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Heavy episodic drinking and deliberate self-harm in young people: a longitudinal cohort study

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Abstract

Aim To assess the association between heavy episodic drinking (HED) and deliberate self-harm (DSH) in young people in Norway.

Design, setting, participants, and measurements We analysed data on past year HED and DSH from the second (1994) and third (1999) waves of the Young in Norway Longitudinal Study (cumulative response rate: 68.1%, n = 2681). Associations between HED and DSH were obtained as odds ratios and population attributable fractions (PAF) applying fixed-effects modelling, which eliminates the effects of time-invariant confounders.

Findings An increase in HED was associated with a 64 % increase in risk of DSH (OR = 1.64, *P* = 0.013), after controlling for time-varying confounders. The estimated PAF was 28% from fixed-effects modelling and 51 % from conventional modelling.

Conclusion Data on Norwegian youths show a statistically significant association between heavy episodic drinking and deliberate self-harm.

Keywords: heavy episodic drinking, deliberate self-harm, young people**,** panel data, prospective, fixed effects modelling, Norway

INTRODUCTION

Suicide and non-fatal deliberate self-harm (DSH) constitute a substantial fraction of the disease burden in young people (1). Among adolescents, past year DSH prevalence tends to be around 5% to 10% (2) and thus involves significant health services costs. Non-fatal DSH is also an important risk factor for completed suicide, and tends to co-occur with other mental health and behavioural problems in young people (3). Self-cutting and overdose are the most common methods of DSH in adolescents. A wish to get relief from a terrible state of mind, and wanting to die, are the most common motives for harming oneself (4). It is well established that alcohol consumption is a significant risk factor for DSH (5-7). There are several underlying mechanisms that may explain the observed associations between alcohol consumption and DSH (8, 9). In our context, addressing young people, it is alcohol use as a proximal—or precipitating—causal factor that is of particular relevance (9, 10). It is suggested that the acute effects of alcohol intoxication may trigger DSH through various mechanisms: they may increase psychological distress, increase aggressiveness, propel suicidal ideation into action, and constrict cognition and implementation of alternative coping strategies (8, 9). Acute alcohol use may thus be a significant causal factor in a prevalent health problem, implying that prevention of heavy drinking episodes (HED) can be an important strategy to prevent DSH. We will in this study, therefore, submit the association between HED and DSH to a stronger test for causality than what is common practice in this field.

The magnitude of the association between HED and DSH has been assessed in various ways. It has often been found that a sizeable proportion of suicide attempts and other cases of deliberate self-harm occurred in a state of alcohol intoxication (11). For instance, across 16 studies on acute alcohol use and suicide attempt, the mean percentage of suicide attempts under the influence of alcohol was 40% (range 10% to 73%) (12). It has also been found that

DSH is more prevalent among those who drink to intoxication, and that the DSH risk tends to increase with increasing frequency of HED (13-16). These associations are, however, of a correlational nature, and do not necessarily indicate the magnitude of alcohol's causal role in the self-destructive act. For instance, alcohol may, in some cases, be drunk in order to relieve pain or remove barriers to hurting oneself (8), and in such cases it is rather part of the act, than a cause of it. The proportion of DSH involving acute alcohol use may therefore overestimate the causal role of alcohol. On the other hand, it is also possible that alcohol intoxication has an indirect effect on DSH by having negative consequences (e.g. break-up with partner, property destruction) that in turn enhance negative feelings and thus may trigger DSH. If such indirect effects were larger than alcohol's role as a mere part of the act, the proportion of DSH involving acute alcohol use would be an underestimate of alcohol's causal role. Moreover, the elevated risk of DSH among those who frequently drink in excess may — at least in part — be attributed to some shared risk factors and therefore represent a biased estimate of a causal association. There are indeed many individual and environmental factors which are likely to confound the association between HED and DSH, including genetics, personality traits, dysfunctional family background and childhood adversities, social network, and mood disorders (17, 18), all of which are, in practice, unlikely to be captured within one and the same study. One method to overcome at least part of the problem with biased estimates due to unmeasured shared risk factors (confounders) is fixed-effects modelling (19, 20). This approach addresses the question, is a change in HED associated with a change in DSH? In essence, this method eliminates confounding due to shared risk factors that are stable across time (e.g. genetics, personality traits, and childhood trauma). Within alcohol and drug research this method still appears underutilised (21), and only a few studies have employed the method to assess causal associations and attributable fractions addressing the role of

alcohol (or other substances) in health and social problems that are typically multifactorial and complex (22, 23).

The magnitude of an association is often estimated as an (adjusted) odds ratio (OR) or relative risk (RR). From a public health perspective it is, however, rather the potentially preventable fraction of disease due to a specific risk factor that is of interest. This fraction is often referred to as the population attributable fraction (PAF) or preventive fraction (24, 25). Estimates of PAF are rarely seen within the literature on deliberate self-harm, in general (24), and the studies that address the role of alcohol use for DSH are no exception to this. There are indeed estimates of DSH attributable to substance use disorders (26), estimates of suicide mortality attributable to HED (27), and estimates of DSH attributable to HED in adolescents (15). But, as these estimates are all based on analyses of cross-sectional data with inherent limitations to the ability to control for confounding, they are likely to be biased.

Against this backdrop the present study will use two-wave panel data to assess the association between heavy episodic drinking and deliberate self-harm in young people by applying fixed-effects modelling to account for stable factors that are likely to confound a causal relationship. Although fixed-effects modelling eliminates the risk for confounding due to covariates that are stable across time, it does not remedy bias due to time-varying factors that affect the outcome as well as the predictor variable. Thus, we considered the following time-varying potential confounders. *Parental heavy drinking* is shown to be associated with both DSH (28) and HED (29). *Poor social network* has previously been shown to be associated with DSH (6) and HED (18), yet in opposite directions. *Depressive symptoms* are a well-established risk factor for DSH (17). While depressive symptoms are also found to be associated with HED in young people, it is less clear to what degree this association is causal, and if it is, which direction causality takes (18).

MATERIAL AND METHODS

Data

Data were obtained from the Young in Norway Longitudinal Study, which has followed a cohort of young people prospectively over a 13 year period, covering a broad range of topics (30). The data that we required were collected in the second (1994) and third (1999) waves. The initial sample was obtained in 1992, and the sampling procedures were designed to obtain a nationwide, representative cross-section of the student population in junior and senior high school (grades 8 through 13) in Norway. All students in the selected schools were included in the 1992 survey (response rate: 97%) and in the 1994 survey. The follow-up in 1999 was confined to respondents who attended 7th or 10th grades in 1992. A total of 2897 respondents participated in both 1994 and 1999 (cumulative response rate: 68.1%). These data collection waves are referred to as T1 and T2 below. Response rates and sample characteristics at T1 and T2 are presented in Table 1. In 1994, questionnaires were distributed and completed in the classroom during a 2-hour session, while a postal survey was carried out in 1999—which partly explains the drop in the response rate. The prevalence of missing data on the key variables in the present study was 8.6%, implying that our analyses included 2647 respondents.

Table 1 here

Measures

Deliberate self-harm was measured by the question, "Have you ever on purpose taken an overdose of pills or in another way tried to hurt yourself?" Those who responded affirmatively were asked how long it had been since the (most recent) episode of DSH. Based on the responses, a variable on past year prevalence of DSH (yes/no) was constructed. This

measure captures both suicide attempts and non-suicidal self-inflicted injuries and has been used in several previous studies of DSH (4, 31, 32).

Heavy episodic drinking (HED) was measured by the question, "During the past 12 months, have you had so much to drink that you felt clearly intoxicated?" There were 6 response options: never (coded 0), once (1), 2 to 5 times (3.5), 6 to 10 times (8), 11 to 50 times (30), and more than 50 times (55).

Time-varying covariates

Parental heavy drinking was measured by a single question about how often the respondents had seen their parents intoxicated. There were 5 response options ranging from never (coded 0) to several times a week (4).

Poor social network was measured by the UCLA Loneliness Scale (33). This scale comprises 5 items with 4 response options ranging from never to often (Cronbach's Alpha = 0.72). The responses were averaged over the five items and the sum score ranged from 1 to 4.

Depressive symptoms were measured as an additive index based on 6 items from Kandel and Davies's Depressive Mood Inventory (DMI) (34): (1) Felt too tired to do things; (2) Had trouble sleeping; (3) Felt unhappy, sad, or depressed; (4) Felt hopeless about the future; (5) Felt tense or keyed up; and (6) Worried too much about things. There were 4 response options: not bothered at all (1), a little bit bothered (2), quite bothered (3), and extremely bothered (4) (Cronbach's Alpha= 0.81). All the above mentioned measures were obtained at both T1 and T2.

Statistical analyses

The data were analysed by applying the first difference (FD) method, which is a more specific form of fixed-effects modelling. In practice, this means that we first calculated change scores

for both self-harm and drinking by subtracting the value at T1 from the value at T2. Next, these variables were collapsed into trichotomous variables taking the values increase (1), stable (0), and decrease (-1). Lastly, these variables were used in ordinal regression analysis. This is an extension of logistic regression to situations where the outcome is an ordinal variable with more than two values (35). Difference scores from T1 to T2 of the time-varying potential confounding variables were correlated with input (HED) and outcome (DSH) measures and those variables that were statistically significantly correlated with both HED and DSH were included in the regression analysis. Considering that the sampling was clustered by school we used clustered robust standard errors with school as cluster variable (36). We also considered the alternative of multilevel modelling. However, our analytic method was supported by the outcome from such a model: the estimated effect of the level 2 factor (school) was clearly insignificant ($b=0.454$; $SE=18.322$, $p>0.99$), and its inclusion affected the estimated HED-effect with a factor equal to 1.00004, while the SE of the estimated HED-effect was affected by a factor equal to 0.9904.
All statistical analyses were conducted using Stata (version 12, Stata Corp, College Station, Texas, USA).

With regard to depressive symptoms, the causal ordering between depression and drinking is, as noted, far from conclusively established. If depression precedes drinking, it should be included as a control variable, but if depression is mediating the alcohol effect, its inclusion will attenuate the estimated impact of drinking. Rather than taking a definite stance on this, we estimated two models, that is, one with and one without depressive symptoms as control variable.

Finally, we estimated the fraction of DSH attributable to HED, applying the formula

$$PAF = \frac{PF * (RR - 1)}{PF * (RR - 1) + 1}$$

where PAF is the population attributable fraction, and PF is the population fraction exposed to the risk factor. RR is the relative risk which was obtained from the estimated odds ratios following standard procedure (37):

$$RR = \frac{OR}{(1 - p_0) + (p_0 * OR)}$$

where p_0 is the prevalence positive among unexposed (in our case, the incidence of DSH at T2 among those who reported no HED at T1).

Whether control for time-invariant confounders in fixed-effects modelling makes a difference, was assessed by comparing the PAF estimates based on the FE-models with the PAF estimate calculated from the cross-sectional data at T1 and T2 (controlling for all available confounding factors in logistic regression models).

RESULTS

At T1 a total of 3.2% of the sample (n = 93) reported DSH in the preceding 12 months, while the corresponding figures at T2 were 1.6% (n = 45) (Table 1). A larger proportion of girls reported DSH at T1 and T2 (4.7% and 2.3 %, respectively) compared to boys (1.4 % and 0.6 %, respectively). Less than one in five who reported DSH at T1 (n = 8) did so also at T2. Thus, from T1 to T2 the vast majority of the respondents (95.5%) were DSH stable; that is, most reported no DSH at both T1 and T2 (n = 2767), while a few reported DSH at both waves

(n = 8), whereas smaller fractions had decreased DSH (2.9%; n = 85) or increased DSH (1.3%, n = 37) (Table 2).

Table 2 here

Regarding HED, at T1 63.5% reported having been clearly intoxicated once or more often in the preceding year, and 27.1% had been intoxicated more than five times in the past year. At T2 the corresponding figures were 86.2% and 58.6%, respectively (Table 1). No significant gender difference in HED frequency was observed at T1, whereas at T2 boys reported more frequent HED as compared to girls. A majority of the respondents (60.5%, n = 1689) had increased their frequency of HED, whereas a fourth (25.4%, n = 708) were stable from T1 to T2, and a smaller fraction (14.1%, n = 395) had decreased their frequency of HED from T1 to T2 (Table 2). Thus, while the prevalence of HED increased markedly from T1 to T2, the small change in DSH went in the opposite direction. This suggests that any possible effect of the upward shift in drinking on self-harm is masked by much stronger counteracting effects of factors related to increasing age.

We then analysed the changes in DSH and HED from T1 to T2 and regressed the former on the latter in ordinal regression models. In the bivariate fixed-effects model, an increase in HED was associated with a statistically significant OR of self-harm equal to 1.75. Difference scores for depressive symptoms and poor social network were both statistically significantly correlated with the difference scores for both DSH and HED and were therefore included in the multivariate model, whereas this was not the case for parental heavy drinking ($P > 0.10$). When controlling for changes in poor social network, the estimate was roughly the same (OR = 1.80, $P=0.004$). When changes in depressive symptoms were also included in the model, this estimate dropped marginally (to 1.64), and statistical significance was retained ($P= 0.013$). Although a decrease in HED was associated with a reduced risk of self-harm, this

estimate was not statistically significant. The OR of 1.75 yields a population attributable fraction of 30%, while the OR equal to 1.64 yields a PAF of 28% (Table 3). The corresponding estimate based on cross-sectional analysis was 51%. The latter estimate was obtained as an average of results from separate analyses of data for T1 and T2 as described above.

Table 3 here

Including gender as covariate in the model did not alter the estimates or statistical significance of the other covariates, and the estimate of gender itself was not statistically significant. This reflects the fact that there was no gender difference in the rate of change in DSH between T1 and T2. We also performed gender-specific modeling. The association was statistically significant in girls (OR= 1.64, *P*=.038), and not statistically significant in boys (OR=2.49, *P*=.05). The difference between the gender-specific estimates was not statistically significant (t-value=0.80).

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DISCUSSION

This study has demonstrated a significant association between heavy episodic drinking and risk of deliberate self-harm in young people, when applying fixed-effects modelling with adjustment for time-invariant confounders and some additional time-varying confounders. By applying this type of modelling, the estimated fraction of deliberate self-harm attributable to heavy episodic drinking was lower than when applying conventional modelling adjusting only for available confounding factors.

Our finding of a significant HED–DSH association in young people is well in line with what has been found in previous work (6, 7, 11), that is, an increased likelihood of DSH with increasing HED. This association has previously been found for DSH with and without

suicide intent (11). The premise of our analyses is that any alcohol effect is contemporaneous and it seems most likely that the direction of the association is that HED exposure increases the DSH risk. Clinical studies have frequently found that DSH has occurred under the influence of alcohol and thus that HED immediately preceded DSH (12). Mechanisms that may explain how HED may affect DSH include the following consequences of HED: increased psychological distress, increased aggressiveness, propelled suicidal ideation into action, and constricted cognition and implementation of alternative coping strategies (8, 9).

When we consider the previous studies that have addressed the fraction of DSH attributable to alcohol use, these have arrived at estimates that vary markedly in magnitude, that is, in the range from 8% to 50% (15, 26, 27). This variation probably reflects in part varying methods applied in the studies, and in part the varying role of alcohol use in DSH across drinking cultures (15). Compared to a few previous studies of the HED–DSH association among Norwegian adolescents based on cross-sectional data (13, 15), our estimate of PAF from cross-sectional data is fairly similar to that reported in one of these (15) but much higher than in the other (13).

Study strengths and limitations

By applying fixed-effects modelling, we obtained an estimate of the alcohol-attributable fraction of DSH in young people that was lower than the estimate based on conventional modelling, which suggests that it is less subject to bias due to insufficient control for confounders. This method requires longitudinal data with identical measures of outcome and exposure variables, preferably supplemented with time-varying confounders, which are rare in this area (38). Further, a large sample is required to obtain sufficient statistical power when addressing fairly low-prevalence phenomena such as DSH. According to our literature survey,

none of the few studies that have assessed the fraction of DSH attributable to HED has used a design that considers the likely impact of confounding factors. Thus, this study adds to a meagre literature on this topic.

However, while the study sample was relatively large compared to similar longitudinal cohort studies of young people (e.g. the Christchurch Health and Development Study (39); the Dunedin Multidisciplinary Health and Development Study (40), Rutger's Health and Human Development Project (41)), it proved to be on the small side for the gender-specific analyses. Furthermore, there is certainly a large number of causes of DSH that were not included in our analyses, and an important issue is how that may have affected our findings. While our methodological approach (fixed-effects modelling) implies that the omission of time-invariant causal factors (e.g., genetic factors) should not yield any bias, the omission of time-varying causal factors that covary temporally with HED, will indeed give rise to bias. We did include some time-varying potential confounders which affected the outcome somewhat. Nevertheless, there may well exist omitted time-varying confounders- for instance related to adolescent development - implying that the estimate of the HED-DSH association may be biased in either direction . Moreover, the probable presence of measurement errors in the explanatory variable (HED) produces a downward bias and it is impossible to ascertain the net effect of the various sources of bias. The DSH measure that we used captures self-harm with and without suicidal intent (4) and the prevalence figure may include false positive and false negative responses; i.e. some students who responded affirmatively may not have filled the criteria for DSH (32) and others may be underreporting DSH. Another limitation is that the data were collected some time ago and over a period when youth drinking in Norway increased (13) which may have impacted the results.

Finally, previous research suggest that the association between drinking and DSH varies markedly across countries (15, 42). It thus seems warranted to probe the generalizability of our findings by analyses of data pertaining to other cultural contexts.

Implications

The finding that a fairly large proportion of DSH is attributable to HED among young people implies that there is a significant potential to prevent DSH cases in young people by curbing their alcohol consumption. Several strategies have proven to be effective in this respect, for example, mandating or increasing a minimum legal age for drinking/purchase of alcohol (43). Young people tend to be more price sensitive, and thus with a price increase they are likely to buy and drink less, and vice versa; with a price decrease they are likely to buy and drink more (44).

The sparse research literature in this area and the noted limitations of the present and previous studies suggest a need for further studies of the DSH–HED association and its underlying mechanisms.

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Table 1. Sample characteristics and frequency distributions of DSH and HED at T1 and T2

Sample characteristics	T1	T2
Response rate	92 %	70 %
Age of respondents (mean, range)	16.5 (14-21 yrs)	21.5 (19-26 yrs)
Gender, per cent (n) Girls Boys	 56.5 (1633)	
Proportion students, percent (n)	100 (2897)	57.6 (1669)
Parental background, per cent (n) Both born in Norway One or both born in another European country Both born in a non-western country Other	 93.6 (2469) 4.5 (118) 1.3 (33) 0.6 (17)	 As for T1
Distribution DSH, per cent (n) No Yes	 96.8 (2804) 3.2 (93)	 98.4 (2852) 1.6 (45)
Distribution HED frequency, per cent (n) 0 1 2-5 6-10 11-50 51 +	 44.9 (1280) 9.4 (267) 18.6 (531) 10.1 (289) 14.5 (414) 2.5 (71)	 13.7 (389) 6.2 (177) 21.4 (607) 16.3 (463) 34.3 (973) 8.0 (227)
UCLA loneliness score (mean, range	1.83 (1.0 – 4.0)	1.81 (1.0 – 3.8)
Depressive symptoms score (mean, range)	1.75 (1.0 - 4.0)	1.72 (1.0 – 4.0)
Parents intoxicated Never A few times or more often	 47.3 (1310) 52.7 (1459)	 28.6 (824) 71.4 (2054)

Table 2. Changes in DSH and HED from T1 to T2. Percentage and number of respondents.

Variable	Change	%	N
DSH	Decrease	2.9	82
	Stable	95.8	2674
	Increase	1.3	36
HED	Decrease	14.1	395
	Stable	25.4	708
	Increase	60.5	1689

Table 3. Estimated associations between changes in HED and DSH in ordinal regression models. Robust clustered standard errors.

	Model 1[a]				Model 2[b]				Model 3[c]			
HED	OR	95% CI	P-value	PAF	OR	95% CI	P-value	PAF	OR	95% CI	P-value	PAF
Decrease	0.64	0.34, 1.21	.170		0.65	0.35 , 1.19	.163		0.72	0.40, 1.32	.289	
Stable (Reference)	1.00				1.00				1.00			
Increase	1.75	1.18, 2.60	.005	30%	1.80	1.21 , 2.69	.004	32%	1.64	1.11, 2.42	.013	28%

[a] Model 1 included no control variables.

[b] Model 2 included difference score on poor social network as control variable.

[c] Model 3 included difference scores on poor social network and depressive symptoms as control variables.

References

1. Evans E, Hawton K, Rodham K, Deeks J. The prevalence of suicidal phenomena in adolescents: a systematic review of population-based studies. Suicide & Life-Threatening Behavior. 2005;35(3):239-50.
2. Muehlenkamp J, Claes L, Havertape L, Plener P. International prevalence of adolescent non-suicidal self-injury and deliberate self-harm. Child Adolesc Psychiatry Ment Health. 2012;6:1-9.
3. Evans E, Hawton K, Rodham K. Factors associated with suicidal phenomena in adolescents: a systematic review of population-based studies. Clinical Psychology Review. 2004;24:957-79.
4. Madge N, Hewitt A, Hawton K, Wilde EJd, Corcoran P, Fekete S, et al. Deliberate self-harm within an international community sample of young people: comparative findings from the Child & Adolescent Self-harm in Europe (CASE) Study. Journal of Child Psychology and Psychiatry. 2008;49:667-77.
5. Goldston DB. Conceptual issues in understanding the relationship between suicidal behavior and substance use during adolescence. Drug and Alcohol Dependence. 2004;76S:S79-S91.
6. Evans E, Hawton K, Rodham K. Factors associated with suicidal phenomena in adolescents: A systematic review of population-based studies. Clinical Psychology Review. 2004;24(8):957-79.
7. Galaif ER, Sussman S, Newcomb MD, Locke TF. Suicidality, dpression, and alcohol use among adolescents: A review of empirical findings. International Journal of Adolescent Medicine and Health. 2007;19:27-35.
8. Hufford MR. Alcohol and suicidal behavior. Clinical Psychology Review. 2001;21:797-811.
9. Brady J. The association between alcohol misuse and suicidal behaviour. Alcohol & Alcoholism. 2006;41:473-8.
10. Esposito-Smythers C, Spirito A. Adolescent substance use and suicidal behavior: A review with implications for treatment research. Alcoholism: Clinical and Experimental Research. 2004;28:77s-88s.
11. Rossow I. Substance use and suicidal behaviour. In: Hawton K, editor. Prevention and treatment of suicidal behaviour From science to practice. Oxford: Oxford University Press; 2005. p. 261-77.
12. Cherpitel C, Borges G, Wilcox HC. Acute alcohol use and suicidal behavior: a review of the literature. Alcoholism: Clinical and Experimental Research. 2004;28:18S-28S.
13. Rossow I, Grøholt B, Wichstrøm L. Intoxicants and suicidal behaviour among adolescents: changes in levels and associations from 1992 to 2002. Addiction. 2005;100(1):79-88.
14. Garrison CZ, McKeown RE, Valois RF, Vincent ML. Aggression, substance use, and suicidal behaviors in high school students. American Journal of Public Health. 1993;83:179-84.
15. Rossow I, Ystgaard M, Hawton K, Madge N, van Heeringen K, de Wilde EJ, et al. Cross-national comparisons of the association between alcohol consumption and deliberate self-harm in adolescents. Suicide & Life-Threatening Behavior. 2007;37(6):605-15.
16. Yamasaki A, Chinami M, Suzuki M, Kaneko Y, Fujita D, Shirakawa T. Tobacco and alcohol tax relationships with suicide in Switzerland. Psychological Reports. 2005;97(1):213-6.
17. Hawton K, Saunders KEA, O'Connor RC. Self-harm and suicide in adolescents. The Lancet. 2012;379:2373-82.
18. Kuntsche E, Rehm J, Gmel G. Characteristics of binge drinkers in Europe. Social Science & Medicine. 2004;59:113-27.
19. Allison PD. Change scores as dependent variables in regression analysis. Sociological Methodology. 1990;20:93-114.
20. Halaby CN. Panel models in sociological research: theory into practice. Annual Review of Sociology. 2004;30:507-44.
21. Rossow I, Norström T. The use of epidemiology in alcohol research. Addiction. 2013;108:20-5.
22. Conner KR, Gunzler D, Tang W, Tu XM, Maisto SA. Test of a clinical model of drinking and suicidal risk. Alcoholism: Clinical and Experimental Research. 2011;35(1):60-8.

23. Fergusson DM, Horwood LJ, Swain-Campbell N. Cannabis use and psychosocial adjustment in adolescence and young adulthood. Addiction. 2002;97:1123-35.
24. Krysinska K, Martin D. The struggle to prevent and evaluate: application of population attributable risk and preventive praction to suicide prevention research. Suicide & Life-Threatening Behavior. 2009;39:548-57.
25. Dube SR, Anda RF, Felitti VJ, Chapman DP, Williamson DF, Giles WH. Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span. JAMA. 2001;286(24):3089-96.
26. Cavanagh JTO, Carson AJ, Sharpe M, Lawrie SM. Psychological autopsy studies of suicide: a systematic review. Psychological Medicine. 2003;33:395-405.
27. Taylor B, Shield K, Rehm J. Combining best evidence: A novel method to calculate the alcohol-attributable fraction and its variance for injury mortality. BMC Public Health. 2011;11:265.
28. Rossow I, Moan IS. Parental intoxication and adolescent suicidal behavior. Archives of Suicide Research. 2012;16:73-84.
29. Zucker RA. Anticipating problem alcohol use developmentally from childhood into middle adulthood: What have we learned? Addiction. 2008;103(Supplement 1):100-8.
30. Strand NP, von Soest T. Young in Norway - Longitudinal. Documentation of design, variables and scales. Oslo: Norwegian Social Research; 2007. 242 p.
31. Tørmoen AJ, Rossow I, Larsson B, Mehlum L. Nonsuicidal self-harm and suicide attempts in adolescents: differences in kind or in degree? Social Psychiatry and Psychiatric Epidemiology. 2013;48:1447-55.
32. Hawton K, Rodham K, Evans E, Weatherall R. Deliberate self harm in adolescents: self report survey in schools in England. BMJ. 2002;325(7374):1207-11.
33. Russell D, Peplau LA, Cutrona CE. The revised UCLA Loneliness Scale: concurrent and discriminant validity evidence. Journal of personality and social psychology. 1980;39:472-80.
34. Kandel DB, Davies M. Epidemology of depressed mood in adolescents: An empirical study. Archives of General Psychiatry. 1982;39:1205-12.
35. McCullagh P. Regression models for ordinal data. Series B (Methodological). Journal of the Royal Statistical Society. 1980;42:109-42.
36. Williams RL. A note on robust variance estimation for cluster-correlated data. Biometrics. 2000;56:645-6.
37. Zhang J, Yu KF. What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. JAMA: The Journal of the American Medical Association. 1998;280:1690-1.
38. Fliege H, Lee J-R, Grimm A, Klapp BF. Risk factors and correlates of deliberate self-harm behavior: A systematic review. Journal of Psychosomatic Research. 2009;66(6):477-93.
39. Wells JE, Horwood LJ, Fergusson DM. Drinking patterns in mid-adolescence and psychosocial outcomes in late adolescence and early adulthood. Addiction. 2004;99(12):1529-41.
40. Casswell S, Pledger M, Pratap S. Trajectories of drinking from 18 to 26 years: identification and prediction. Addiction. 2002;97:1427-37.
41. Warner LA, White HR. Longitudinal Effects of Age at Onset and First Drinking Situations on Problem Drinking#. Substance Use & Misuse. 2003;38(14):1983-2016.
42. Cherpitel CJ, Borges GLG, Wilcox HC. Acute alcohol use and suicidal behavior: a review of the literature. Alcoholism: Clinical and Experimental Research. 2004;28(s1):18S-28S.
43. Wagenaar AC, Toomey TL. Effects of minimum drinking age laws: Review and analyses of the literature from 1960 to 2000. Journal of Studies on Alcohol. 2002:206-25.
44. Elder RW, Lawrence B, Ferguson A, Naimi TS, Brewer RD, Chattopadhyay SK, et al. The effectiveness of tax policy interventions for reducing excessive alcohol consumption and related harms. American Journal of Preventive Medicine. 2010;38:217-29.