Associations between emotional distress and heavy drinking among young people: A longitudinal study

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Introduction and Aims. This study adds to the meagre body of longitudinal research on the link between emotional distress and alcohol use among young people. We address these research questions: Are symptoms of anxiety and depressed mood likely to be causally related to heavy episodic drinking (HED), and does the association change as individuals move from adolescence to early adulthood?

Design and Methods. Data stemmed from a national sample of young people in Norway that was assessed in 1992 (T1; mean age= 14.9 years), 1994 (T2), 1999 (T3), and 2005 (T4) (response rate: 60%, n=2 171). We applied fixed-effects modelling, implying that intra-individual changes in the frequency of HED were regressed on intra-individual changes in emotional distress. Hence, confounding due to stable underlying influences was eliminated. Self-perceived loneliness was included as a time-varying covariate.

Results. Emotional distress was unrelated to HED in adolescence (T1 to T2). In the transition from adolescence to early adulthood (T2 to T3), changes in depressiveness were positively and independently associated with changes HED, while changes symptoms of anxiety were not. A similar pattern emerged in early adulthood (T3 to T4).

Discussion and Conclusions. The potential causal relationship between emotional distress and heavy drinking did not manifest itself in adolescence, but increased symptoms of depressiveness were related to more frequent HED in subsequent periods of life. Hence, this study provides conditional support to the notion that emotional distress and HED may be causally related, and indicates that the association among young people may be specific to depressiveness.

Key words: Anxiety, depressed mood, emotional distress, heavy drinking, adolescence, early adulthood.
Introduction
Depression has generally been found to be associated with heavy drinking and alcohol use disorders [1-4]. There is also evidence of a link between anxiety and extensive use of alcohol [5-8], but this association has been less extensively scrutinized and the findings seem less clear. Thus, some studies of young people report null-findings [9] or a link between anxiety and heavy drinking that is attributable to concurrent symptoms of depression and/or other confounding factors [10-12].

The bulk of previous studies focus on severe forms of internalizing problems and harmful drinking, and have typically relied on diagnoses or other dichotomous measures. Obviously, such research may be of clinical significance, yet the findings do not necessarily apply to mild forms of emotional distress and non-pathological drinking [13]. To address subclinical conditions is important in its own right, not least because they may contribute to the total burden of harm and social cost at least as much as the clinical ones – simply by being far more prevalent.

Furthermore, studies of adult samples predominate in the field, and the association between emotional distress and heavy drinking may be different among young people. The onset of drinking typically occurs in adolescence, and the consumption of alcohol and related harm escalate during the late teens and reaches a peak in early adulthood [14, 15]. Problems related to mood and emotions also tend to increase in this period of life [16]. Moreover, it has been hypothesized that risk factors related to mental health may become increasingly important in influencing drinking behaviour as individuals approach adulthood, reflecting the challenges of leaving a protected environment, and entering an independent adult role [17].

Indeed, a recent study showed that the association between depression and alcohol use disorders was stronger in early adulthood than in adolescence [18]. The present paper pursues this issue further by analysing four-wave panel data on less severe forms of emotional distress and heavy drinking from a general population study of young people.

Causality and directionality
Associations between internalizing problems and heavy drinking may in part reflect causality, or they may arise solely because of shared underlying influences. A range of factors may indeed confound these associations, including genetic susceptibility, disadvantaged upbringing conditions and poor social integration [1, 5]. A number of such likely confounders have been accounted for in previous research, but one and the same study cannot possibly control for all conceivable sources of confounding.

Fixed-effects modelling may substantially reduce the problem of unmeasured common influences because it eliminates bias due to temporally stable covariates [19, 20]. Despite its merits, Fergusson et al. [21] seem to be the only ones that have applied this method in relation to the topic at issue. They focused on symptoms of major depression and alcohol use disorder, and concluded that such problems seem to be causally related. However, the issue of causality is still far from settled, and the directionality of the potential causal effect is also not clear.
The self-medication hypothesis has been invoked as a possible causal explanation for the high comorbidity of mental illness and pathological drinking, and for associations between subclinical forms of such problems [22-24]. Indeed, there is ample evidence that negative affectivity is linked to self-medication motives for using alcohol [25-27], which in turn are predictive of heavy and harmful drinking [28-31]. Moreover, a study of undergraduate women recently showed that minor symptoms of depressed mood predicted heavy episodic drinking (HED) over one week intervals, while there was no effect of HED on subsequent depressiveness [32]. Another study found that adolescents whose first-time drinking episode was motivated by a wish to relieve sadness had an elevated risk of involvement in heavy drinking [33]. Two recent studies of adults provide additional evidence that subclinical symptoms of anxiety and/or depression may impact on alcohol use but not vice versa [34, 35]. Hence, the present study relies on the assumption that the main effect goes from emotional distress to heavier drinking rather than the other way around.

The present study

While most previous studies on the topic at issue have focused on either depression or anxiety, we analyse data on both kinds of emotional distress and examine whether they are related independently to heavy drinking. In addition, we apply a compound index for emotional distress. Further, by using fixed-effects modelling, we eliminate confounding due to stable covariates. This method does not remedy bias due to time-varying shared influences, and we include loneliness as such a potential confounder. Feeling lonely is indicative of a lack of support and emotional closeness to others, and such feelings are related to both anxiety and depression [36-38]. Associations with heavy drinking have also been found, yet some studies indicate that loneliness is inversely related to alcohol use [39, 40], while others have found a positive correlation between the two [41, 42].

We addresses the following research questions: Are symptoms of anxiety and depressed mood likely to be causally related to heavy episodic drinking among young people, and does the association change as individuals move from adolescence to early adulthood?

Methods

Data stemmed from the Young in Norway Longitudinal Study, which has been described in detail elsewhere [43, 44]. A cohort of young people was assessed in 1992 (T1), 1994 (T2), 1999 (T3) and 2005 (T4). The T1 survey comprised students attending junior high school (grades 8-10) and senior high school (grades 11-13), and the sample was selected to yield a national representative cross-section of this student population (response rate: 97%). At T2, students who were still in their original school filled in questionnaires in the classroom – as they did at T1. Those who had graduated from either junior or senior high school since the initial assessment received postal questionnaires. Only the former group achieved a high response rate (92%). Therefore, all subsequent follow-ups were restricted to students who attended the same school at T1 and T2 (i.e. the 8th and 11th graders at T1). The majority (91%)
of these students consented at being traced for future participation in the study, of which 84% responded at T3 and 82% responded at T4. The cumulative response rate was 60%. The study was conducted in accordance with the National Guidelines for Research Ethics in the Social Sciences and approved by the Norwegian Social Science Data Services.

Our analyses were confined to respondents who participated in all four waves, and who were 13-17 years at T1. From this subsample, we excluded 3% due to high rates of missing data. The final study sample comprised 2171 respondents, of whom 57% were females.

**Key measures**

Heavy episodic drinking (HED) was captured by asking about the frequency of consuming alcohol to the point of feeling clearly intoxicated in the past 12 months. There were six 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).

Emotional distress were assessed by asking to what extent the respondents had been bothered by various symptoms in the past week, using this four-point scale: not at all (1), a little bit bothered (2), quite bothered (3), and extremely bothered (4). The following three items from the Hopkins Symptoms Check List [45] were added up and averaged to construct an index for symptoms of anxiety: (1) Suddenly scared for no reason; (2) Constantly scared or worried; and (3) Nervousness or shakiness inside. The internal consistency showed little variation across the four waves (Cronbach’s alpha 0.75 - 0.78), while the stability coefficients (Pearson’s r) ranged from 0.37 (T2 to T3) to 0.44 (T1 to T2).

Depressed mood was also measured as an additive index, using six items from The Depressive Mood Inventory [46]: (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 (Cronbach’s alpha: 0.82-0.85). The lowest stability coefficient was 0.42 (T2 to T3) while the highest was 0.55 (T1 to T2).

The cross-sectional correlations between the indices for anxiety and depressed mood ranged from 0.55 (T2 to T3) to 0.65 (T4), and we also constructed a compound index for emotional distress based on all the nine items mentioned above (Cronbach’s alpha 0.82-0.87). The stability coefficients for this measure varied between 0.42 (T2 to T3) and 0.55 (T3 to T4).

**Time-varying covariate**

We used four items from UCLA Loneliness Scale [47] to construct an index for self-perceived loneliness. The respondents considered statements such as “No one really knows me very well” and “People are around me but not with me”, and reported on a scale ranging from 1 (never) to 4 (often) how often they felt this way (Cronbach’s Alpha: 0.72-0.78).
Statistical analyses

First, we performed descriptive statistics of the key variables at all four assessments. Changes in proportions between waves were tested using z-tests, while changes in means were examined using t-tests for dependent samples. Subsequently, we estimated a set of fixed-effects models [19, 20]. More precisely, we calculated change scores for both HED and each of the three measures on emotional distress by subtracting T1 from T2, T2 from T3, and T3 from T4. Next, the latter change scores were included in multiple OLS regressions to estimate associations with changes in the frequency of HED. As illustrated below, this implies that the time-invariant confounders are cancelled out:

\[
\begin{align*}
    HED_{1i} &= \beta_1 E_D_{1i} + \beta_2 C_i + \epsilon_{1i} \\
    HED_{2i} &= \beta_1 E_D_{2i} + \beta_2 C_i + \epsilon_{2i}
\end{align*}
\]

In model (1) \(HED_{1i}\) and \(E_D_{1i}\) refer to heavy episodic drinking and emotional distress, respectively, for individual \(i\) at T1. \(C_i\) denotes other causes of HED that are stable across time. If \(ED\) and \(C_i\) correlate, the estimate of \(\beta_1\) will be biased. Model (2) is equivalent, but refers to T2. If we subtract (1) from (2), \(C_i\) is eliminated, and thereby that particular source of bias.

All estimates based on fixed-effects modelling were adjusted for changes in self-perceived loneliness. To assess whether the link between changes in anxiety and changes in the frequency of HED was modified when controlling for changes in depressiveness, we estimated one model excluding (model 1), and another model including (model 2) the latter covariate. We used the same approach when assessing the corresponding association between depressed mood and HED, i.e. symptoms of anxiety were added to the equation in model 2.

Finally, we examined how the change scores for the compound measure on emotional distress were associated with changes in HED.

The above-mentioned analyses were conducted for all the respondents, and for males and females separately. When statistically significant gender differences were detected, this is reported. Moreover, because a four-year age range is substantial in adolescence, we performed age specific analyses for this developmental period. That is, in addition to analyses of the whole sample, we conducted separate analyses of the youngest (13-15 years at T1) and the oldest respondents (16-17 years at T1), and compared the estimates using T-test.

Due to the large sample size, we chose the 1% level for statistical significance. As the sampling was clustered by school, we used robust clustered standard errors with school as cluster variable [48]. The statistical analyses were conducted using Stata (version 12) and SPSS (version 21).

Results

The proportion reporting HED as well as the mean frequency of HED increased from T1 to T2 and reached a peak at T3 (Table 1). The level of anxiety did not vary across the three first
waves, but was clearly lower at the final assessment. Symptoms of depressed mood also declined from T3 to T4, yet the highest level was observed at T2. The mean scores for the compound measure of emotional distress decreased slightly from T2 to T3, while the decrease from T3 to T4 was quite substantial.

Table 1. *Descriptive statistics on key variables at the four waves of data collection.*

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (SD=1.5)</td>
<td>14.9</td>
<td>16.4</td>
<td>21.8</td>
<td>28.3</td>
</tr>
<tr>
<td>% Reporting heavy episodic drinking ≥ 1 time</td>
<td>33.1</td>
<td>53.1**</td>
<td>87.1**</td>
<td>83.3**</td>
</tr>
<tr>
<td>Frequency of heavy episodic drinking, M (SD)</td>
<td>3.90 (9.78)</td>
<td>6.88** (12.24)</td>
<td>16.97** (16.66)</td>
<td>13.93** (15.62)</td>
</tr>
<tr>
<td>Symptoms of anxiety¹, M (SD)</td>
<td>1.34 (0.47)</td>
<td>1.34 (0.47)</td>
<td>1.33 (0.49)</td>
<td>1.28** (0.45)</td>
</tr>
<tr>
<td>Symptoms of depressed mood¹, M (SD)</td>
<td>1.71 (0.55)</td>
<td>1.75** (0.57)</td>
<td>1.72* (0.59)</td>
<td>1.69** (0.59)</td>
</tr>
<tr>
<td>Symptoms of either kind of emotional distress¹, M (SD)</td>
<td>1.53 (0.45)</td>
<td>1.55 (0.47)</td>
<td>1.52* (0.48)</td>
<td>1.42** (0.45)</td>
</tr>
</tbody>
</table>

*p<0.01;  **p<0.001 = significantly different from the proportion or the mean at the previous wave of data collection. ¹ Scale: 1 – 4

Table 2 shows the outcomes of the fixed-effects models. As can be seen, there were no associations between any measure of emotional distress and HED in adolescence (T1 to T2). We also conducted age specific analyses, and found that the estimates for youngest respondents (13-15 years at T1) were all far from statistically significant, as were the estimates for the oldest (16-17 years at T1) (estimates not shown).

In the transition from adolescence to early adulthood (T2 to T3), there was initially a weak positive impact of changes in anxiety on changes in HED (model 1). However, it weakened and was far from statistically significant when concurrent changes in depressed mood were taken into account (model 2). In contrast, there was a highly significant impact of changes in depressed mood, which barely attenuated when we accounted for changes in anxiety. The impact of the composite measure on emotional distress was also statistically significant in this period.

The general pattern of findings in early adulthood (T3 to T4) echoed that of the preceding period. However, for the first time, a statistically significant impact of anxiety was
observed (model 1), yet it attenuated and was above the 1% level of statistical significance when we added the measure for depressed mood to the equation (model 2).

**Table 2. Changes in the frequency of heavy episodic drinking regressed on changes in anxiety (ΔAnx), depressed mood (ΔDep) and in the overall level of emotional distress (ΔEmo) in adolescence (T1 to T2), in the transition from adolescence to early adulthood (T2 to T3), and in early adulthood (T3 to T4). Fixed-effects models with robust clustered standard errors.**

<table>
<thead>
<tr>
<th>Model¹</th>
<th>Adolescence</th>
<th>Adolescence / early adulthood</th>
<th>Early adulthood</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Est</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>ΔAnx</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-0.82</td>
<td>0.68</td>
<td>0.229</td>
</tr>
<tr>
<td>2</td>
<td>-0.75</td>
<td>0.67</td>
<td>0.265</td>
</tr>
<tr>
<td>ΔDep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-0.44</td>
<td>0.55</td>
<td>0.420</td>
</tr>
<tr>
<td>2</td>
<td>-0.19</td>
<td>0.52</td>
<td>0.714</td>
</tr>
<tr>
<td>ΔEmo</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-0.65</td>
<td>0.54</td>
<td>0.233</td>
</tr>
</tbody>
</table>

¹ Model 1: Controlling for changes in loneliness. Model 2: Controlling for changes in loneliness and depressed mood (in analyses of anxiety) and for changes in anxiety (in analyses of depressed mood).

The results in table 2 imply that an increase by one unit on the compound index for emotional distress in early adulthood, e.g. from “a little bit bothered” to “quite bothered” (representing 2.1 SDs) would give an average increase in the number of heavy drinking episodes of 2.4 times a year. The corresponding number for the transition from adolescence to early adulthood is about three times a year.

Finally, we conducted fixed-effects modelling for males and females separately, and compared the estimates. We detected no differences below the 1% level for statistical significance, but the impact of depressed mood on HED in early adulthood was significant for females (Est=2.54, SE=0.86, p=0.005) but not for males (Est=1.87, SE=1.30, p=0.157) (t=2.46, p<0.05).
Discussion

In this study, we assessed intra-individual associations between different forms of emotional distress and HED using fixed-effects modelling. Such associations seem more relevant in relation to the topic at issue than are inter-individual associations. Thus, some individuals may increase their drinking in periods of elevated distress, yet this does not necessarily imply that they drink more and have more symptoms of internalizing problems than other people when such periods occur. Because fixed-effects modelling eliminates bias due to time-invariant confounders, our study also sheds light on the issue of causality.

Neither anxiety nor depressed mood were related to HED in adolescence, while the results for the subsequent periods that we assessed were mixed. Changes in anxiety had a positive and highly significant impact on changes in HED, but only in early adulthood, and only when we did not control for concurrent changes in depressiveness. In contrast, increased levels of depressed mood was independently related to more frequent HED – both in the transition from adolescence to early adulthood, and in the early adult period. The composite index on emotional distress also had a statistically significant impact on HED in these two periods of life. The results thus lend conditional support to the notion that emotional distress and heavy drinking may be causally related, but the magnitude of the effects was moderate.

The present study also indicates that the association between internalizing symptoms and heavy drinking among young people may be specific to depressed mood. However, the internal consistency of our measure on anxiety was somewhat lower than that of depressed mood, and the results may have been affected accordingly. On the other hand, the findings seem to agree with the extant body of research on the issue. Thus, while a link between depression and heavier drinking has been found repeatedly [1-4], the association between anxiety and alcohol use seems less clear. It is true that both forms of emotional distress rarely have been assessed in one and the same study, but Skogen et al. [49] recently reported that depressive symptoms were related to frequent intoxication among late teen youth, while symptoms of anxiety were not. Similar results emerged in another recent study of young people [10].

Our findings are also congruent with the assumption that mental health risk factors are likely to become increasingly important in influencing drinking behaviour as individuals move from adolescence into adulthood [17]. Furthermore, the age gradient in the associations that we observed corroborates the results reported by Briere et al. [18] in their study of comorbidity between depression and excessive drinking from adolescence to adulthood.

As regards the absence of associations between emotional distress and HED in adolescence (T1 to T2), one should keep in mind that a solid majority (66%) of the respondents reported no HED at the first wave of data collection. The same was true for 47% at the second survey 1.5 years later. Hence, to a substantial extent, the aggregate level changes in drinking from T1 to T2 reflected age-typical transitions from abstinence to drinking, or from early, experimental drinking to more regular HED.
Irrespective of their level of emotional distress, most young people start to drink sometime during adolescence [50]. To initiate drinking, and to gradually increase one’s alcohol consumption, may be considered normative behaviour in this developmental period. Moreover, adolescents most often drink for social and enjoyment reasons [28, 51], and one may assume that drinking to relieve emotional distress is relatively more prevalent among older and more experienced drinkers. Indeed, Cooper et al.’s [52] longitudinal study showed that self-medication motives for drinking were reported gradually more often during the transition from adolescence into early adulthood.

The research findings cited above all seem relevant in the context of the present study, but one should keep in mind that the directionality of the associations at issue is still open to question. Based on recent studies of subclinical conditions, including two studies of young people, we assumed that emotional distress increases the risk of heavier drinking rather than the other way around. However, this assumption may not necessarily be correct.

In their review of the literature, Boden and Fergusson [1] concluded that alcohol use disorders increase the risk of major depression rather than vice versa. However, this conclusion relied mainly on one single study (i.e. [21]), and has therefore been called into question [53]. Based on more recent evidence, Bell et al. [35] hypothesized that internalizing problems may increase the risk of heavier drinking until symptoms of alcohol dependence begin to emerge, whereby the causal direction of the association is likely to reverse.

Indeed, there is little doubt that excessive drinking may deteriorate individuals’ mental health, among other things because it can bring about social, financial and relationship problems [1]. Our finding that emotional distress and HED were unrelated in adolescence may reflect that teenagers rarely consume so much alcohol that their mental health or well-being is affected. As they grow older and increase their drinking, the risk of such adverse outcomes is also likely to increase.

**Methodological considerations**

There are limitations of our study that warrant attention. We assessed symptoms of emotional distress during the past week, while there was a one-year time frame for the frequency of HED. However, the stability in the level of both anxiety and depressiveness was far from negligible across the developmental periods that we assessed, indicating that the differences in the temporal point of reference may not pose major problems.

Another weakness is that the frequency scale for HED was fairly crude, and the ensuing measurement errors imply that the estimated associations are likely to be deflated. Moreover, self-perceived loneliness was the only potential confounder that we accounted for, yet there are probably many other time-dynamic factors that impact on both emotional distress and HED. For instance, there is solid evidence that negative life events increase the risk of depression [54], and an association with heavier drinking has also been found [55, 56]. Social support is another time-variant factor that is likely to confound the association between depression and alcohol use [57].
Conclusions

This longitudinal study of young people lends conditional support to the notion that emotional distress and HED are likely to be causally related. There were no associations between the two in adolescence, but increased levels of depressiveness correlated significantly with more frequent HED in the transition from adolescence to early adulthood, and in the early adult period. The corresponding associations between symptoms of anxiety and HED were either non-existent or less clear.

Acknowledgements

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