How do the prevalence and relative risk of non-suicidal self-injury and suicidal thoughts vary across the population distribution of common mental distress (the p-factor)? Observational analyses replicated in two independent UK cohorts of young people

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Abstract:

Objectives: To inform suicide prevention policies and responses to youths at risk by investigating whether suicide risk is predicted by a summary measure of common mental distress (CMD, (the p-factor)) as well as by conventional psychopathological domains; to define the distribution of suicide risks over the population range of CMD; to test whether such distress mediates the medium-term persistence of suicide risks.

Design: Two independent population-based cohorts.

Setting: Population-based in two UK centres.

Participants: Volunteers age 14-24 years recruited from primary health care registers, schools and colleges, with advertisements to complete quotas in age-sex-strata. Cohort 1 is the Neuroscience in Psychiatry Network (NSPN; N=2403); Cohort 2 is the ROOTS sample (N=1074).

Primary outcome measures: Suicidal thoughts (ST) and non-suicidal self-injury (NSSI).

Results: We calculated a CMD score using confirmatory bifactor analysis and then used logistic regressions to determine adjusted associations between risks and CMD; curve-fitting was used to examine the relative prevalence of suicidal thoughts (ST) and non-suicidal self-injury (NSSI) over the population distribution of CMD. We found a dose-response relationship between levels of CMD and risk of suicide. The majority of all subjects experiencing ST and NSSI (78% and 76% in Cohort 1, and 66% and 71% in Cohort 2) had CMD scores no more than two standard deviations above the population mean; higher scores indicated the highest risk but were, by definition, infrequent. Pathway mediation models showed that CMD mediated the longitudinal course of both ST and NSSI.

Conclusions. NSSI and ST in youths reflect common mental distress that also mediates their persistence. Universal prevention strategies reducing levels of CMD in the whole population

without recourse to screening or measurement may prevent more suicides than approaches targeting youths with the most severe distress or with psychiatric disorders.

Strengths and limitations:

- The samples were population-based with several self-reported outcomes regarding suicidal risk.
- Replication of the findings in two independent cohorts strengthens confidence in the findings.
- Results were robust across different statistical models and approaches to data classification.
- Sample attrition was a limitation in both cohorts.
- Multiple imputations mitigated biases arising from attrition.

Introduction

Adolescence sees the onset of a range of psychopathology including suicidal thoughts (ST) and non-suicidal self-injury (NSSI)[1-3] that individually or together convey heightened risk of suicide attempts[4-6]. Non-suicidal and suicidal self-harm predict completed suicide[7], the second most common cause of deaths among 10 to 24-year-olds, worldwide[8]. Moreover, ST and NSSI are significant problems in their own right, representing a considerable burden to individuals, their families and health services. Prediction and prevention of self-harm and suicide in young people are priorities but NSSI (5-42% in community samples[9,10]) and ST (15-25% in community samples[11,12]) are common so it is difficult to predict who will ultimately make a serious attempt[13] or die by suicide. Indeed, the usefulness of clinical risk protocols relying on the identification of a psychiatric diagnosis is questionable[14,15]. The same problems affect public health suicide prevention programmes. A seminal study revealed a high prevalence of false-negatives in prospective identification of suicide[16]. Prevention policies that embrace the whole population might overcome these difficulties but lack theoretical or empirical foundations[1].

Suicidal thoughts and behaviours are routinely considered as markers of depression (e.g., in DSM-5) but by no means all young people dying by suicide have had a mood disorder[17]. NSSI is strongly associated with the risk of suicide when occurring in combination with any internalising or externalising symptoms[18,19], or with any psychiatric diagnosis[20], particularly multiple diagnoses[21]. Thus, this risk might be better predicted by multiple symptoms rather than by the presence of a single disorder, such as depression.

Recent studies suggest that a broad range of symptoms conventionally seen as components of distinct disorders are better construed as manifestations of a single, latent dimension distributed within the general population. This dimension has been variously referred to as the p-factor[22], general psychopathology[23] or, as we prefer here, common mental

distress (CMD)[24,25]. Parsimonious statistical models with dimensions that encompass low-prevalence phenomena such as psychotic experiences, fit empirical data better than models with distinct disorders[22,26]. High co-morbidity of psychiatric diagnoses, shared causal factors and treatments, and trans-diagnostic psychological and neural correlates support the validity of a CMD concept[22-24,26-29]. Suicide risk is related to multiple symptoms or disorders (and thus to higher CMD scores), not the presence of one specific symptom or disorder, so it is important to understand the nature of dose-response relationships between CMD and suicide risks. This could guide a clinical response in the face of suicide risk[30] and also shape population-based suicide prevention.

In this study, we describe the presence of a CMD dimension in young people aged 14-26 years and the occurrence of ST and NSSI referred to collectively, hereafter, as a suicide risk. We draw on a psychometric study[25] that demonstrated high theoretical validity and high measurement qualities of the CMD factor comprising measures of common mental illness (depression, anxiety, psychotic experiences, obsessions and compulsions) as well as traits and characteristics commonly considered to contribute to the general level of mental health (antisocial trait, well-being, self-esteem). Our approach had three steps whereby we:

1. Tested associations between CMD and suicide risk, and contrasted CMD with specific psychopathological domains, exploring the utility of this summary measure;
2. Defined the prevalence and relative risk of NSSI and ST across the distribution of CMD;
3. Established whether the CMD_{T2} dimension measured at time 2 mediate the relationship between ST_{T1} and $NSSI_{T1}$ at time 1 and $NSSI_{T3}$ and ST_{T3} at time 3.

We used data from two population-based cohorts with complementary designs and very similar measures. In step two we used cross-sectional data from Cohort 1, time 1 (used as a

discovery sample) and Cohort 2 (used as a stepwise replication sample); in the third step we used three longitudinal waves of Cohort 1 (see details in Method).

Method

Study Design and Participants

Cohort 1

Participants in the NSPN 2400 Cohort[31] were recruited largely via postal invitations sent through general practitioners and schools in Cambridgeshire and Greater London, UK. Data collection was carried out in two research centres: University College London and the University of Cambridge between November 2012 and December 2016. Purposive sampling obtained at least 200 males and 200 females from the community in 5 age groups: 14-15, 16-17, 18-19, 20-21, 22-24 years. Three data collections took place a year apart (T1-T3). At T1, 2403 individuals returned questionnaires (average age 18.9 years, SD=3.0; 54% females); at T2, 1815 returned questionnaires (76% response, average age 20.0 years, SD=3.1; 56% female), and 1245 at T3 (52% of baseline; average age 21.0 years, SD=3.1; 59% female).

Cohort 2

The ROOTS study[32] was used for replication of findings from Cohort 1. Two-stage sampling involved random selection of 27 schools in Cambridgeshire, UK. Eighteen schools agreed to participate; invitations were sent to 14-year-olds randomly selected from class registers and to their parents; 1238 students participated in the initial data collection (55% female) (and further 4 data collection waves took place). Note that in the current analysis we used only the data from the third data sweep collected between February 2008 and December 2009, when participants were of average age 17.5 years, *SD*=0.3 (N=1074, 56% female; 87% of baseline sample), the closest age to T1 of Cohort 1.

Both cohorts comprised predominantly white European (77% in Cohort 1 and 87% in Cohort 2) young people, consistent with the self-ascribed demographics of the two study populations. Written consent from participants age 14 or 15 years was supplemented by written consent from their parent or legal guardian; older participants gave their own written consent. Ethical approval was obtained for Cohort 1 from the National Health Service Research Ethics Service (# 97546) and for Cohort 2 from the Cambridgeshire 2 REC (# 03/302).

Measures

Sociodemographic information was collected using routine methods[31,33]. The Index of Multiple Deprivation (IMD), a summary measure of the socioeconomic status of participants' residential neighbourhood, is calculated from census information[34]. Questionnaires of mental illness and wellness are set out in Table 1 and items are listed in the Supplementary table 1. Scores in questionnaires were computed according to published manuals or validation studies (cited in Table 1), standardized to unify their measurement scales.

Table 1

Statistical analysis

Confirmatory bifactor analysis with a weighted least square mean and variance adjusted (WLMSV) estimator in Mplus 7.4 was used to compute factor scores for CMD in the three data sweeps of Cohort 1 and Cohort 2 based on the model validated elsewhere[25] (see CMD measures in Table 1 beneath; the list of used items and details of bifactor modelling can be found in the Supplementary table 1). CMD factor scores were then used in all subsequent computations. Next, we addressed attrition in Cohort 1 by means of multiple imputations (see details in the Supplement).

To prove that NSSI and ST were predicted by multiple psychopathological domains and also by CMD (which represents a summary of those domains), we used Stata 12 to

compute for Cohort 1_{T1} and Cohort 2 data sensitivity / specificity indicator – the area under the curve (AUC – reported in the Supplementary table 2) for NSSI and ST as criteria. We computed a series of logistic regressions, estimating odds ratios (OR) with confidence intervals for each predictor (treated as categorical with the cut-off point above 1SD and then continuous), while we controlled for effects of age and sex (Figure 1).

For step two, distributions of CMD scores in both cohorts were plotted against lines representing percentages of subjects reporting NSSI and ST within bands of CMD expressed as standard deviations (upper panel of Figure 2) and against bar histograms representing NSSI and ST frequencies in both cohorts (lower panel of Figure 2). In addition, NSSI and ST information curves were computed to determine in what range of the CMD dimension these items are located (see Supplementary figure 1).

Using Cohort $1_{T1\text{-}T3}$ data for step three, we examined the longitudinal relationship between CMD, NSSI and ST (in particular the predictive role of CMD in persistence of NSSI and ST): we computed direct and mediation (via CMD_{T2}) effects of ST_{T1} and $NSSI_{T1}$ on $NSSI_{T3}$ and ST_{T3} in a pathway mediation model with confidence intervals in Mplus 7.4 (computing bias-corrected bootstrapping was not possible due to the use of multiply imputed datasets). We computed this model for the total sample (Figure 3) and then for both sexes separately (Supplementary figure 2) using the Multiple Group Method, so as to test a moderated-mediation model (with CMD_{T2} as a mediator, and sex as a moderator). Age was a control variable. In both pathway analyses CMD_{T2} factor scores (computed on imputed data, as described above) were modelled as observed variables.

Results

Step one: Associations of NSSI and ST with demographic and psychopathological variables

In both cohorts NSSI and ST were unrelated to demographic variables, including sex and age (See Supplementary tables 3 and 4); CMD was negatively related to male gender (Supplementary table 5). When examined descriptively over the pooled age groups, the prevalence of NSSI and ST mirrored the CMD levels (see Supplementary figure 3). CMD and all "conventional" mental health disorders predicted NSSI and ST (i.e., had statistically significant ORs in logistic regression models - see Figure 1 and Supplementary table 2).

Figure 1

Prevalence of NSSI and ST in the two cohorts

In Cohort 1 (N=2403) there was no statistically significant change in the prevalence of NSSI (within the last month) over the three time points: in the imputed data 9.3% (n=223) reported $NSSI_{T1}$, 8.3% (n=199) $NSSI_{T2}$ and 8.2% (n=197) $NSSI_{T3}$. Similarly, there was no statistically significant change in prevalence of ST (within the last two weeks) over the three time points: 10.1% (n=243) ST_{T1}, 11.4% (n=274) ST_{T2} and 11.7% (n=281) ST_{T3} (see Supplementary tables 6 and 7).

In Cohort 2 (N=1074), 11.7% (n=126) reported lifetime NSSI and 5.4% (n=58) reported ST within the two last weeks. Accuracy and precision of these prevalence estimates were affected by attrition (see *Discussion: limitations*). Attrition in Cohort 1 at T2 and T3 was only marginally related to demographic and exposure variables at T1 (Spearman's rho 0.05-0.12), but unrelated to the outcome – NSSI and ST (see Supplementary table 8).

Step two: Associations of NSSI and ST with CMD

Next, we focused on absolute risk[i] and the numbers of NSSI and ST events generated by these risk functions. The dose-response curves in the upper panel of Figure 3 show that relative risks[ii] of NSSI and ST increased markedly with increasing severity of CMD, the highest risks being in those with very high scores beyond two standard deviations above the

mean. On the other hand, most participants from both cohorts who reported NSSI or ST had mild (one SD above the mean) to moderate (two SD above the mean) CMD scores (lower panel of Figure 3). CMD was normally distributed (see Supplementary figure 4) so these scores were much more common; only a minority of the total reports came from the few participants with very high CMD (>2 standard deviations above mean CMD). Thus, the majority of subjects experiencing ST or NSSI (Cohort 1: 78% and 76%; Cohort 2: 66% and 71%, respectively) had CMD scores within two standard deviations above the population mean. Very high CMD scores indicated the highest suicide risk but were rare, so generated the minority of events.

Figure 2

Step three: Mediating effect of CMD on suicide risks in Cohort 1 over time

Cohort 1 CMD_{T2} contributed to the persistence of NSSI and ST over time (i.e. $NSSI_{T1}$ predicted $NSSI_{T3}$ directly, and via mediation through CMD_{T2}; it also completely mediated the longitudinal effect of $NSSI_{T1}$ on ST_{T3}). Moreover, CMD_{T2} contributed to the persistence of ST over time (i.e. ST_{T1} predicted ST_{T3} directly, as well as via mediating variable – CMD_{T2}). Overall, CMD_{T2} was a stronger predictor of $NSSI_{T3}$ and ST_{T3} than the antecedent variables measured at T1 (see Figure 3). The mediation effects of CMD_{T2} were similar for boys and girls (i.e., the effects were not moderated by sex – Supplementary figure 2 and Supplementary table 9). Age_{T1} was not a significant predictor of any variable in the model; the results when age was controlled for were very similar to those without controlling for age.

Figure 3

Discussion

In the present study, all the domains of psychopathology and mental wellness available (depression, anxiety, self-esteem, well-being, psychotic-like experiences, antisocial trait, schizotypal trait, conduct problems, obsessions and compulsions) predicted risk of non-suicidal self-injury (NSSI) and suicidal thoughts (ST). Thus, the common mental distress factor with a normal population distribution appeared a parsimonious and efficient summary of these domains and was, itself, a key predictor of suicide risk in both cohorts. NSSI and ST were not confined to participants scoring in the very high, quasi-clinical range for CMD. Around half of all participants expressing NSSI or ST came from those scoring up to one standard deviation above mean CMD in a dose-response manner. The majority expressing these phenomena (two thirds to three quarters) scored within 2SD above the mean (Figure 2). Regarding medium-term determinants of persistent NSSI and ST we showed (Figure 3) that CMD_{T2} mediated the persistence of NSSI and ST over two years, independent of gender and age. This mediation operates in two stages: first, ST and NSSI persist because these behaviours are markers for worsening CMD in the general population. This extends findings in adolescents with depressive disorder, where suicidal thoughts are a predictor of poor outcome[35]. Second, this greater CMD, itself, predicts the risk for further suicidal thoughts and behaviours.

Strengths

Both cohorts were designed on epidemiological principles to capture behavioural and psychological variation in the population during the post-pubertal epoch during which risk for psychopathology accelerates. Replication of the findings in these independent cohorts strengthens confidence in the findings, as does internal consistency between cross-sectional associations found in both cohorts, and longitudinal associations found in Cohort 1.

Limitations

Sample attrition was the main bias in both cohorts. Each retained more young women than men; we found marginally higher attrition among lower socio-economic class, participants of non-white ethnicity and those with higher CMD (Supplementary table 8). Cohort 1 is robustly representative of the England and Wales population[31], whereas Cohort 2 under-represents participants with lowest socioeconomic status[32]. However, we have no reason to suppose that attrition biased our results, as it was unrelated to NSSI and ST (Supplementary table 8). If there was a bias, it probably limits power rather than skewing an effect and is mitigated by replication between the cohorts. We used multiple imputation to minimise this bias.

There was only modest reliability of our obsessionality measure and a skewed measure of conduct problems in Cohort 1. A completely comprehensive range of psychopathological (and behavioural) items was unavailable; we did not have measures of unstable or abnormally elevated mood, addictions, eating disorders or hyperactivity. Thus, our measurement of CMD focused primarily on internalising rather than externalising symptoms. Future studies could include a broader range of measures and extend the investigation into clinical populations to improve measurement precision at the highest levels of CMD. Although ethnicity and socioeconomic status (indicated by IMD) were unrelated to ST and NSSI (Supplementary tables 3 and 4), and thus were not included in our analyses, we did not control for the effect of other possible confounders such as adverse life experiences, early trauma, family structure or more detailed information about family socio-economic situation (unemployment, poverty etc.). Finally, we could not account for the effects of clustered design in the modelling, due to unavailability of the information about clustering of participants in both cohorts.

Implications & Conclusions

Our findings provide yet more evidence that a latent mental distress factor, conceptually akin to the p-factor, is a useful summary measure of psychopathology in the general population[24], diagnostic[22], and clinical[23] samples. We speculate that psychopathological items accumulate in a probabilistic manner rather than in diagnostic clusters, with common phenomena concerning depression and anxiety much more likely to occur before rarer phenomena such as NSSI, ST or psychotic experiences. Less frequent phenomena begin to co-occur as the severity of psychological disorder (or CMD) increases, in terms of more mental and behavioural phenomena or symptoms. This begins to yield clusters linked by common items that current diagnostic systems tend to ignore. This is consistent with the co-occurrence of suicidal risk and psychotic experiences seen in other[36-38] studies of young people, and with the present IRT analysis showing that NSSI and ST are measuring the higher end of CMD (Supplementary figure 1). The approach we have followed illustrates the value of moving away from categorical classification and embracing an empirically-rooted, dimensional, hierarchical taxonomy in psychopathology research[39]. Such hierarchical approaches to phenomenological classification had been put forward before[40] or shortly after[41] the publication of DSM-3 and its successor classifications. Hierarchical models merit renewed interest[42], as they may resolve problems of comorbidity[26] as well as overlapping causes and biological mechanisms for suicide risk and other phenomena[43,44]. In contrast to the CMD idea, there is also increasing interest in approaches focusing on individual symptoms and experiences, particularly to guide individual clinical interventions, rather than grouping the symptoms into diagnostic categories or higher-order constructs[45]. Future studies may investigate and compare the utility of such novel approaches (CMD and item-focused approach) for clinical practice and public health policies.

Our findings also have major implications for intervention and prevention of suicidal thoughts and behaviours. Clinically, the results suggest that NSSI and ST should never be dismissed or downplayed when they occur in young people without clear evidence of psychiatric disorder, a logical fallacy because NSSI and ST are *themselves* indicators of higher distress on a CMD factor. NSSI and ST will usually, but not always occur with other, more common psychopathology and their co-occurrence is a strong risk factor for suicide attempts[6]. Thus, NSSI and ST merit a swift professional response regardless of whether or not they occur with other symptoms that take individuals beyond conventional clinical thresholds and trigger traditional clinical risk protocols. Our findings help explain why research focused on high-risk subjects has yet to translate into useful clinical prediction tools[14,15,45].

From a public health and prevention perspective, the fact that rates of NSSI and ST begin to accelerate at levels of CMD well within a normal or non-clinical range argues strongly for universal interventions overtly aimed at lowering the population mean CMD and shifting the curve to the left. This should be alongside targeted approaches and effective clinical services[46]. Strategies concentrated on clinical populations, those with evidence of a psychiatric disorder or other individual markers will miss the majority of individuals experiencing ST or engaging in NSSI because there are so few compared with those at lower risk: the *prevention paradox*[30].

Defining putative universal interventions to shift the population distribution of CMD will require careful research that can draw from other areas of medicine such as cardiovascular disease and stroke[30]. Elements have been widely scoped in the USA[15] and elsewhere, but not for constructs of population health and wellbeing such as CMD. Interventions may involve decreasing common triggers[15] or improving young people's abilities to cope with stressors[47, 48, 49].

Conflict of Interest Disclosures

E.P., S.N., I.M.G., and J.S. have no competing interests. E.B., P.F., and P.B.J. are in receipt of National Institute for Health Research (NIHR) Senior Investigator Awards (NF-SI-0514-10157, and NF-SI-0514-10117. P.F. was in part supported by the NIHR Collaboration for Leadership in Applied Health Research and Care (CLAHRC) North Thames at Barts Health NHS Trust. P.W. has recent/current grant support from NIHR, Cambridgeshire County Council and CLAHRC East of England. P.W. discloses consulting for Lundbeck and Takeda; P.B.J. discloses consulting for Janssen and Ricordati. E.B. is employed half-time by the University of Cambridge and half-time by GlaxoSmithKline in which he holds stock.

Data sharing/ Data availability

E.P. had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The data are deposited in the University of Cambridge Data Repository, with the placeholder DOI https://doi.org/10.17863/CAM.25331 available to researchers via openNSPN@medschl.cam.ac.uk.

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Role of the Funder/Sponsor

The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

NSPN Consortium Information –see the Supplement

Patient and public involvement: Patients and the public were not involved in the design or planning of the study.

Table 1. Measures used in both cohorts

Variables	*Measures*	Cohorts	
Outcome variables:		$NSPN_{T1-T3}$ (1)	$ROOTS_{age\ 17}$ (2)
Suicidal thoughts (ST)	One item from the MFQ[50]: I thought about killing myself. Responses were recoded into a binary format: no ST (original response option *Never)* and ST (original response options *Sometimes* or *Mostly* or *Always*).	×	×
Non-suicidal self-injury (NSSI)	One question from the Drug, Alcohol and Self-Injury (DASI)[25] questionnaire asking about engaging in self-injury without suicidal intent during the last month. Responses were recoded into a binary format indicating the occurrence of NSSI or lack of thereof.	×	
	One question asking about the occurrence of lifetime NSSI (DASI)[25]		×
Predictors:			
Conduct problems	11-item Antisocial Behaviour Questionnaire[25]	×	×
Anxiety	28-item Revised Children's Manifest Anxiety Scale[51]	×	×
Depression	29 items from the 33-item MFQ[50] (all items except for 4 items measuring suicidality)		
Obsessions and compulsions	11-item Revised Leyton Obsessional Inventory[52]	×	×
Psychotic-like experiences	11 items selected from the 74-item Schizotypal Personality Questionnaire (SPQ)[53]	×	
	11 items from the 20-item semi-structured interview from the Diagnostic Interview Schedule for Children-IV[54]		×
Self-esteem	10-item Rosenberg Self-Esteem Questionnaire (*)[55]	×	×
Well-being	14-item Warwick-Edinburgh Mental Well-Being Scale(*)[56]	×	×
Impulsivity	15 items from the 30-item Barratt Impulsiveness Scale[57] selected based on exploratory factor analysis - loadings above .25	×	
Antisocial traits	Total score from the 17-item Antisocial Process Screening Device (APSD)[58]	×	
Schizotypal traits	Total score from the 74-item Schizotypal Personality Questionnaire (SPQ) [53]	×	×

*scales were reversely scored, thus higher scores indicated lower self-esteem and well-being; for all other measures higher score indicates more psychopathology

Figures' legends:

Figure 1: Odds ratio in logistic regressions for suicidal thoughts (ST) and non-suicidal self-harm (NSSI) as outcomes predicted by psychopathological predictors (listed on the left) here treated as continuous variables; regressions were computed separately for each predictor and effects of age and sex were controlled in each regression for in both cohorts (see Supplementary Table 2).

Figure 2: Upper panel shows the dose-response effect of Common Mental Distress on non-suicidal self-harm (NSSI) and suicidal thought (ST) in Cohort 1 and Cohort 2. The lower panel shows the proportion of total reports in non-suicidal self-injury (NSSI) and suicidal thought (ST) broken down by standard deviations of Common Mental Distress; these add up to 100% from left to right. The normal population distribution of CMD, which was strikingly similar, but not identical, in Cohort 1 and 2, is shown by the purple line (see density plots in Supplement, Figure 1).

Figure 3: Mediation effect of Common Mental Distress at time 2 in Cohort 2: Standardised pathway coefficients with confidence intervals in square brackets.

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P.B.J. – provided senior supervision, conceptualised the study, advised on statistical analyses, read and critically apprised the manuscript, re-drafted and edited the manuscript

J.S. – provided statistical advice, replicated multiple imputations, provided data from multiple imputations, read and critically apprised the manuscript

S.N. – advised on handling missing data, replicated multiple imputations, read and critically apprised the manuscript

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[i] Absolute risk is the probability or chance of an event. It is usually used for the number of events (e.g., a suicide) that occurred in a group, divided by the number of people in that group.

[ii] A relative risk compares the risk of a health event (e.g., a suicide) among one group with the risk among another group.