes' to this question during Year I were coded as 'exposed.' These individuals are referred to as the exposed or the exposed group in this article.

The primary outcome for this study was the occurrence of suicide attempts during the previous year. The survey question used to determine whether a participant had made an attempt was: 'During the past 12 months, how many times did you actually attempt suicide?' This question was recoded into a binary variable (none versus one or more). The outcome was measured in both Year I and Year II allowing examination for a cross-sectional association (Year I) and prospective relationship (Year II) between exposure to peer suicide attempt (measured in Year I) and respondent suicide attempts. Since there is overlap for the assessment of exposure and outcome during Year I, we do not know if exposure precedes the outcome for any particular individual.

The survey also included assessments of potential confounders based on previous literature on suicide and self-harm (Barzilay and Apter, 2014; Christiansen et al., 2014; Fliege et al., 2009; Haw et al., 2013; Hawton and van Heeringen, 2009; Hawton et al., 2012). These included questions about participant physical health, depressive symptoms, impulsivity, fearfulness, anxiety symptoms, other mental health symptoms, relationship with parents, relationship with friends, sexual orientation, visible minority status, self-esteem, weight image, physical and personality attractiveness

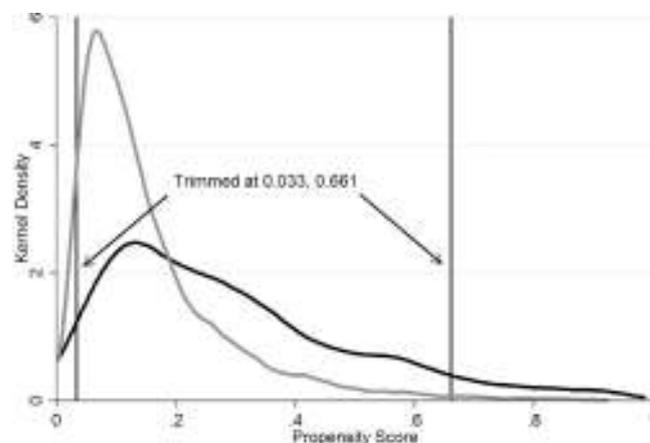
(rated by interviewer), parental use of public assistance, intelligence (self-reported and visual IQ test performance), school environment, neighborhood environment, relative physical development, optional thinking, exercise, substance use counseling, psychological counseling, drug and alcohol use, involvement in fights and violence, and exposure to violence. A total of 50 variables were created based on survey questions related to the areas noted above. These variables were chosen because they represented factors which could drive assortative relating, and/or are risk factors associated with increased risk of suicidal behavior.

## 2.3. Analysis

The analysis adjusted for the clustering and weighting used in the survey design. Survey weights from the second wave of the study were utilized. Percent of the sample with exposure and outcome was determined. Percentages were also derived by sex due to the differences in suicidal behavior and risk factors between males and females. The 50 created variables were entered simultaneously into a logistic regression model, stratified by sex, in order to derive the propensity score. The propensity score is the conditional predicted probability that an individual would be exposed given the 50 variables included in the regression model. Asymmetric trimming was performed to remove non-overlapping portions of the sample and to remove participants with excessively large weights. The methodology for trimming is based on methods discussed by Stürmer et al. (2010). Participants with propensity scores larger than the 99th percentile of the non-exposed group were trimmed as well as those with propensity scores smaller than the 1st percentile for the exposed group. Graph 1 illustrates the propensity score distributions of the two groups and the trim points used.

After trimming, weights were applied to approximate the 'Average Treatment Effect (ATE),' 'Average Treatment Effect for the Treated (ATT),' and 'Average Treatment Effect for the Untreated (ATU).' The ATT estimates the actual effect in the sample, since it estimates the effect on those who are exposed compared to a counterfactual population with similar propensity scores. The ATU estimates the effect that would have occurred if the unexposed population had been exposed. The ATE shows the effect across the whole sample population. After weighting and trimming, no significant associations occurred between any of the 50 variables and exposure status.

GLM regression, with inverse-probability-of-treatment weights derived from the propensity score, was used with a log link to obtain risk ratios (RR). Since covariates are adjusted by weighting then only the exposure variable is included in the regression



Graph 1. Kernel density plot of Propensity score by exposure status with trim points. Legend for graph 1: (a): Exposed, (b): Unexposed.

model. Regressions were performed for exposure and outcome during Year I once for each of the three weighting methods (i.e. ATE, ATT, ATU). A second set of regressions were done using exposure from Year I and outcome measured during Year II for each of the three weights. Regressions were also done without using the propensity-score derived weights to estimate the unadjusted associated between exposure and suicide attempts. *p*-values and 95% confidence intervals were derived for all of the analyses.

If there is a contagion effect then these estimates are potentially biased due to two factors. The first is that the propensity-score model was specified under the assumption that the ‘assortative relating’ hypothesis was the sole explanation for the effect – meaning it may not be properly specified to obtain the true treatment effects. This would likely cause an underestimation of the true effect due to over-fitting the model. The second is that it is unclear whether the stable unit treatment value assumptions (SUTVA) are met in this study (Rubin, 1990). This potential bias could reduce the power if it is towards the null. However, it is unlikely that a violation of SUTVA would result in an increased chance of a false positive. Therefore, if a correlation remains after adjustment then that would be evidence that contagion could be at least partially responsible for the correlation. The main goal of this analysis is not to determine an unbiased effect estimate, but attempt to falsify the contagion explanation. The effect estimate represents the remaining unexplained association between exposure and outcome and the confidence in that estimate.

A lowess graph was produced showing the proportion with suicide attempts in Year II (*Y*-axis) over predicted risk of suicide in Year II (*X*-axis) by exposure status. The predicted risk of suicide was determined using a logistic regression model including the 50 confounding variables. Lastly, the association of exposure with suicide attempt risk was examined stratified by sex, depression, age, any substance use, any exposure to violence, whether respondent believed their friends cared about them, and whether the individual had learned about suicide in school. All analyses were done using STATA SE 13 (StataCorp, 2013).

### 3. Results

#### 3.1. Occurrence of exposure and outcome

During Year I 19.16% (unweighted  $n=888$ ) of the sample reported that a friend had made a suicide attempt in the year before the interview (see Table 1). Suicide attempts were reported by 3.69% (178) of students during Year I and 3.98% (192) of students during Year II. Compared to males, females were more than twice as likely ( $RR=2.34$ ;  $95\%CI=1.65-3.31$ ) to report having made a suicide attempt in the past year in both years of the survey. Females were also 1.75 times as likely to have reported a friend attempting suicide during the previous year ( $95\%CI=1.49-2.06$ ).

**Table 1**  
Occurrence of exposure and outcome.

		Male % (n)	Female % (n)	Total % (n)
Year I	Exposure to suicide attempt	13.97% (312)	24.48% (576)	19.16% (888)
	Respondent suicide attempts	2.22% (51)	5.20% (127)	3.69% (178)
Year II	Respondent suicide attempts	2.30% (52)	5.69% (140)	3.98% (192)

Weighted percentage and unweighted *n*.

#### 3.2. Inverse-probability-of-treatment weighted regression analysis

Observations were trimmed for those with propensity scores less than 0.033 or greater than 0.661 (Graph 1). Table 2 shows the results of the inverse-probability-of-treatment weighted regression analysis. The increased risk of suicide attempts for participants was similar for both the treated and untreated, particularly at 1-year follow-up (Year II). The ATT estimate at baseline was 1.93 ( $95\%CI=1.23-3.04$ ;  $p=0.005$ ) and 1.70 at 1-year follow-up ( $95\%CI=1.12-2.60$ ;  $p=0.014$ ). The ATU estimate at baseline was 2.27 ( $1.49-3.47$ ;  $p<0.001$ ) and 1.68 at 1-year follow-up ( $1.10-2.59$ ;  $p=0.018$ ). The ATE estimate was 2.18 at baseline ( $1.46-3.25$ ;  $p<0.001$ ) and 1.69 at 1-year follow-up ( $1.14-2.51$ ;  $p=0.009$ ).

#### 3.3. Effect of exposure by risk of suicide attempt

Graph 2 shows the proportion of the sample with a suicide attempt at 1-year follow-up (*Y*-axis) by the predicted risk for suicide attempt (*X*-axis), according to the logistic regression model containing the 50 confounding variables, by exposure status. Those exposed to friends with suicidal behavior experienced a consistently higher occurrence of suicide attempts compared to non-exposed adolescents with similar predicted risk. The sole exception is for those with predicted risk close to zero, as the lines for exposed and unexposed appear to converge at this point – however there is not enough detail to determine whether the lines actually converge at any point.

#### 3.4. Potential effect modification of sex and suicide education

Results were not significant for gender, age, depression, exposure to violence, and whether respondents believed their friends cared about them. There also was no significant modification effect between exposure and whether the respondent had received education about suicide. Substance use was the only factor which was found to have a significant effect ( $p<0.01$ ), however this effect was only significant at baseline. At baseline, exposed individuals among those with no reported alcohol or drug use more often reported a suicide attempt ( $RR=3.50$ ;  $95\%CI=1.88-6.53$ ). However, those reporting drug and alcohol use had no elevated occurrence of suicide attempts if exposed to suicidal behavior amongst their friends at baseline ( $RR=1.03$ ;  $95\%CI=0.57-1.88$ ). Therefore those reporting substance use had a higher risk of being exposed, but their risk of suicide attempts was unaffected by the exposure.

#### 3.5. Post Hoc sensitivity analysis

Another analysis examined whether exposure to peer suicidal behavior, or suicidal ideation/attempts before the study period were confounding the results. This analysis was performed using exposure data from Year II, instead of Year I. The propensity score was recalculated for the Year II exposure and additional variables were added for Year I exposure, suicidal ideation/behavior, and mental health variables from Year II. The result for regression on Year II outcome was similar to the previous estimates ( $RR=1.92$ ;  $95\%CI: 1.28-2.88$ ).

## 4. Discussion

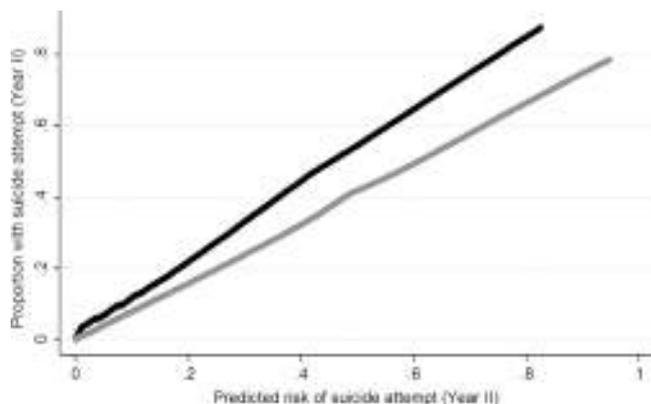
After adjustment for observed potential confounders, exposure to a friend's suicidal behavior was associated with a significant increase in the probability of a suicide attempt. This provides evidence against the hypothesis that assortative relating is the solely responsible for the increased risk associated with being

**Table 2**  
Effect of exposure to suicide attempt among friends on risk for suicide attempt

Trimmed Propensity model <sup>†</sup>											
	Sample	RR	95%CI		p-Value	Sample	RR	95%CI		p-Value	
Year I	UW	4.48	3.48	5.77	< 0.001	Year II	UW	3.16	2.38	4.18	< 0.001
	ATT	1.93	1.23	3.04	0.005		ATT	1.70	1.12	2.60	0.014
	ATU	2.27	1.49	3.47	< 0.001		ATU	1.68	1.10	2.59	0.018
	ATE	2.18	1.46	3.25	< 0.001		ATE	1.69	1.14	2.51	0.009

UW=unweighted; ATT=estimated effect for exposed; ATU=estimated effect for unexposed; ATE=estimated effect for sample. Exposure measured in Year I.

<sup>†</sup> Observations with propensity scores less than 0.033 or greater than 0.66 trimmed.



**Graph 2.** Proportion with suicide attempt in year II over predicted risk of suicide attempt. Legend for graph 2: (a) Exposed, (b): Unexposed.

exposed to peer suicidal behavior. Due to the common occurrence of exposure to suicidal peers, and the size of the remaining association, these data suggest that suicide contagion may be related to a concerning proportion of suicide attempts among adolescents.

There has been little empirical examination around the adequacy of the assortative relating hypothesis as an alternative explanation for previous research on suicide contagion (Haw et al., 2013). This is the first paper to our knowledge that used a large number of variables, measuring a wide range of potentially confounding factors, needed to test this alternate hypothesis. Despite controlling for a large variety of potential confounding variables, the relationship between exposure to peer suicidal behavior and respondent suicidal behavior remained. This study cannot delineate how much of the relationship between exposure and suicidal behavior for which these two explanations are individually responsible. It is likely that an appreciable portion of the elevated risk is due to assortative relating – high risk individuals being friends with other high risk individuals. However, some variables can conceivably be both involved in assortative relating and mediators of the exposure effect. Depression and depressive symptoms are examples of this. Depressed individuals may cluster together, but depression may also result from exposure to peer suicidal behavior. Other methods have been used in an attempt to isolate the effect in similar situations. Network analysis is one such method. However, to our knowledge, no research to date has had the proper repeated measurement, over smaller time periods, needed to isolate the true effect of exposure from the confounding in these variables – including the current study. Network analysis has been proposed as a method of conducting this type of research, but has also been criticized for its inability to discern a true effect of contagion from homophily (a more general term for assortative relating; Shalizi and Thomas, 2010). Therefore it is uncertain whether it is possible to determine an unbiased estimate for the contagion effect with currently available methods and data. The results presented in this article likely represent a lower bound for the effect of exposure. The remaining effect still represents a

significant concern, since it suggests that 11.8% of the events may be due to this exposure. The population attributable risk would be approximately 0.47%.

A third explanation may also be plausible, namely that some of the association is from shared stressful events between friends. However, the persistence of the risk into Year II seems counter to an acute shared stressor being the responsible factor. Also, it is likely that shared stressors would be mediated by depression or other psychological issues, which were included in the propensity-score model.

Perhaps the most interesting and useful finding is that increased risk of suicidal behavior does not appear to be limited to high-risk individuals. Mechanisms supporting these findings are unclear. The explanation that exposure to suicidal friends could lead distressed individuals to consider and attempt suicide is not congruent with these results. A social identity theory explanation is possible (De Leo and Heller 2008). When people identify as members of a group and observe other members of said group engaging in suicidal behavior they may perceive this as being a characteristic of the group (De Leo and Heller 2008). A person's social identity in the group then could influence their individual identity, and cause them to associate suicidal behavior with themselves. This association might not need to be explicit as recent research has found that implicit associations can influence suicidal behavior (Nock and Banaji, 2007a, 2007b; Randall et al., 2013).

#### 4.1. Strengths and limitations

This study draws upon a large representative cohort of American school-going youth. The dataset also contains a range of variables that measure potentially confounding risk factors that were adjusted for during analysis. This study adjusted for a considerable selection of potential confounding variables. This study also utilized an outcome question that specifically examines suicide attempts, rather than self-harm without distinction between suicidal and non-suicidal self-harm.

Residual confounding is possible. The propensity-score model was not capable of explaining most of the variance in outcome (indeed no model yet derived is capable of this). This means that it is still possible that unmeasured confounders could still exist. Although the set of conditioning variables was extensive, measurement error may have occurred.

Due to the school-based sampling, it is possible there is a violation of the SUTVA assumption (Rubin, 1990) of the counterfactual methods. This is due to the possibility that people in the same cluster can affect each other's treatment value between the Year I assessment and the Year II assessment. However, we believe it is unlikely to cause a Type I error. The interference is likely to be exposed individuals exposing non-exposed individuals, after exposure was measured. Therefore it would most likely bias towards non-significance, though a bias towards significance is still possible.

Recall bias may have occurred, or some individuals may have answered untruthfully to appear more socially acceptable. While recall bias would pull the estimate away from the null, it is unclear if socially acceptable responses would bias the result in any particular direction. However, sensitive questions were administered via self-response to recorded questions, and recall bias should not be large enough to affect the results. It is possible that suicidal individuals are more likely to recall suicidal behavior in their peers. The sensitivity analysis adjusted for Year I suicidality and found similar results, which suggests that this bias is not causing this results. However, we cannot entirely rule out that this bias is occurring. Also, it is possible that the exposure and outcomes questions did not perfectly differentiate between suicidal and non-suicidal acts. Without follow-up questions to clarify the intent of the behavior, measurement error remains a possibility. Since exposure and outcome were measured over the same time period for the Year I outcome we are not able to determine the order of these events. The Year II outcome does not suffer from this limitation.

Incomplete data is also a potential problem. Although non-response to individual questions was very low, due to the large number of variables used in the propensity score prediction 13.1% of the sample participants did not have complete data. Trimming for the regression analysis means that only 82.1% of the Year II sample was used in the regression analysis. However, the portion removed by trimming should not bias the effect estimates (Stürmer et al., 2010). Lastly, the data used in this study was collected two decades ago. It is unlikely that there would be any significant difference between now and when the data was collected with respect to the hypotheses being tested. However the possibility of some change over time cannot be entirely ruled out.

## 5. Conclusion

This study failed to explain the association between exposure to peer suicidal behaviors and future risk of suicidal behavior based on pre-existing risk factors. These results support the suicide contagion explanation for this association. This increased risk is also not limited to those with pre-existing risk factors. Future research should examine whether interventions can mitigate the effect of this exposure.

## Funding source

Preparation of this article was supported by research grants from the University of Manitoba's GETs program (JR Randall), and by support from the Canada Research Chairs program (I Colman).

## Financial disclosure statement

None of the authors have any financial disclosures to declare.

## Conflict of interest

None of the authors have any conflicts of interest to declare.

## Contributor's statement

Jason R Randall: JRR conceptualized and designed the study, performed the data analysis, drafted the initial manuscript, and approved the final manuscript as submitted.

Nathan C Nickel, Ian Colman: NCN and IC conceptualized and designed the study, reviewed and made revisions to the manuscript draft, and have approved the final manuscript.

All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

## Acknowledgments

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by Grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

## Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jad.2015.07.001>.

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