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The mediating role of deviant peers on the link between depressed mood and harmful drinking: Analyses in a population-based sample of adolescents

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Abstract

Purpose—One's peer group can have a strong impact on depressed mood and harmful drinking in adolescence. It remains unclear whether affiliation with deviant peers explains the link between these traits. Our study aims to: a) explore the developmental relationship between harmful drinking and depressed mood in adolescence; and b) establish to which extent affiliation with deviant peers explains this relationship.

Methods—4,863 adolescents from the Avon Longitudinal Study of Parents and Children (ALSPAC) were assessed between the ages of 14 and 16. Harmful drinking was established using age-appropriate measures: the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) in mid-adolescence (age 14) and the Alcohol Use Disorders Identification Test (AUDIT) in late adolescence (age 16). Depressed mood was measured by the Short Mood and Feelings Questionnaire (SMFQ) at both ages. Affiliation with deviant peers was assessed at age 15.

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Declaration of interest
None.

Implications and Contribution:
Harmful alcohol use at age 14 was found to be linked to higher levels of depressed mood two years later. This association acted indirectly via affiliation with deviant peers at age 15 and suggests that isolation from the normative peer group due to alcohol misuse increases risk of depression.

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Results—Harmful drinking at age 14 predicted depressed mood two years later. This association was explained by affiliation with deviant peers and remained present even after adjustment for earlier depressed mood. Depressed mood at age 14 predicted harmful drinking at age 16 via affiliation with deviant peers; however, this indirect effect disappeared when adjusting for adolescents' earlier harmful alcohol use (age 14). No gender differences were observed.

Conclusions—Adolescents who engage in early harmful drinking and subsequently become affiliated with a deviant peer group may be at particular risk of later depressed mood.

Keywords

harmful drinking; depressed mood; adolescence; deviant peers; ALSPAC; prospective birth cohort study; longitudinal

Introduction

Harmful drinking and depression frequently co-occur and the combination of both traits is associated with increased risk of a range of adverse outcomes, including lower quality of life, social impairment and suicide [1]. A number of studies have attempted to clarify the developmental nature of this relationship but results have been inconsistent [1–3].

Mixed results may be due to variation in research methodology and design across studies [4]. Discrepancies can also point to a complex aetiology which may be best understood by taking into account third variables to explain this developmental relationship [5]. The peer group represents a crucial social influence in adolescents' lives and plays a role in alcohol initiation and progression [6, 7] as well as depression [8, 9] with evidence that specific risk factors may only lead to substance misuse in the presence of other risk factors [10].

Two hypotheses offer plausible explanations for the link between depression, drinking, and peer influence. The social learning hypothesis suggests that adolescents who are rejected by their normative peer group, due to either depression or harmful drinking, may gravitate towards a deviant peer group to reduce their sense of isolation [8, 11]. Subsequently, they adopt the group's behaviours and norms through imitation and reinforcement [11]. The self-selection hypothesis suggests that adolescents actively seek peers who engage in similar behaviours [7, 12, 13]. Based on evidence that depression co-occurs with conduct problems, depressed adolescents may pursue antisocial activities and engage with deviant peers who share similar behaviours [14–16]. Similarly, teenagers who engage in harmful drinking may pursue peers who share their drinking norms. Affiliation with deviant peers increases levels of depression as this peer group does not offer suitable support and activities conducted with deviant peers may lead to negative consequences (e.g., academic failure) [8].

Mid-adolescence is a critical age as it is characterised by an increase in depression rates and drinking behaviour and, additionally, the role of peers is at its peak [17–19]. Thus, it is important to understand the relationship between these domains in order to develop effective interventions. However, to our knowledge, no study has explored this relationship. Using data from a large birth cohort study, we aimed to fill this gap and establish:

1. the relationship between: a) depressed mood at age 14 and harmful drinking at age 16, and b) harmful drinking at age 14 and depressed mood two years later;
2. to what extent these relationships are mediated by affiliation with deviant peers at age 15 (as depicted in figure 1).

Owing to evidence that depressed mood is a stronger predictor of harmful drinking for females than males [20, 21], we also aimed to:

3. explore potential gender differences.

Materials & Method

Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is an ongoing longitudinal cohort study, which started in 1991-1992. The core sample consisted of 14,541 pregnancies. These initial pregnancies resulted in 14,062 live births with 13,988 children alive at 1 year of age. The study website contains details of all the data available through a fully searchable data dictionary [22]. The ALSPAC cohort is similar to the overall UK population as indicated by comparisons with the 1991 census [23]. Ethical approval for the study was obtained from the ALSPAC Law and Ethics Committee and Local Research Ethics Committees.

In the present study, adolescents were assessed at ages 13 (mean = 12.8, SD = 0.2), 14 (mean = 13.8, SD = 0.02), 15 (mean = 15.5, SD = 0.3) and 16 (mean = 16.7, SD = 0.2). At age 16, 9,996 questionnaires were sent to the study children and 5,126 were returned. Young people who returned their questionnaires were more likely to be female (43% vs. 28% male; $\chi^2(1) = 338.8$, $p<.001$), from a family with higher social class (48% vs. low 31%; $\chi^2(1) = 326.1$, $p<.001$) and education level (57% vs. low 32%; $\chi^2(1) = 547.9$, $p<.001$). Respondents who were less likely to return the questionnaires were more likely to be smokers (44% vs. non-smoker 36%; $\chi^2(1) = 14.8$, $p<.001$) and to report an onset of alcohol use prior to age 13 (40% vs. later 35%; $\chi^2(1) = 12.4$, $p<.001$). The analyses presented below are based on 4,863 (60% female) respondents with complete information on the outcome measures (i.e., depressed mood and harmful drinking).

Measures

Alcohol measure at age 14—Alcohol information at age 14 (predictor in model 2) was collected using the adolescent version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) [24]. Following the methodology used by Saraceno and colleagues (2002), four items were used to estimate a harmful drinking measure: 1) frequency of drinking without parents' permission; 2) frequency of having a whole drink; 3) largest number of whole drinks within a 24-hour period; and 4) whether the adolescent had ever been drunk ($\alpha = .78$).

Alcohol measures at age 16—At age 16, when alcohol use is more established than at age 14, we used the Alcohol Use Disorder Identification Test (AUDIT; outcome in model 1) [25]. This is a brief screening tool to identify individuals with alcohol-related problems

and comprises 10 multiple-choice items (α=.77). Two AUDIT items were dropped as they were rare in the sample (<10%; revised 8-item α= .78); i.e., 'how frequently have you had a drink first thing in the morning' and 'a family member/friend/doctor has shown concern about your drinking'. Previous studies, similarly, found these items did not seem to capture harmful drinking in adolescence [26].

Depressed mood—Depressed mood was measured using the Short Mood and Feelings Questionnaire (SMFQ) at ages 14 (i.e., predictor in model 1) and 16 (i.e., outcome in model 2) [27]. The questionnaire comprises 13 items (age 14: α = .86; age 16: α = .91). To improve model estimation, we dropped two items at age 14 as they were rare (<10%); i.e., I have 'been a bad person' and 'done everything wrong' (revised 11-item α = .84).

Deviant peers at age 15—Affiliation with deviant peers (i.e., mediator) was assessed using items from the Edinburgh Study of Youth Transitions and Crime (ESYTC) to capture deviant activity in the peer network. The questionnaire asked respondents to report whether their friends had engaged in delinquent behaviour (e.g., shoplifting) and substance use in the past year. Rare (<10%) or highly frequent (>80%) items were excluded from the analyses as they were not very informative and produced small cell-counts which can lead to estimate bias. Hence, a 12-item construct was derived (α= .85).

Confounders—Background confounders were selected *a priori* because they are related to depressed mood, deviant peers and harmful drinking [1, 28]. They were from two domains: family environment (i.e., parental drinking and depression at 12 years), and socio-economic status (i.e., financial difficulties at 11 years and parental education at the 32nd week of gestation) as reported by the child's mother as well as partner.

The model was also adjusted for the Strengths & Difficulties Questionnaire (SDQ) conduct problem scale which consists of five items based on carers' report at age 13 [29]. Carers were asked whether their children had engaged in specific deviant behaviours (e.g., lying) in the past six months. Stealing 'from home, school or elsewhere' was rare (i.e., <10%) and we dropped this item. The reliability Cronbach's α for the four-item scale (α= .58) was comparable to that reported for the five-item scale [29]. The scores at ages 13 and 16 were correlated (rho = .52, 95% CI: .50 to .54), indicating consistency over time.

The model was also adjusted for peers' deviant behaviour at age 13. This consisted of five items (α=.70) equivalent to those assessed at age 15 (step 2). Respondents were asked whether their friends had engaged in delinquent behaviours in the past six months and offered a 'yes/no' response.

Statistical analysis

Descriptive statistics on the main measures were calculated by summing the questionnaire items for complete cases. Our main aims were tested using structural equation modelling (SEM) on the imputed data. Latent constructs in the SEM analysis were defined using questionnaire items (Table S1).

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Preliminary confirmatory factor analyses were conducted to assess the measurement model fit to the data. The weighted least squares mean variance (WLSMV) estimator in Mplus 7 [30] was used and model fit evaluated with the following goodness-of-fit indices: chi-square ($p > .05$); Tucker-Lewis Index (TLI > .95); Comparative Fit Index (CFI >.95); and Root Mean Square Error of Approximation (RMSEA <.05) [31]. SEM analyses were conducted to assess whether affiliation with deviant peers explained the relationship between depressed mood at age 14 and harmful drinking at age 16 (model 1; figure 1a) and, similarly, between harmful drinking at age 14 and depressed mood at age 16 (model 2; figure 1b).

The indirect impact of the deviant peer group on the relationship between depression and harmful drinking was estimated using the product of coefficient method (i.e., path a*path b), which has good power and low type I error [32]. To estimate the strength of the indirect effect, we calculated the effect proportion mediated (i.e., $B_{indirect}/B_{indirect}B_{direct}$). This proportion was calculated using the absolute values for the direct and indirect effects [32, 33]. Standard errors were calculated using the Delta method, which allowed us to calculate 95% confidence intervals equivalent to those obtained using 5000 bootstrap cycles (Table S2).

Following our main analyses, we conducted a series of sensitivity analyses where we adjusted our models for the confounders described above [33]. In step 1, we adjusted the model for background variables while in step 2 we adjusted it for both background variables and the SDQ conduct problems scale. This step allows us to assess whether affiliation with deviant peers is mostly driven by the adolescents' own conduct problem, following evidence that harmful drinking and depressed mood are co-morbid with conduct problems [14]. Finally, following recent guidelines [34], we further adjusted the models for an earlier measure of the mediator (i.e., affiliation with deviant peers at age 13; step 3) and earlier outcome measures (i.e., age. model 1: harmful drinking; model 2: depressed mood; step 4) at age 14.

Moderation by gender—We assessed whether gender moderated the direct, indirect and total effects. This was achieved using a multi-group approach in which additional parameters capturing the difference in the strength of the direct, indirect and total paths between males and females (i.e., female – male) were derived using the model-constraint option. An advantage of this approach was that the resulting z-test (i.e., estimate/SE) of the difference effect can be pooled across imputed datasets, while this is not the case for likelihood based chi-square tests.

Prior to exploring gender-specific patterns, measurement invariance across gender was tested using multi-group confirmatory factor analysis (MGCFA) to ensure our main measures were well-specified and captured equivalent constructs across genders. This method compares two models; i.e., a baseline model where loadings and thresholds are unconstrained vs. a model where they are constrained across genders [30]. Measurement invariance is established if: 1) the constrained model offers a good fit to the data (i.e., configural invariance), and 2) the difference of model fit between the constrained and the unconstrained model is small (ΔCFI<.01 & RMESEA <.015; i.e., metric invariance) [35].

Missing data and imputation—All longitudinal studies are affected by attrition [36] and the ALSPAC study, which has been running for 20 years, is no exception. However, sophisticated imputation strategies have been developed which address bias introduced by drop-out. We used multiple imputation by chained equations (MICE) in Stata 11 with 50 imputations. Multiple imputation relies on the assumption that data are missing at random (MAR) [37]. We included earlier measures of the main variables and auxiliary variables, associated with the outcomes and their missingness, in our imputation model to increase the likelihood of the MAR assumption and improve model estimation [37].

Our analyses are based on imputed cases with complete information on the outcome measures, as outcome imputation has been found to lead to biased estimates [37]. Sensitivity analyses showed that the distributions of the imputed items and complete cases were comparable. Moreover, analyses based on imputed and complete cases produced equivalent result patterns (Table S3).

Results

Descriptive statistics and intercorrelations are summarised in Table 1 for complete cases.

The confirmatory factor analyses showed that the measurement model offered a good fit to the data for both models (Table S4). The structural model fit and results for the model adjusted with all confounders (step 4) are reported in Figure S1.

Model 1 (depressed mood and later harmful drinking)

Depressed mood at age 14 was associated with more deviant peer behaviour at age 15 (B =. 21, 95% CI: .16 to .26, z = 8.2, p<.001; path a) which was, subsequently, associated with more harmful drinking at age 16 (B =.34, 95% CI: .30 to .38, z = 18.9, p<.001; path b). Depressed mood at age 14 predicted greater harmful drinking two years later (B_{total} =.08, 95%CI: .04 to .11, z = 3.8, p<.001) and 96% of this association was explained by affiliation with deviant peers ($B_{indirect}$ = 0.07, 95% CI: .05 to .09, z = 7.8, p <.001). Adjusting for the earlier measure of deviant peers reduced the indirect effect, which disappeared after subsequently adjusting for adolescents' earlier harmful alcohol use at age 14 (Table 2).

Model 2 (harmful drinking and later depressed mood)

Harmful drinking at age 14 was associated with more deviant peer behaviour at age 15 (B =. 50, 95%CI: .46 to .54, z = 23.7, p<.001; path a) which was, subsequently, associated with greater depressed mood at age 16 (B =.18, 95%CI: .13 to .23, z = 7.3, p<.001; path b). Harmful drinking at age 14 predicted greater depressed mood two years later (B_{total} =.09, 95%CI: .05 to .13, z = 4.2, p<.001) and 95% of this association was explained by deviant peers ($B_{indirect}$ = 0.09, 95% CI: .07 to .12, z = 7.2, p<.001). After adjusting the model for depressed mood and deviant peers in early adolescence, the indirect effect was weakened but still present ($B_{indirect}$ = .04, 95% CI: .02 to .06; Table 3), explaining 38% of the total effect (Figure S1b).

Moderation by gender

The MGCFA showed the unconstrained model offered a good fit to the data and constraining factor loadings across genders did not decrease the model fit (Table S6). We, therefore, conducted multi-group analyses with factor loadings and thresholds fixed to be equal across gender. These analyses showed that the total, indirect and direct effects did not differ for males and females (Table 4).

Discussion

Our results showed that depressed mood experienced at age 14 was associated with greater harmful drinking at age 16 and, conversely, harmful drinking at age 14 was linked to higher levels of depressed mood two years later. This is, to our knowledge, the first study to indicate that affiliation with deviant peers mediates this association. These mechanisms were equivalent across gender. A previous study, based on ALSPAC data, found that depressed mood at age 10 predicted harmful drinking at 14 among females only [20]. No observed gender differences may be due to the fact that, by the age of 15, British male and female adolescents engage in similar drinking patterns [19]. Moreover, our findings show that the indirect mechanisms via peers were not explained by family environment or socio-economic status.

The indirect effect via peers was weakened, but remained present in both models, after adjusting for deviant peers at age 13. These results suggest that affiliation with deviant peers is not driven by the adolescents' own deviant behaviour. Thus, results support the social learning hypothesis that adolescents, isolated by their normative peer group due to their depressed mood or alcohol misuse, engage with deviant peers. Subsequently, they adopt their behaviours and may experience greater depressed mood [8, 11].

Further sensitivity analyses showed that affiliation with deviant peers at age 15 accounted for approximately 40% of the relationship between harmful drinking at age 14 and depressed mood two years later (model 2) after adjusting for depressed mood at baseline. This suggests that affiliation with deviant peers creates a vulnerability to developing depressed mood in youngsters who engage in harmful drinking. After adjusting the model for earlier depressed mood, a negative direct link was found between harmful drinking at age 14 and depressed mood two years later. Thus, our results suggest that higher levels of depressed mood at age 16 are mostly elicited by affiliation with deviant peers and early depressed mood rather than the physiological effects of alcohol [38]. Our results seem to disentangle the association between harmful drinking at age 14 and depressed mood two years later by capturing two separate processes, which may be related to drinking motives [39]. Indeed, we observed a (positive) indirect effect via deviant peers which captures the mechanism for adolescents who, rejected by their normative peer, gravitate towards a deviant peer group and may engage in drinking to conform (i.e., social learning hypothesis). As discussed, affiliation with deviant peers leads to negative mood as they do not offer suitable support [8]. In contrast, the (negative) direct effect may capture drinking amongst those adolescents who engage in alcohol use to socialise. Our findings are in line with evidence that alcohol use is widespread among UK adolescents [19] and that drinking to socialise is a common practice in adolescence [40]. Drinking to socialise may not lead to depressed mood as it is associated

with increased friendship quality and quantity [40]. Future studies aiming to replicate our findings and to further explore these hypotheses will shed further light on the differences in risk of depression amongst alcohol-using adolescents.

In contrast, adjusting for harmful drinking at baseline weakened both the total effect of depressed mood on harmful drinking at age 16 and its indirect effect via peers (model 1). Our study corroborates evidence that alcohol use in early adolescence is a strong predictor of drinking patterns in late adolescence [41]. Thus, a more extended timeframe starting before age 14 would allow more optimal opportunities to capture the relationship between depressed mood and later harmful alcohol use.

The strengths of our study include a large prospective cohort study design and measures validated in adolescence. The study suffers from attrition; however, we used multiple imputation methods to reduce bias and increase power [37]. There are further limitations to acknowledge. Two items from the AUDIT and SMFQ were rare in the present sample and, therefore, dropped from the analyses. These exclusions may limit comparisons with existing research findings. However, inclusion of these items would likely have hindered the predictive ability of our model and lead to bias. The item indexing the number of peers who had ever drunk alcohol was excluded from the peer's construct as approximately 98% of the adolescents reported knowing someone who drinks. Items assessing peers' alcohol consumption (i.e., quantity and frequency) would have allowed us to capture harmful drinking but were not available. A further limitation is that the term 'friend' was not further defined in the questionnaire. Hence, we are not able to describe the nature of the affiliation in more detail. Nonetheless, the term captures the wider peer network which is generally influential during adolescence [42, 43]. Finally, information on peers was reported by the study children themselves and this can lead to over-estimation and confirmation bias [44]. Nonetheless, research shows that perception of peers' drinking is an important risk factor for heavy drinking in adolescence [45].

Owing to the fact that harmful drinking was assessed using age-appropriate but different measures, we were not able to conduct cross-lagged model analyses. Nonetheless, we adjusted the model for baseline measures to take into account the developmental nature of both harmful drinking and depressed mood. Finally, drinking measures were based on self-report, which may be subject to over- or under-reporting, however, the ALSPAC study team employed methods to ensure confidentiality procedures by ensuring anonymity and confidentiality [41].

In summary, our results indicate that effective intervention programs which focus on adolescent drinking and depression should take into account their co-occurrence. Mental health providers who are in contact with young people with depressed mood should be aware of the likelihood of harmful drinking. In addition, they may be able to identify adolescents at risk of depression among those engaging in harmful drinking and attending alcohol programs.

Our results also indicate that interventions should take into account adolescent's peer affiliations and socialisation. These programs could aim to modify norms within the group

and reduce peers' influence on vulnerable individuals, thereby addressing group level processes and individual susceptibility to such processes. Indeed, universal, school-based programs, which challenge beliefs that alcohol misuse is normative and provide adolescents with intrapersonal skills (e.g., assertiveness), which boost their resistance self-efficacy, have been successful at reducing harmful drinking [46].

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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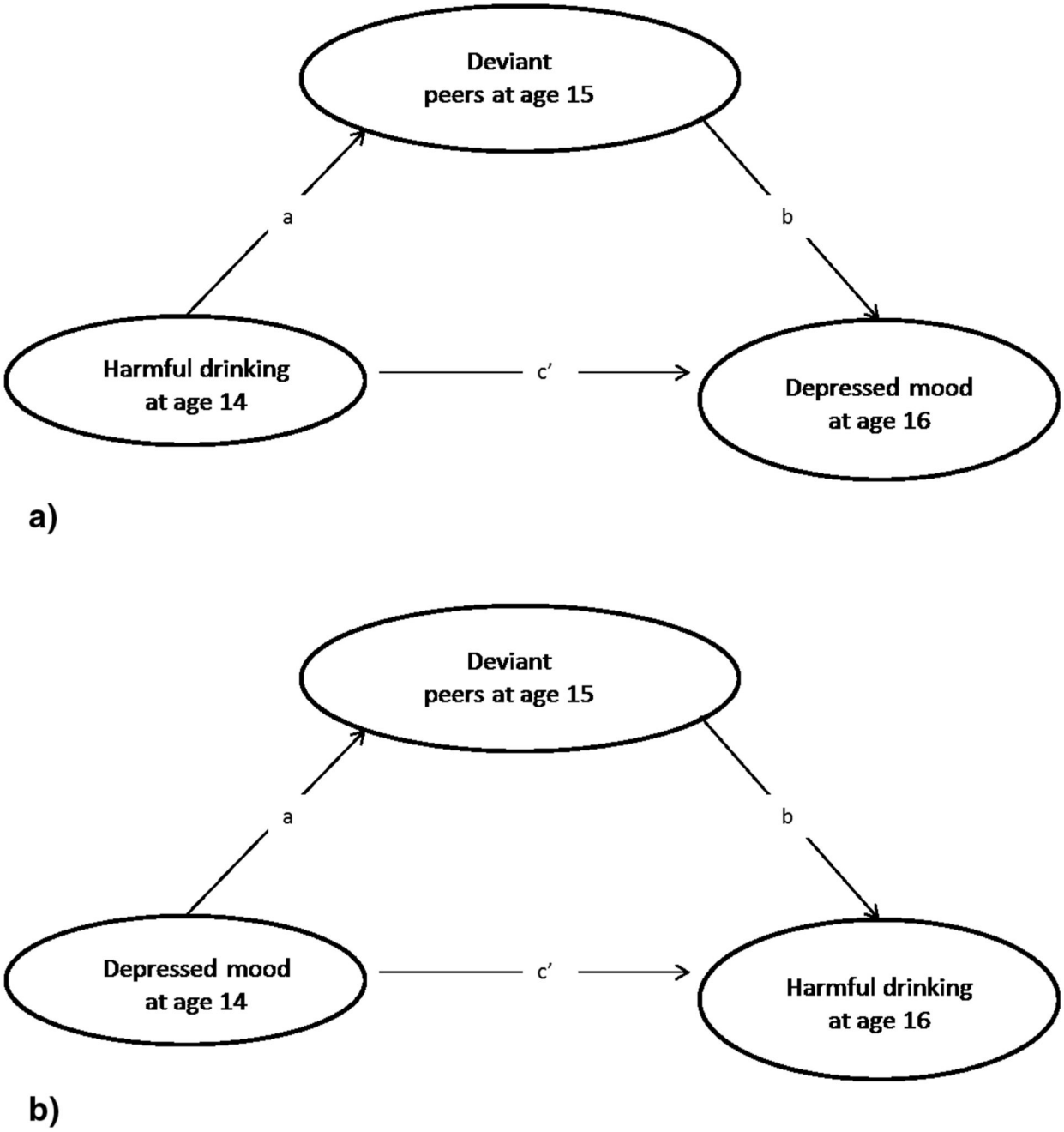

Figure 1. Representation of the mediation models

Panel a depicts model 1 which explores the predicting role of depressed mood on later problem drinking via affiliation with deviant peers; panel b depicts model 2 which explores the predicting role of problem drinking on depressed mood via deviant peer group.

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Table 1

Spearman correlations, means and standard deviation (SD) for main variables. These figures are based on the scales' scores for cases with complete information on both models (N = 1,883) and were calculated in Stata 11.

	1. AUDIT 16	2.SMFQ 16	3.SSAGA[1] 14	4. SMFQ 14	5.PEERS 15	6.PEERS 13	7. SDQ 13
1	1.00						
2	0.30**	1.00					
3	0.32**	0.33**	1.00				
4	0.33*	0.43**	0.13*	1.00			
5	0.44**	0.24**	0.29**	0.18**	1.00		
6	0.12*	0.09*	0.25**	0.11*	0.22**	1.00	
7	0.03ns	0.10*	0.001ns	0.08ns	0.01ns	0.04ns	1.00
Mean	**6.5**	**5.6**	**0.1**	**4.8**	**2.9**	**0.8**	**2.2**
SD	***5.0***	***5.3***	***1.2***	***4.3***	***2.8***	***1.0***	***0.8***

** p-values <0.001

* p-values < 0.0

[1] Following the approach followed by Saraceno and colleagues (2010), harmful drinking at age 14 was constructed using a Principal Component Analysis (PCA) approach. PCA extrapolates components with mean of 0 and standard deviation of 1.

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Table 2

Imputed data analyses: Model 1: Total, indirect and direct effects of depressed mood (i.e., predictor) on harmful drinking (i.e., outcome) via deviant peers (i.e., mediator) with [95% CI]

	Step 0	Step 1	Step 2	Step 3	Step 4
Total Effect	.075 [.04, .11]	.074 [.03, .11]	.056 [.02, .10]	.022 [-.02, .06]	-.014 [-.05, .03]
Indirect effect	.072 [.05, .09]	.069 [.05, .09]	.055 [.04, .07]	.021 [.003, .04]	.008 [-.004, .02]
Direct effect	.003 [-.04, .04]	.005 [-.03, .04]	.001 [-.04, .04]	.001 [-.04, .04]	-.022 [-.06, .02]
Effect proportion mediated (indirect/total ratio)	96%	93%	100%	100%	27%

Step 0: Unadjusted for covariates

Step 1: Adjusted for background covariates: financial difficulties, family education level, parents' alcohol consumption, and parents' depression

Step 2: Adjusted for background covariates + conduct problems scale (SDQ)

Step 3: Adjusted for background covariates + conduct problems scale (SDQ) + earlier mediator at age 13 (i.e., deviant peers)

Step 4: Adjusted for background covariates + conduct problems scale (SDQ) + deviant peers at age 13 + earlier outcome at age 14 (i.e., harmful drinking)

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Table 3

Imputed data analyses: Model 2: Total, indirect and direct effects of harmful drinking (i.e., predictor) on depressed mood (i.e., outcome) via deviant peers (i.e., mediator) with [95% CI]

	Step 0	Step 1	Step 2	Step 3	Step 4
Total Effect	.092 [.05, .13]	.079 [.04, .12]	.042 [.01, .08]	.015 [-.03, .06]	-.026 [-.07, .01]
Indirect effect	.087 [.07, .12]	.087 [.06, .11]	.071 [.05, .09]	.046 [.03, .07]	.039 [.02, .06]
Direct effect	-.005 [-.05, .04]	-.008 [-.06, .04]	-.029 [-.08, .02]	-.032 [-.08, .02]	-.065 [-.11, -.02]
Effect proportion mediated (indirect/total ratio)	95%	91%	71%	59%	38%

Step 0: Unadjusted for covariates

Step 1: Adjusted for background covariates: financial difficulties, family education level, parents' alcohol consumption, and parents' depression

Step 2: Adjusted for background covariates + conduct problems scale (SDQ)

Step 3: Adjusted for background covariates + conduct problems scale (SDQ) + earlier mediator at age 13 (i.e., deviant peers)

Step 4: Adjusted for background covariates + conduct problems scale (SDQ) + deviant peers at age 13 + earlier outcome at age 14 (i.e., depressed mood)

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Table 4

Imputed data analysis: Total, indirect and direct effects [95% CI] for male and female respondents, estimated difference score (i.e., female-male) with [95%CIs) and p value. Model 1: Effect of depressed mood (i.e., predictor) on harmful drinking (i.e., outcome) via deviant peers (i.e., mediator) & Model 2: Effect of harmful drinking (i.e., predictor) on depressed mood (i.e., outcome) via deviant peers (i.e., mediator)

		Model 1		Model 2	
Estimate & 95%CIs	**Difference [95%CIs]**	**Estimate & 95%Cis**	**Difference [95%CIs]**		
Total effect	Male	.04 [-.02, .10]	.47 [-.03, .13], p= 0.2	.09 [.03, .16]	-.01 [-.09, .07],
	Female	.09 [.04, .14]		.08 [.03, .13]	p = 0.8
Indirect effect	Male	.08 [.05, .12]	.001 [-.04, .04], p = 1.0	.12 [.07, .17]	-.01 [-.07, .05],
	Female	.08 [.06, .11]		.11 [.08, .14]	p = 0.8
Direct effect	Male	-.04 [-.11, .02]	.05 [-.03, .12], p= 0.3	-.03 [-.10, .05]	-.002 [-.10, .10],
	Female	.01 [-.05, .05]		-.03 [-.09, .03]	p= 1.0