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Alcohol misuse among English youth, are harms attributable to alcohol or to underlying disinhibitory characteristics?

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Word count 2016.

Are harms attributable to alcohol or underlying disinhibition?
Key words: alcohol-related disorders, alcohol drinking, conduct problems, hyperactivity, adolescent.
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Despite recent declines, alcohol consumption by UK young people remains high by international standards. The rate of last 30 days self-report alcohol intoxication among English 15-year-olds was 22% (NHS Digital 2019). According to the European School Survey Project on Alcohol and other Drugs (ESPAD 2015, 2019), the corresponding figure for European youth was 13%, and, according to the Monitoring the Future study, it was below 10% in the US (Miech et al 2019).

International comparisons across Europe suggest that high levels of intoxication used to characterise Northern, and to some degree Eastern European youth populations, versus lower rates in Mediterranean drinking cultures (Andersson et al 2007). More recent data suggest residually low levels of intoxication in Portugal, Greece and Macedonia but France, Italy and Spain have become more ‘Northern’ in their patterns of youth drinking. Remarkably, according to ESPAD data, from being among the most likely to experience intoxication, Swedish young people have become relatively abstinent, a decline also seen in previously high use English speaking countries including the USA.

The deep origins of these differences and trends in alcohol use are likely to be complex. Evidence includes traditional drinking culture, relative educational attainment, overall wellbeing of populations and availability of treatment for substance use and related comorbidities (Beard et al 2019, Strang 2019, Case et al 2015, UNICEF 2013, Marcotte 2011). The literature also refers to the apparently increasing importance of parental
‘monitoring’ in Iceland (Amarsson et al 2018) and the absence of a social gradient in a Swedish survey (Thor et al 2019). More recently, Borodovsky et al (2021) has argued that declines in substance use including alcohol among American youth may be attributable to the displacement of in-person socialising by immersive online activities. The relative importance of these proposed influences on drinking behaviour remains unclear and may vary across cultures and periods.

Whatever the correlates, heavy drinking places does appear to place youth at risk of other substance use, impaired school and vocational attainment, accidents, assault and suicide, all potentially damaging life opportunities, wellbeing and longevity (Hall et al 2016, Vigo et al 2016). In the UK, concern about the effect of alcohol on youth well-being and health has led to Chief Medical Officer advice not to drink below 15 years of age (Donaldson 2009).

However, Hall et al (2016) have also argued that ‘the research (linking alcohol and harm) …has… often used… designs poorly suited to assessing causality’. Indeed, longitudinal studies show that many of the reported alcohol behavioral sequelae are also characteristic of the externalizing or conduct problems said to predispose to and precede adolescent alcohol misuse (Lynskey et al 1995, Mannuzza et al 2008, Moffitt et al 2002).

Also, family and genetic studies probing causality suggest that a range of adolescent disruptive behaviours including alcohol misuse have a common developmental origin, termed ‘psychological dysregulation’ (Clark 2004), ‘behavioral disinhibition’ (Hicks et al 2004) or an ‘externalizing-like factor’ (Borodovsky 2019). For instance, using DSM IV (APA 1994) diagnoses of disruptive behavior disorders as markers of disinhibition, Hicks et al (2004) argue that this ‘… broad behavioral system … …underlies all the disorders in the
externalizing spectrum’. This spectrum is likely to have environmental and genetic roots and is said to include attention deficit hyperactivity, conduct and antisocial personality as well as drug and alcohol use disorders (Elkins et al 2018). It is likely to be disproportionately evident in a minority of youth, potentially driving wider trends (Lynskey et al 1995).

Hence, it is possible that the adolescent behavioral harms said to be associated with alcohol might be better attributed to underlying psychological and emotional dysregulation or disinhibition rather than alcohol, itself just one other manifestation of an underlying common diathesis. If true, public health efforts should include consideration of measures targeted at behavioral disinhibition and its early manifestations, even before alcohol initiation.

In order to assess this question, we modelled the relationship between measures of dysregulation and disinhibition, alcohol consumption and subsequent alcohol-related harms in a sample of Emergency Department (ED) attenders. These young people had been opportunistically screened for alcohol use and identified as a high-risk group based on scoring 3 or more on the AUDIT-C (Coulton et al 2019). Those who consented were allocated to an intervention group as part of a randomized controlled trial (Deluca et al 2020). They were followed up at 6 and 13 months after randomization. Our hypothesis was that alcohol-related harms at 12 months would be associated with a measure of behavioral disinhibition rather than quantity of alcohol consumed.

Method
Data were obtained from a randomised controlled trial to evaluate the effectiveness and cost-effectiveness of brief alcohol intervention strategies compared with screening alone in 14-17-
year-olds, scoring 3 or more on AUDIT-C (Coulton et al 2019), attending 10 emergency departments (EDs) in England (Deluca et al 2020).

Alcohol measures
Alcohol consumption was derived from the AUDIT-C extended version and presented as mean units consumed per week in the past 6 months. The AUDIT-C has been validated by comparison with other established measures of alcohol consumption. The AUDIT-C extended version adds additional categories to allow for calculation of alcohol consumed and is scored in a similar manner (Coulton et al 2019).

Behavioural measurement
Behavioral disinhibition was measured by the conduct and hyperactivity items on the self-report Strengths and Difficulties Questionnaire, a widely used clinical and research measure of child and youth psychopathology with good sensitivity and specificity for child psychiatric disorder (Goodman et al 2000, Goodman 2001); scores above the 90th centile predict independently diagnosed psychiatric disorder with an odds ratio for the self-report of 6.2 (Goodman 2001). Hyperactivity is genetically linked to conduct problems and is likely to be an early manifestation of vulnerability to the broader syndrome of conduct problems (Thapar et al 2001, Taylor et al 1991). Externalizing behaviours including hyperactivity and conduct problems are regarded as indicators of behavioural disinhibition (King et al 2004).

Harms
Alcohol-related harms were assessed by question 22 from the ESPAD (2015) survey, “Because of your own alcohol use, how often during the last 12 months have you experienced the following?” Items included fighting, accident or injury, parent, peer, school,
and police problems, hospitalisation, unsafe sexual activity and regretted sex. Outcomes are frequencies over the last 12 months. ESPAD attended to data quality by anonymity and by eliminating ‘all cases with responses to less than half of the core items… (or) patterns involving repetitive marking of extreme values’. The outcome was operationalized by summing the frequency of each category of alcohol-related harm in the reference period.

Analysis

In order to assess the hypothesis we conducted a mediation analysis. Our primary outcome was alcohol-related harms 12-months after screening in ED. We modelled the relationship between domains derived from the SDQ at baseline and alcohol-related harms at month 12, exploring for the mediating influence of alcohol consumed at six months (Figure 1). We included a number of covariates that are also considered to influence alcohol-related harms; age, gender and baseline alcohol consumption and, as the data were derived from a randomised controlled trial, we included allocated group as a covariate. In addition, we employed a multi-level model to adjust for the fact that young people were nested within EDs.

Mediation analysis was conducted using the statistical product of coefficients approach (STATA IC v15.1). The Stata command sgmediaton was employed in the analysis and direct and indirect effects derived using bootstrapped 95% confidence intervals. The model fit was assessed by exploring visual plots of the residuals.
Ethical approval

The study was conducted in accordance with ethical approval from the National Health Service Multi-Centre Research Ethics Committee (ref: 12/L0/0799) and was registered in an appropriate trial registry (ref: ISRCTN 45300218).

Findings

Table 1. shows demographic, SDQ and ESPAD alcohol harm data. The published British SDQ conduct problem mean score for adolescents is 1.5 (SD 1.7) and for hyperactivity, 3.2 (SD 2.6), compared to which the scores here are somewhat elevated. Greater alcohol consumption was associated with being white, smoking tobacco, increased frequency of intoxicification and frequency of alcohol-related harms.

The results of the mediation analysis are presented in Table 2. There is a significant direct effect of conduct score on alcohol-related harms at 12 months and also evidence of a similar relationship for hyperactivity scores. Notably, none of these effects are mediated by quantity of alcohol consumed at six months.

Discussion

We analysed a longitudinal data-set to explore whether underlying behavioural disinhibition could better explain harms attributed to alcohol use by young people. These show that behavioural disinhibition is a significant predictor of harms but this effect is not mediated through the quantity of alcohol consumed.

These data are consistent with long term follow-up studies of conduct problem children which show significant independent associations between early measures of behavioural

Findings are also consistent with research in US college students concerning alcohol and ‘regretted sex’. Data suggest a past month regretted sex rate as high as 25% with alcohol as a common association (Orchowski 2012). However, multivariate longitudinal studies controlling for underlying disinhibitory personality traits among male students suggest that disinhibition rather than alcohol itself is the key independent predictor of predatory sexual behaviour (Testa et al 2017).

The weaker effect of hyperactivity, on the face of it a quintessential marker of disinhibition, has been noted previously (Lynskey et al 1995). An early US follow-up study of hyperactive children (Gittelman et al 1985) reported that hyperactivity predicted adolescent substance misuse, including alcohol misuse. However, as in the New Zealand study (Lynskey et al 1995), this required the developmentally early emergence of conduct disorder, for which hyperactivity appeared to be a potent risk factor. In a larger follow up study of 208 hyperactive children to adulthood, the risk of substance, including alcohol, misuse was raised compared with non-hyperactive controls. However, in those with both hyperactivity and conduct disorder, the risk increased by a factor of 5 (Dalsgaard et al 2014).

Nevertheless, hyperactivity and conduct disorder are genetically closely related, the combined syndrome being more disruptive to typical development than either alone (Thapar et al 2001). In another genetically informed study, Hicks et al (2004) concluded that the effect of ‘hyperactivity-impulsivity’ was mediated ‘through transmission of a liability toward
behavioral disinhibition …’. Hyperactivity may be a developmentally early marker of liability but, alone, only one component of a substance and alcohol misuse predispositional syndrome.

Conduct problems are additionally linked with weakened associations with school and family and preferential associations with so-called ‘deviant peers’. These are likely to be older and facilitate introduction to substances including alcohol (Fergusson et al 1999). That deviant peers may have been displaced by on-line activities is a fascinating addition to this theory (Borodovsky et al 2019, 2021). In recent decades, buffered by immersive on-line activities, the importance of underlying disinhibition may also have declined, manifesting in reduced youth crime, risky sexual behaviour as well as substance use. Borodovsky et al (2021) also note recent indices of delayed maturation in US youth so that this buffering might not be entirely benign.

Disinhibition is not necessarily deterministic. Metanalyses and systematic reviews describe the protective influence against substance misuse in general of parental behaviours suggestive of authoritative parenting (Yap et al 2017, Meque et al 2019). Pathway analyses tend to confirm reduction of deviant peer associations via parental reduction of offspring externalising/disinhibited behaviour, and hence alcohol and other substance use (Kuo et al 2021). However, even where it exists, authoritative parenting can be severely challenged.

A novel study of Chinese adolescents using aldehyde dehydrogenase polymorphism to mark drinkers and non-drinkers (Chao et al 2017) detected a direct effect of alcohol on antisocial behaviour. However, over half of those in ‘emerging adulthood’ in China are non-drinkers (Lu et al 2019) suggesting a cultural significance of drinking that is distinct from the UK, US
or the West in general. Also, Chao et al (2017) did not include a measure of disinhibition so that confounding of the drinking variables with underlying attributes cannot be excluded.

Implications and Conclusions

Alcohol use by young people may be a marker of underlying issues that may have been disrupting connections to family and school, well prior to alcohol misuse. This developmental perspective tends to engage young people, families and others through often novel insights, more comprehensive multi-service interventions addressing the young person as a whole and offers public health opportunities for secondary prevention.

Limitations

The measure of disinhibition used here was the SDQ, designed for capturing a range of behavioural and emotional difficulties as well as indices of disinhibition. Also, the data are self-report, capturing an important perspective, but by omitting that of parents and teachers possibly understating disinhibition. The sample was of adolescents attending accident and emergency departments in England and so may not be representative of adolescents more generally.
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Figure 1: Mediation path diagram - conduct

Low risk

*Indirect pathway*

Quantity of alcohol consumed

0.078

Covariates

- Age
- Sex
- Intervention

High risk

*Indirect pathway*

Quantity of alcohol consumed

0.341

Covariates

- Age
- Sex
- Intervention

Direct effect 0.057 (-0.012; 0.126)

Alcohol-related harms at month 12

0.248

Direct effect 0.452 (0.256; 0.648)

Alcohol-related harms at month 12

0.081
Figure 2: Mediation path diagram - hyperactivity

**Low risk**

*Indirect pathway*

- Quantity of alcohol consumed: 0.0672 - 0.248

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Age</th>
<th>Sex</th>
<th>Intervention</th>
</tr>
</thead>
</table>

SDQ Hyperactivity Score at baseline

Direct effect: 0.027 (-0.015; 0.069)

**High risk**

*Indirect pathway*

- Quantity of alcohol consumed: 0.239 - 0.087

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Age</th>
<th>Sex</th>
<th>Intervention</th>
</tr>
</thead>
</table>

SDQ Hyperactivity Score at baseline

Direct effect: 0.099 (-0.022; 0.222)

**Covariates**

- Age
- Sex
- Intervention
Table 1. Association of different levels of alcohol, SDQ scores and harms

<table>
<thead>
<tr>
<th></th>
<th>n=756</th>
</tr>
</thead>
<tbody>
<tr>
<td>% male</td>
<td>49.8</td>
</tr>
<tr>
<td>% white</td>
<td>84.9</td>
</tr>
<tr>
<td>% smoker</td>
<td>38.2</td>
</tr>
<tr>
<td><strong>Mean age in years (SD)</strong></td>
<td><strong>16.1 (0.9)</strong></td>
</tr>
<tr>
<td><strong>Mean weekly alcohol units</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.47</td>
</tr>
<tr>
<td><strong>% drunk in past 30 days</strong></td>
<td>29.7</td>
</tr>
<tr>
<td><strong>SDQ Score</strong></td>
<td></td>
</tr>
<tr>
<td>Mean SDQ Conduct (SD)</td>
<td>2.4 (1.7)</td>
</tr>
<tr>
<td>Mean SDQ Hyperactivity (SD)</td>
<td>4.3 (2.3)</td>
</tr>
<tr>
<td><strong>Alcohol-related harms</strong></td>
<td></td>
</tr>
<tr>
<td>% fighting</td>
<td>19.6</td>
</tr>
<tr>
<td>% accident or injury</td>
<td>32.8</td>
</tr>
<tr>
<td>% parent problems</td>
<td>17.2</td>
</tr>
<tr>
<td>% peer problems</td>
<td>24.8</td>
</tr>
<tr>
<td>% school problems</td>
<td>14.3</td>
</tr>
<tr>
<td>% victim of theft</td>
<td>17.0</td>
</tr>
<tr>
<td>% police problem</td>
<td>11.6</td>
</tr>
<tr>
<td>% hospitalized</td>
<td>19.4</td>
</tr>
<tr>
<td>% regretted sex</td>
<td>15.7</td>
</tr>
</tbody>
</table>

<sup>a</sup> Where 1 unit equates to 8g of ethanol
Table 2: Estimate of effects on Total harms at 12 months from mediation analysis (mediator alcohol consumption at 6 months)

<table>
<thead>
<tr>
<th>SDQ Domain</th>
<th>Statistic</th>
<th>n=536</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conduct</strong></td>
<td>a coefficient</td>
<td>0.341</td>
</tr>
<tr>
<td></td>
<td>b coefficient</td>
<td>0.0806</td>
</tr>
<tr>
<td></td>
<td>Indirect effect (a x b)</td>
<td>0.0275</td>
</tr>
<tr>
<td></td>
<td>Alcohol consumption 6 months</td>
<td>(-0.00726 to 0.0623)</td>
</tr>
<tr>
<td></td>
<td>(95% bootstrap CI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Direct effect of baseline</td>
<td>0.452</td>
</tr>
<tr>
<td></td>
<td>conduct score</td>
<td>(0.256 to 0.648)</td>
</tr>
<tr>
<td></td>
<td>(95% bootstrap CI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total effect</td>
<td>0.479</td>
</tr>
<tr>
<td></td>
<td>Proportion of total effect</td>
<td>0.0574</td>
</tr>
<tr>
<td></td>
<td>mediated</td>
<td></td>
</tr>
<tr>
<td><strong>Hyperactivity</strong></td>
<td>a coefficient</td>
<td>0.239</td>
</tr>
<tr>
<td></td>
<td>b coefficient</td>
<td>0.087</td>
</tr>
<tr>
<td></td>
<td>Indirect effect (a x b)</td>
<td>0.0208</td>
</tr>
<tr>
<td></td>
<td>Alcohol consumption 6 months</td>
<td>(-0.00517 to 0.0469)</td>
</tr>
<tr>
<td></td>
<td>(95% bootstrap CI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Direct effect of baseline</td>
<td>0.0996</td>
</tr>
<tr>
<td></td>
<td>hyperactivity score</td>
<td>(-0.0224 to 0.222)</td>
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<tr>
<td></td>
<td>(95% bootstrap CI)</td>
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<tr>
<td></td>
<td>Total effect</td>
<td>0.120</td>
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<tr>
<td></td>
<td>Proportion of total effect</td>
<td>1.21</td>
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