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The Developmental Relationship between Depressive Symptoms in Adolescence and Harmful Drinking in Emerging Adulthood: The Role of Peers and Parents

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Abstract

Depressive symptoms have been linked to the development of harmful drinking in adolescence but it remains unclear to what extent this effect continues into emerging adulthood. Deviant peers represent a risk factor while parental monitoring is a protective factor for harmful drinking. The study explored the relationship between depressive symptoms and harmful drinking between early adolescence and emerging adulthood. We also assessed to what extent this relationship is mediated by the influence of deviant peers and whether parental monitoring weakens this process. The sample consisted of 2,964 adolescents (64% females) from the Avon Longitudinal Study of Parents and Children (ALSPAC) study assessed between the ages of 14 and 19. Using Structural Equation Modelling, we found that affiliation with deviant peers mediated the association between depressive symptoms and harmful drinking after adjustment for socio-demographic variables, parental drinking and depression, teenager's sex, conduct problems as well as drinking and depressive symptoms in early adolescence. We also found that parental control and solicitation reduced the influence of deviant peers on harmful drinking. The results indicate that prevention programs should offer adolescents training for refusal skills with peers and monitoring skills training for parents may have a long-term effect at weakening peer influences on harmful drinking.

Keywords

harmful drinking; depressive symptoms; parental monitoring; deviant peers; ALSPAC; prospective birth cohort study

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Introduction

Emerging adulthood is an important developmental stage characterized by the transition between adolescence and early adulthood and changes in several areas of life, such as employment or education, and relationships (Arnett 2005; Arnett 2000). This phase is marked by the development of new social networks and peers become more important as individuals start to establish their identity and gain autonomy from their families (Doumen et al. 2012). Alcohol consumption tends to escalate through late adolescence and peaks in emerging adulthood (McCambridge et al. 2011) and alcohol problems present at this stage are strongly related to later morbidity and mortality (Hingson et al. 2009). Depression experienced in adolescence has been identified as a risk factor for higher alcohol consumption and alcohol-related problems in adolescence as well as emerging adulthood (Saraceno et al. 2012; Marmorstein 2009). Research exploring the risk and protective factors that may explain this association can provide a basis for targeted interventions.

Deviant Peers

The peer group represents one of the strongest risk factors for the development of alcohol use throughout adolescence and into emerging adulthood (Van Ryzin et al. 2012a; Monahan et al. 2009). Emerging adults develop new social networks as they enter new social environments (e.g., university or work) (Kendler et al. 2015). Research has been conducted in college students to understand how these new networks influence drinking in emerging adulthood and two alternative hypothesis have been explored to explain the impact of peers on alcohol use: social selection and social learning (Kendler et al. 2015; Parra et al. 2007). According to the social learning hypothesis, young people adopt the group's behaviors and norms through imitation and reinforcement (Kandel 1983). In contrast, the self-selection hypothesis suggests that they may actively seek peers who engage in similar behaviors (e.g., heavy alcohol use) (Osgood et al. 2013b). It is particularly important to understand these alternative hypotheses among individuals who experience depressive symptoms as they are at greater risk of peer rejection and, hence, more likely to affiliate with deviant peers to reduce feelings of social isolation (Laird et al. 2001; Monahan et al. 2009). Evidence also shows that children and adolescents who, due to their deviant behavior, experience rejection by their normative peer group, tend to affiliate with each other (Laird et al. 2001; Coie et al. 1995). In a previous study, we found that affiliation with deviant peers at age 15 partly mediated the association between depressive symptoms experienced at age 14 and harmful drinking at age 16 (Pesola et al. 2015). Indeed, adolescents who experienced depressive symptoms were more likely to affiliate with deviant peers and, subsequently, reported higher levels of harmful drinking. This indirect effect was still present after adjusting for the adolescent's own antisocial behavior. This suggests that affiliation with deviant peers is not purely driven by self-selection but may also be explained by social learning. No study to date has explored the long-lasting effect of depressive symptoms, experienced in adolescence, on peer networks and drinking patterns in emerging adulthood.

Parental monitoring

Evidence shows that parental monitoring represents a protective factor in alcohol and substance use (Tilton-Weaver et al. 2013; Fletcher et al. 2004) by delaying onset of use and

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reducing the risk of misuse (DeVore and Ginsburg 2005). Moreover, the protective effect of parental monitoring has been found to last throughout adolescence into emerging adulthood (Bahr et al. 2005; Wood et al. 2004). Parental monitoring is characterized by a number of activities implemented by parents in order to gain knowledge of their offspring's whereabouts (Kerr and Stattin 2000; Stattin and Kerr 2000). Stattin and Kerr (2002) suggested that parental monitoring, often operationalized as knowledge, actually consists of different types of activities that either facilitate the child's willingness to disclose information or active monitoring via solicitation of information and control by carers. Voluntary disclosure, as reported by both parents and children, has previously been found to be more effective than control and solicitation at deterring deviant behavior (including alcohol and substance use) among 14-year olds (Stattin and Kerr, 2000). In contrast, Fletcher and colleagues (Fletcher et al. 2004) found that parental control and solicitation were good predictors of lower substance use and problem behavior in a sample of 14-to18-year olds. Collectively, the literature supports the existence of different types of monitoring activities, but is inconclusive on which aspect is more effective at reducing risk behaviors.

Parental monitoring and deviant peers interplay

Parental monitoring is hypothesized to act directly by limiting the opportunities for engagement in risky behaviors as well as indirectly by reducing the influence of deviant peers (Kiesner et al. 2010; Simons-Morton and Chen 2005). However, research exploring the interplay between monitoring and peers' influence is generally cross-sectional (Bahr et al. 2005) or focuses on narrow time points (Aseltine 1995), which do not allow us to discern the development over time. To our knowledge, only one study to date has explored the effect of this interplay on substance use longitudinally from age 12 to 23 (Van Ryzin et al. 2012b). Results showed that deviant peers directly predicted substance use across the whole period. In contrast, parental monitoring was found to indirectly predict substance use by reducing affiliation with deviant peers throughout adolescence, but had no effect in emerging adulthood. The study, however, did not specifically focus on adolescents with depressive symptoms, who represent a vulnerable group, and indexed monitoring as knowledge rather than exploring the impact of the different monitoring activities.

Overall, research to date indicates that, while deviant peers represent a risk factor for alcohol use, parental monitoring is associated with lower levels of substance abuse; however, research is not conclusive on whether the effect of monitoring acts directly or indirectly by weakening the influence of peers. Additionally, research has not identified the monitoring activity which is more likely to weaken the effect of peers on drinking behavior. Our study aimed to address this gap in the literature and explore the interplay between parental monitoring and deviant peers on the long-term association between depressive symptoms experienced during adolescence and harmful drinking in emerging adulthood.

The current study

Using data from a longitudinal birth cohort study, the present study addresses 3 research questions. The first question is whether depressive symptoms experienced in adolescence have a long-lasting impact on harmful drinking at age 19. We hypothesized that depressive

symptoms at age 14 would be associated with increased harmful drinking five years later based on previous studies that observed a developmental relationship between depressive symptoms in adolescence and later drinking behavior (Saraceno et al. 2012; Marmorstein 2009). The second question is whether the association between depressive symptoms and harmful drinking is mediated by affiliation with deviant peers at age 18. Based on our previous study (Pesola et al. 2015), we hypothesized that adolescents who experienced depressive symptoms at age 14 would be more likely to affiliate with deviant peers at age 18 and, subsequently, affiliation with deviant peers would be linked with increased harmful drinking at age 19. The third question was whether parental monitoring could indirectly reduce harmful drinking by weakening the indirect effect of peers. Moreover, we were interested in identifying the most effective monitoring activity and, finally, whether parental monitoring impacted more strongly on the relationship between depressive symptoms and affiliation with deviant peers (path a; Figure 1) or on the relationship between affiliation with deviant peers and harmful alcohol use (path b; Figure 1). These associations are explored using a comprehensive model that also takes confounders into account. Indeed, evidence indicates that harmful drinking and depressive symptoms are co-morbid with conduct problems (Mason et al. 2008; Simons et al. 1991). Moreover, family histories of depression and alcohol use together with family socio-demographic characteristics are associated with depressive symptoms, harmful drinking and deviant peers (Saraceno et al. 2009; Fergusson and Horwood 1999).

Method

Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is an ongoing longitudinal birth cohort study, which started in 1991-1992 in the UK (http://www.bristol.ac.uk/alspac/). The core sample consisted of 14,541 pregnant women. These initial pregnancies resulted in 14,062 live births with 13,988 children alive at 1 year of age. These initial women, their children and partners have been regularly followed over the past two decades via postal questionnaires, clinic interview or clinic computing tasks. The study website contains details of all the data available through a fully searchable data dictionary (ALSPAC 2012). The ALSPAC cohort sample is similar to the overall UK population as indicated by comparisons with the 1991 census and the 1970 Child Health and Education Study (Boyd et al. 2013; Golding et al. 2001). The original aim of the ALSPAC study was to explore how environmental and genetic factors interact and influence health. Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and Local Research Ethics Committees.

The present sample consisted of young people who were assessed at age 13 (M = 12.8, SD = .23), 14 (M = 13.9, SD = .21), 18 (M = 17.8, SD = .46), and 19 (M = 18.7, SD = .49). The questionnaires at age 19 were sent to 9,505 individuals (68% of the total sample) and returned by 3,228 (34% of the contacted sample). Our final sample consisted of 2,964 (64% female) participants who returned the questionnaires with complete information on harmful drinking at age 19 (i.e., outcome). This decision was driven by guidelines on handling missing data (see the multiple imputation section). Respondents who provided information

on their alcohol behavior at age 19 were more likely to be female (64% vs. 36% male; $\chi^2(1) = 372.7$, $p < .001$) and from a higher social class ($\chi^2(5) = 286.4$, $p < .001$).

Measures

Alcohol use—Harmful drinking at age 19 was measured using the Alcohol Use Disorder Identification Test (AUDIT) (Babor et al. 2001). This is a brief self-report screening tool to identify individuals with alcohol-related problems and comprises 10 multiple-choice items with good internal validity (α =.75). Respondents are required to rate the items in relation to the past year (e.g., "How often were you unable to stop drinking"). We dropped two AUDIT items as they were rare in the sample (< 5%; revised 8-item α = . 75); 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 among adolescents (Chung et al. 2002).

Depressive symptoms—Depressive symptoms were measured using the Short Mood and Feelings Questionnaire (SMFQ) at age 14 (Angold et al. 1995; Angold et al. 2002). The self-report questionnaire requires respondents to provide information on how they have been feeling in the last 2 weeks across 13 items (e.g., "I felt miserable") with good internal consistency at age 14 (α = .84). To improve model estimation, we dropped two items at age 14 as they were rare in the sample (< 5%); i.e., I have 'been a bad person' and 'done everything wrong' (revised 11-item α = .84).

Deviant peers—Deviant activity in the peer group was indexed using a self-report questionnaire from the Edinburgh Study of Youth Transitions and Crime (ESYTC) at age 18 (Smith and McVie 2003). Considering the changes experienced by emerging adults and the development of new friendships (Doumen et al. 2012), peers frequented at age 18 are expected to reflect this new networks. The questionnaire asks respondents to report whether their friends had engaged in delinquent behavior (e.g., "shoplifting"). To improve model estimation, we excluded rare (< 5%) items from the analyses as they produced small cell-counts, which can lead to estimate bias (e.g., burglary or racial attack). Hence, a 5-item construct was derived (α = .73).

Parental monitoring—Parental monitoring was assessed using the Parental Monitoring Questionnaire (Stattin and Kerr 2000) completed by adolescents at age 14. The questionnaire consists of 25 items, which ask respondents to rate the frequency of specific behaviors. The questionnaire's items load onto four separate subscales. One scale assesses parental monitoring (10 items, α = .81), which captures parental knowledge of their child's whereabouts and activities using items such as "frequency carers know what teenager does in free time"; this scale will be referred to as 'parental knowledge'. The other scales capture different sources of parental knowledge: child disclosure (5 items, α = .74) using items such as "frequency teenager tells carers about what they did/where they were in the evening"; parental solicitation (5 items, α = .68) with items such as "frequency carers ask teenager what happened in free time"; and parental control (5 items, α = .74) with items such as "frequency teenager has to ask carers, before they can make plans for a Saturday night".

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Background confounders—The background confounders included sex as well as variables from two domains: family environment (i.e., parental drinking and depression, collected at child age 33 months), and socio-economic status (i.e., financial difficulties at child age 11 years and parental education at the 32nd week of gestation) as reported by the child's mother and partner.

Strengths and Difficulties Questionnaire—The conduct problem scale consisted of five items based on carers' report of their children's conduct problems (e.g., 'lying and cheating') when the child was 13 years old (Goodman 1997). Carers reported that stealing 'from home, school or elsewhere' was rare (< 5%) and we dropped this item to avoid potential estimate bias. The Cronbach's α for the four-item scale was .52, which is comparable to the value obtained using the five-item scale in our sample (5-item α = .53) and in previous population-based studies (Goodman 1997; Giannakopoulos et al. 2009; van Leeuwen et al. 2006).

Alcohol use at age 13—The alcohol measure was constructed using three items from the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al. 1994; Saraceno et al. 2012). These items were: number of drinks in the past 30 days, number of drinks in a typical week, and frequency of having 3 or more drinks in 24 hours (α = .89).

Deviant peers at age 13—The deviant peers' construct was created using five items from the Edinburgh Study of Youth Transitions and Crime (ESYTC) administered at age 13 (e.g., "stealing"). These items corresponded to those used to define deviant peers at age 18 (α = .63).

Statistical analyses

Descriptive statistics on the main measures were based on the questionnaires' sum scores for complete cases. We conducted Structural Equation Modelling (SEM) analysis to assess the developmental relationships between depressive symptoms and harmful drinking via peers. Specifically, we aimed to evaluate whether adolescents who experienced depressive symptoms at age 14 were more likely to affiliate with deviant peers at age 18 and, subsequently, whether having a deviant peer group at age 18 predicted harmful drinking at age 19 (Figure 1). Latent constructs in the SEM analysis were defined using categorical observed items (i.e., questionnaire items; listed in Appendix 1). These latent constructs were continuous with higher scores indicating more 'at risk' behavior.

Preliminary confirmatory factor analyses were conducted to assess how well the measurement model fit our data. The weighted least squares mean variance (WLSMV) estimator in Mplus 7 (Muthén and Muthén 1998-2011) 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) (Hooper et al. 2008; Schreiber et al. 2006).

We estimated the indirect impact of depressive symptoms on harmful drinking via deviant peers using the product of coefficient method (i.e., path a*path b), which has demonstrated good power and low type I error (MacKinnon et al. 2002). To estimate the strength of the

indirect effect, we calculated the ratio between the indirect effect and the total effect (i.e., $B_{indirect}/B_{total}$; effect proportion mediated) (Shrout and Bolger 2002). This proportion was calculated using the absolute values for the direct and indirect effects (MacKinnon et al. 2002; MacKinnon 2008). Standard errors were calculated using the Delta method (Muthén and Muthén 1998-2011).

Following our main analyses, we adjusted the model for confounders using a step-wise approach as sensitivity analysis. In step 1, we adjusted for background variables while in step 2 we adjusted for both background variables and the SDQ conduct problems scale. Finally, following recent guidelines (Cole and Maxwell 2003) we adjusted the model for antisocial peer behavior (step 3) and alcohol use (step 4) assessed at age 13. These last two steps allow us to assess to what extent the processes captured between the ages of 14-19 years might be explained by pre-existing factors (Heron et al. 2012).

Parental monitoring—We assessed the potential impact of parental monitoring on the indirect paths (i.e., a & b; Figure 1) of the fully adjusted model (step 4), using a multi-group approach by implementing the model constraint option in Mplus (Muthén and Muthén 1998-2011). To achieve this, we split the sample into two parental monitoring groups using a median-split approach for each monitoring scale, which allowed us to identify adolescents who reported high vs. low monitoring. Thus, this multigroup approach enabled us to capture the difference in the strength of the indirect paths between adolescents who reported low and those who reported high monitoring. An advantage of this approach is that the resulting Wald 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 conducting multigroup analysis, we tested for measurement invariance across high and low scorers on the four monitoring measures using multi-group confirmatory factor analysis (MGCFA). MGCFA compares two models; i.e., a baseline model where loadings and thresholds are unconstrained vs. a model where these parameters are constrained across groups (Muthén and Muthén 1998-2011). 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) (Chen 2007; Elousa 2011; You et al. 2008). This step allowed us to establish whether we could conduct further comparisons with fixed loadings and thresholds across monitoring groups.

Missing data and imputation—As described in the methods section, like all longitudinal studies (Young et al. 2006), the ALSPAC study is affected by attrition with 34% of contacted respondents returning their questionnaires. However, sophisticated imputation strategies have been developed which address bias introduced by participant drop-out. We used multiple imputation by chained equations (MICE) in Stata 11 with 50 imputations to estimate missing data among 2,964 respondents who had complete information on harmful drinking (i.e., outcome) at age 19. This is in line with recent guidelines which indicate that outcome imputation leads to biased estimates as indexed by Monte Carlo error estimates (White et al. 2011).

Multiple imputation relies on the assumption that data are missing at random (MAR) (White et al. 2011). We included earlier measures of the main variables (e.g., depressive symptoms and alcohol use in previous years) plus auxiliary variables (i.e., crowding index, household income and smoking status), associated with the outcome and its missingness, in our imputation model (Table 1). This was done to increase the likelihood of the MAR assumption and, thus, improve the model estimation (White et al. 2011; Melotti et al. 2011).

Table 2 reports the proportion of missing data across predictors and covariates for cases with complete information on the outcome. Sensitivity analyses showed that the distributions of the imputed and complete cases were comparable.

Results

Descriptive statistics

Descriptive statistics and inter-correlations across the scales' sum scores for complete cases are summarized in Table 3.

Association between depressive symptoms and harmful drinking

The confirmatory factor analyses showed that the measurement model provided a good fit to the data ($\chi^2(321)$: 1587.8, $p< .001$; TLI: .95; CFI: .96; RMSEA: 0.036, 90%CI: .034, .038). Depressive symptoms at age 14 were positively associated with harmful drinking at age 19 (B_{total} =.053, 95%CI: .014 to .092, $z = 2.4$, $p = .02$).

Mediation effect of deviant peers

The mediation analysis showed that 91% of the association between depressive symptoms and harmful drinking was explained by affiliation with deviant peers ($B_{indirect} = 0.048$, 95%CI: .028 to .068, $z = 4.6$, $p < .001$). Adolescents who experienced depressive symptoms at age 14 were more likely to affiliate with deviant peers at age 18 ($B = 0.16$, 95%CI: .10 to .22, $z = 4.8$, p < .001) and the association with deviant peers was related to greater harmful drinking at age 19 ($B = 0.31$, 95%CI: .25 to .37, $z = 9.7$, $p < .001$). The direct effect of depressive symptoms on harmful drinking was attenuated after taking this indirect effect into account ($B_{direct} = 0.005$, 95%CI: -.034 to .044, $z = 0.2$, $p = .83$).

Adjusting for background confounders and pre-existing conduct problems produced small changes in the size of the indirect and total effects (Step 2, Table 4). After adjusting the model for alcohol use and deviant peers in early adolescence (step 4), the indirect effect was attenuated but still present ($B_{indirect} = 0.033$, 95%CI: .013 to .053, $z = 3.2$, $p = .002$), explaining 79% of the total effect.

Effect of parental monitoring on the indirect effect of peers

The MGCFA showed the constrained model offered a good fit to the data (Table 5) and, therefore, we conducted multi-group analyses with factor loadings and thresholds held equal across low and high monitoring groups.

Active monitoring was found to have an effect on the association between deviant peers at age 18 and harmful drinking at age 19 (i.e., path b Figure 1). Specifically, parental control reduced the influence of deviant peers at age 18 on subsequent harmful drinking ($w = 2.4$, $p = .006$). Indeed, the association between deviant peers and harmful alcohol use was weaker among adolescents who reported high parental control ($B = 0.23$; 95%CI: .11 to .35, $z = 3.7$, $p < .001$) compared to those who reported low control ($B = 0.46$; 95%CI: 32 to .60, $z = 6.4$, $p < .001$).

Similarly, parental solicitation reduced the influence of deviant peers on harmful drinking ($w = 2.8$, $p = .005$) where this association was weaker among adolescents who experienced high levels of solicitations (B = 0.23; 95%CI: .11 to .35, $z = 4.0$, $p < .001$) compared to those who experienced low levels (B = 0.44; 95%CI: .30 to .58, $z = 6.4$, $p < .001$). In contrast, knowledge and disclosure did not moderate the strength of path b. More detailed results are presented in Table 6.

Furthermore, our results showed that none of the parental monitoring scales affected the link between depressive symptoms at age 14 and affiliation with deviant peers at age 18 (i.e., path a, Figure 1).

Discussion

Evidence shows that depression experienced in adolescence represents a risk factor for harmful drinking in adolescence as well as emerging adulthood (Saraceno et al. 2012; Marmorstein 2009); however, longitudinal research aiming to understand the underlying mechanisms that may explain this association is limited. Deviant peers represent one of the main risk factors for alcohol and substance use (Monahan et al. 2009; van den Bree and Pickworth 2005) while parental monitoring has been identified as a protective factor (Stattin and Kerr 2000). Nonetheless, the interplay between these two factors on the developmental association between depressive symptoms and harmful drinking is not clear as most research in the field has been cross-sectional (Bahr et al. 2005). Finally, research is not conclusive on which monitoring activity (disclosure vs. active monitoring, e.g., solicitation) is protective for deviant behavior (incl. alcohol use) (Fletcher et al. 2004; Stattin and Kerr 2000). Yet, understanding these processes would provide guidance for targeted interventions. Our study addressed this gap in the literature by exploring the interplay between monitoring and deviant peers on the longitudinal association between depressive symptoms experienced in adolescence and harmful drinking in emerging adulthood.

The results showed that depressive symptoms experienced at age 14 are associated with harmful drinking in emerging adulthood (age 19), when alcohol-related problems and dependence are most likely to develop (HSCIC 2013). These findings support existing literature that similarly observed a longitudinal relationship between depressive symptoms in adolescence and harmful drinking in early adulthood (Marmorstein 2009, 2010). These two studies, however, were based on wide age ranges (e.g., wave I: 11-21 years). Owing to the fact that the ALSPAC study design involved the recruitment of all participants born between 1991-92, the current study allows us to draw inferences about quite specific developmental stages. Moreover, we observed that the association between depressive symptoms and

harmful drinking was mostly mediated by affiliation with deviant peers at age 18. The results indicate that adolescents with depressive symptoms are more likely to affiliate with deviant peers and, subsequently, this increases their risk of harmful drinking in adulthood. These findings extend those from our previous study (Pesola et al. 2015) and indicate that this mechanism is not only present in adolescence but continues to be relevant in emerging adulthood.

The indirect influence of deviant peers was weakened but still present after adjusting the model for measures of deviant peers and harmful drinking at age 13. Thus, harmful drinking in emerging adulthood appears not to be solely explained by pre-existing conduct problems, alcohol use and peer networks. These results are in contrast with those from our previous study where we found that the indirect link between depressive symptoms at age 14 and harmful drinking at age 16 via deviant peers at age 15 was no longer present when the model was adjusted for alcohol use at age13 (Pesola et al. 2015). Thus, it seems that the link between depressive symptoms and harmful alcohol use in mid-adolescence is explained by alcohol use in early adolescence; however, the present results suggest that harmful drinking in emerging adulthood is influenced by socialization and not simply by pre-existing behavior. Thus, the two studies indicate that different mechanisms may operate across different developmental periods and highlight the need for separate examination of different ages. Based on our findings, it appears that adolescents with depressive symptoms, who may drink to self-medicate (Saraceno et al. 2009), may experience rejection by their normative peer group and, hence, affiliate with other individuals who share similar drinking patterns (i.e., self-selection). Our results are in line with students finding that young people who engage in harmful drinking tend to affiliate with peers who share similar drinking behaviors (Parra et al. 2007; Kendler et al. 2015).

Our results further indicated that, although parental monitoring may not be able to influence peer selection at age 18, parental control and solicitation (i.e., active monitoring) experienced during adolescence weakened the influence of peers on harmful drinking at age 19. Specifically, the association between affiliation with deviant peers at age 18 and harmful drinking at age 19 was stronger among young people who had reported poor parental control and low solicitation compared to those who had experienced high control and solicitation. Hence, active parental monitoring of adolescents, who experience depressive symptoms, has long-lasting effects which weaken the influence of deviant peers in emerging adulthood. Fletcher and colleagues (Fletcher et al. 2004) had similarly found that solicitation and control were associated with reduced involvement in deviant behavior among 14- to 18-year olds, although the evidence was cross-sectional and did not consider the interplay with peers.

A number of explanations may account for these results. Parents who implemented a controlling parenting style during adolescence may still exert a direct influence in emerging adulthood through continued parental involvement in the young adult's life. Alternatively, it is possible that the young adult has "internalized" the parents' views on deviant behavior and lives accordingly. Finally, it is possible that solicitation of information may reflect open dialogue between parents and offspring, which creates a warm relationship and can, thus, exert a continuing protective influence (Stattin and Kerr 2000). Additionally, our results

show that disclosure, which may capture a warm relationship with parents, has no long-term weakening effect on peer influence; this may be due to the fact that as individuals develop their independence during emerging adulthood (Doumen et al. 2012), they may be less likely to willingly disclose information to their parents. These interpretations, however, are speculative and future studies should aim to understand these mechanisms. Qualitative studies could help us to establish how active monitoring experienced in early adolescence achieves the long-lasting effects observed in emerging adulthood. Overall, results indicate that active monitoring is effective in the current age group of interest but replication is needed.

Our study contributes to the field exploring protective and risk for harmful drinking. Indeed, to our knowledge, this is the first study to explore the interplay between peers and parental monitoring on the developmental relationship between depressive symptoms in adolescence and harmful drinking in emerging adulthood. The strengths of the study include a large prospective cohort study design with a sample that is representative of the UK population as indicated by comparisons with the 1991 census and the 1970 Child Health and Education Study (Boyd et al. 2013; Golding et al. 2001). Hence, we can interpret the present results in relation to the general population rather than a clinical sub-population. A key strength of the study is the longitudinal design and the fact that the predictor, mediator and outcome measures were separated in time. This increases our understanding of the potential developmental mechanisms and avoids any potential estimate bias caused by cross-sectional and pseudo-longitudinal designs (Cole and Maxwell, 2003). A further strength of the study is the use of standardized measures that have been validated in adolescent age group. Finally, we were able to adjust our model for a range of variables that had been selected *a priori* based on existing literature and might have confounded our results.

There are also limitations. Following model adjustment, the total effect between depressive symptoms and harmful drinking was not present. Thus, it may be argued that we should have not proceeded to test the indirect pathway via peers. However, our approach is consistent with a growing literature that recommends that indirect mechanisms should be explored when researchers have a theoretically-driven rationale even when no total effect is detected (Shrout and Bolger 2002; Rucker et al. 2011). Based on the literature reviewed in the introduction and our previous study, we had strong theoretical grounds to proceed with our mediation analysis. The proportion mediation effect approach is generally used to quantify the effect size in mediation analyses; however, there is evidence that this estimate can be unstable when the direct effect is less than 0.2 or the sample is small (<500). These scenarios do not apply to our data and, hence, the proportion of the mediated effect was considered to offer a good estimate of the effect size. We should also acknowledge attrition, typical of longitudinal research (Young et al. 2006), as a limitation. In order to deal with missing data, we used multiple imputation as this technique reduces bias and increases power (White et al. 2011). We are confident in our approach as results conducted on the imputed data produced equivalent results to the ones obtained with complete cases (results are available on request) and, finally, rates of harmful drinking and depressive symptoms in our sample are consistent with figures from other UK studies (Thapar et al. 2012; Fuller 2012). As an additional limitation, we found a stronger relationship between deviant peers and alcohol use (1-year lag) than between depressive symptoms and deviant peers (4-year lag). This time-lag may

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explain the difference in the strength of these associations and is due to the timing of the assessment of each measure in the ALSPAC study. Future studies should aim to replicate the present findings while ensuring equivalent time-lags among measures.

An additional limitation is the fact that two items from the AUDIT and SMFQ were rare in the present sample and dropped from the analyses. These exclusions may prevent us from comparing our results to previous studies. However, inclusion of items with low variance would likely have impacted upon the predictive ability of our model and lead to bias. Furthermore, family history information (e.g., depression and alcohol use) used to adjust the model was collected at 33 months, as this information was not available for the age of interest. Circumstances might have changed over time and, hence, future cohort studies should collect this information throughout the study period; however, it is possible that family history has long-lasting effects. Moreover, we are interested in family histories of alcohol use rather than parents as role models. Peers' deviant behavior was not based on self-report but rated by the study participants who may over-estimate their friends' deviant behavior (Bekman et al. 2010). Future studies should include peers' self-report. A further limitation is that monitoring was reported by the adolescents rather than their parents or carers. However, perceived monitoring and parental reports of monitoring have been found to produce equivalent results (Li et al. 2000; Stattin and Kerr 2000). Additionally, parents' reports have been found to be biased with parents tending to report themselves as stricter than they are perceived (Bahr et al. 2005). Nonetheless, future studies may include estimates of monitoring behavior derived from parents' and adolescents' reports. Overall, shared method variance may be an issue as all measures were reported by the young people and this limitation should be addressed in future studies.

Conclusion

Our study contributes to the field of alcohol research prevention. By using a longitudinal design, we were able to explore a developmental mechanism that may lead to harmful drinking in emerging adulthood among individuals who experienced depressive symptoms in adolescence. Depressive symptoms experienced at age 14 were associated with harmful drinking five years later and, additionally, that 79% of this association was explained by affiliation with deviant peers at age 18. In contrast, active parental monitoring (i.e., control and solicitation) experienced during early adolescence was found to act as a protective factor for harmful drinking in emerging adulthood. Specifically, active monitoring weakened the impact of deviant peers on subsequent harmful drinking, although it did not reduce the likelihood of adolescents with depressive symptoms affiliating with deviant peers. Future studies should implement a mixed-method approach to replicate our findings and further elucidate the developmental processes that underlie the interplay between risk and protective factors on the development of harmful drinking in emerging adulthood.

Our study shows that risk and protective factors present in adolescence influence harmful drinking in emerging adulthood. Thus, our findings highlight the need to implement screening programs in early adolescence to identify individuals experiencing depressive symptoms because they represent a vulnerable group for harmful drinking. These programs should offer support for depressive symptoms as well as prevention for harmful drinking.

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Additionally, such programs should provide training to empower young people to cope effectively with the impact of deviant behavior by their peer group. Indeed, school-based programs should include peer resistance training and norm changing as they have been found to be effective at reducing harmful drinking (Faggiano et al. 2010). These programs should also provide parents with effective monitoring skills training, which can weaken the influence of deviant peers on drinking behavior. Moreover, evidence shows that parental skill training is a more amenable intervention than changing peer networks (Osgood et al. 2013a). Overall, our results suggest that multilevel prevention programs that address social influences and facilitate parental involvement may be beneficial to deter the development of harmful drinking in emerging adulthood among individuals who experienced depressive symptoms in adolescence.

Appendix Table 1: Items used to define each latent variable

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Latent variable	Item
Harmful drinking at age 19	How often do you have an alcoholic drink How many units do you normally drink on a typical day How often do you have six or more units How often were you unable to stop drinking How often have you failed to do what was expected of you because of drinking How often have you felt guilty after drinking How often have you been unable to remember events because of drinking Have you or someone else been injured because of your drinking
Depressive symptoms at age 14	I felt miserable or unhappy I didn't enjoy anything at all I felt so tired I just sat around and did nothing I was very restless I felt I was no good any more I cried a lot I found it hard to think properly or concentrate I hated myself I felt lonely I thought nobody really loved me I thought I could never be as good as other kids
Deviant peers at age 18	Using illegal drugs Displaying loud or rowdy behavior Physically attacking someone Shoplifting Damaging property
SDQ – Conduct Problems at age 13	Teenager often has temper tantrums Teenager is generally obedient Teenager often fights or bullies others Teenager often lies and cheats
Monitoring at age 14	Frequency carers know what teenager does in free time Frequency carers know what teenager spends money on Frequency past month, carers unaware where teenager was at night Frequency carers know who teenagers friends are, outside of school Frequency carers know where teenager goes when out with friends at night Frequency carers know type of homework teenager has Frequency carers know when teenager has exam/test at school Frequency carers know when teenager has exam/test at school Frequency carers know how teenager is doing in different subjects Frequency carers know where teenager goes and what they do after school
Disclosure at age 14	Frequency teenager keeps secrets from carers about what they do in free time Frequency teenager keeps things from carers about what they do nights/weekends Frequency teenager tells carers about what they did/where they were in the evening Frequency teenager tells carers about friends, without being asked Frequency teenager wants to tell carers about how they are doing at school
Solicitation at age 14	Frequency carers ask teenager what happened in free time Frequency past month carers started conversation about teenagers spare time Frequency carers take time to listen, when teenager talks about what happened in free time Frequency carers talk with teenagers friends, when come to teenagers house Frequency carers ask teenager what has happened at school, on normal school day
Control at age 14	Frequency teenager needs carers permission before they go out on week nights Frequency carers demand to know what teenager is going to do/who with/where before teenager goes out Frequency carers ask what teenager spends money on Frequency carers expect teenager to explain why, when have stayed out longer than allowed Frequency teenager has to ask carers, before they can make plans for a Saturday night
Deviant peers at age 13	Playing truancy Breaking something for fun Setting things on fire Stealing Getting into fights
Alcohol use at age 13	Number of drinks in the past 30 days Number of drinks in a typical week Frequency of having 3 or more drinks in 24 hours

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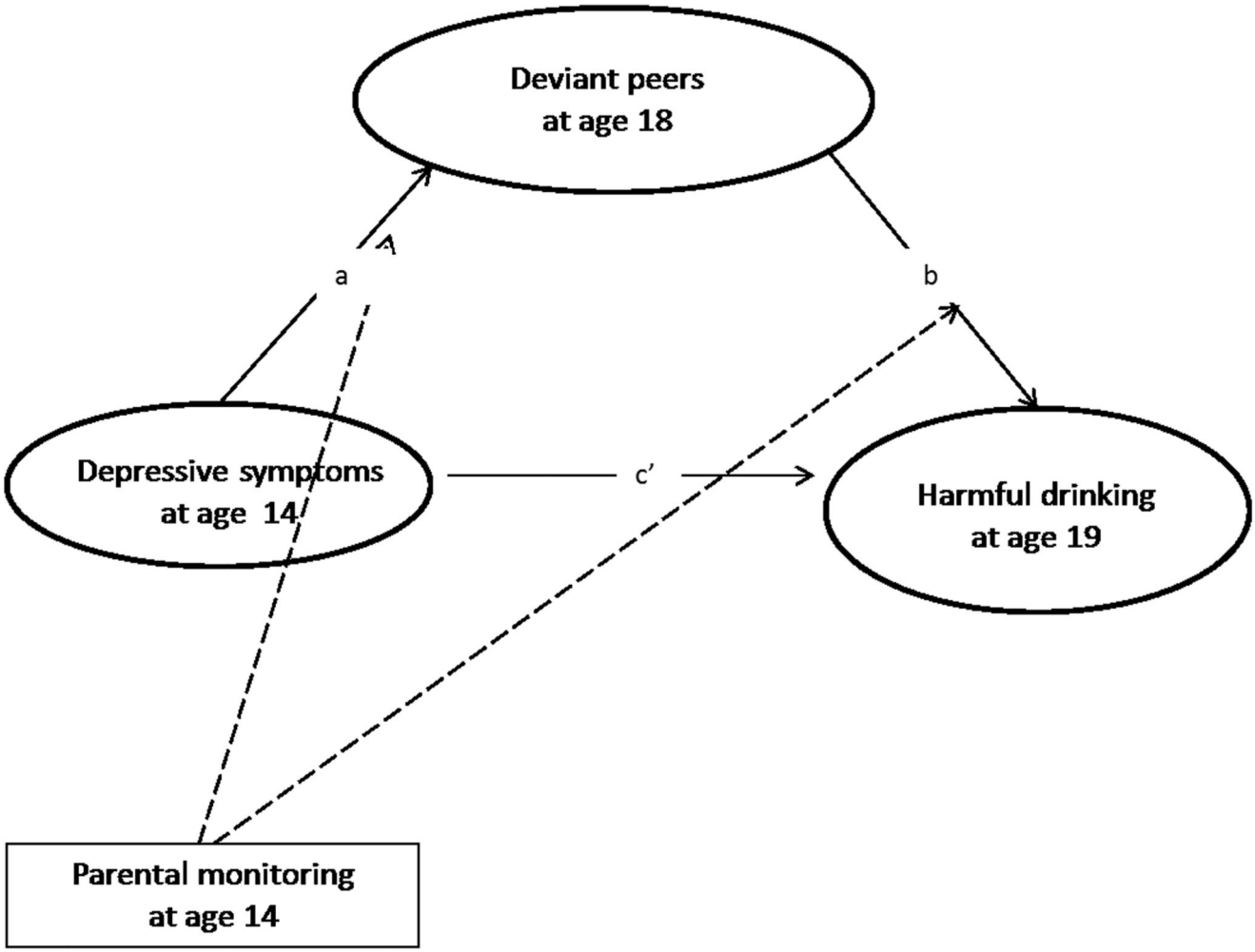

Figure 1.

Representation of the model which explores the predicting role of depressive symptoms on later harmful drinking via affiliation with deviant peers and the moderating role of parental monitoring on the indirect effect through peers.

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Table 1

Variables included in the imputation model and number of cases with complete data on these measures

Class	Variables	Measure	Age
Main variables	Harmful drinking	AUDIT	19y
	Depressive symptoms	SMFQ	14y
	Deviant peers	ESYTC	18y
	Monitoring	Parental Monitoring (PM)	14y
	Disclosure	PM	14y
	Solicitation	PM	14y
	Control	PM	14y
Covariates	Parental drinking	Family environment	145m
	Parental depression	Family environment	145m
	Financial difficulties	Socio-economic status	11y
	Parental education	Socio-economic status	32wk
	Conduct problems	SDQ	13y
	Problem drinking	SSAGA	13y
	Deviant peers	ESYTC	13y
Earlier measures	Depressive symptoms	SMFQ	10y
	Depressive symptoms	SMFQ	13y
	Harmful drinking	AUDIT	16y
	Parental depression	Family environment	21m
	Parental alcohol	Family environment	21m
	Conduct problems	SDQ	9y
	Financial difficulties	Socio-economic status	21m
	Deviant peers	ESYTC	15y
Auxiliary variables	Crowding index	Socio-economic status	21m
	Household income	Socio-economic status	33m
	Child smoking status		14y

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Table 2

Proportion of missing information across latent variables and covariates for cases with complete information on the outcome (n=2,964)

Latent component	% complete
Depressed mood	77%
Deviant peers	45%
Monitoring	75%
Disclosure	75%
Solicitation	75%
Control	75%
SDQ-Conduct problems	84%
Parents depression	93%
Parents alcohol use	93%
Family education level	99%
Financial difficulties	86%

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Table 3

Correlations, means and standard deviation (SD) for main variables. These figures are based on the scales' sum scores for cases with complete information on all measures (N = 976) and were calculated in Stata 11.

	1.AUDIT19	2.SMFQ14	3.Peers18	4.Peers13[§]	5.SDQ	6.Alcohol13	7.Knowledge	8.Disclose	9.Solicit	10.Control
1	1.00									
2	0.11**	1.00								
3	0.39**	0.12**	1.00							
4	0.17*	0.16**	0.21**	1.00						
5	-0.02^{ns}	0.11**	-0.04^{ns}	0.07*	1.00					
6	0.18**	0.17**	0.13**	0.32**	-0.01^{ns}	1				
7	-0.27**	-0.29**	-0.25**	-0.26**	-0.01^{ns}	-0.24**	1.00			
8	-0.29**	-0.29**	-0.28**	-0.24**	-0.03^{ns}	-0.25**	0.71**	1.00		
9	-0.04^{ns}	-0.10*	-0.09*	-0.13**	0.01^{ns}	-0.13**	0.42**	0.41**	1.00	
10	-0.17**	-0.03^{ns}	-0.19**	-0.20**	0.02^{ns}	-0.19**	0.48**	0.38**	0.44**	1.00
Mea	**9.2**	**4.9**	**2.2**	**0.8**	**2.2**	**-0.003**	**28.1**	**13.5**	**13.7**	**14.2**
SD	***5.4***	***4.5***	***1.5***	***1.1***	***0.8***	***1.1***	***5.8***	***3.4***	***2.8***	***3.6***

Note:

ns = not significant, *p < .05, **p<.001

[ß] main variables in our model

[§] confounder variables used to adjust our model

[¥] scales of the monitoring questionnaire

[#] Following the approach followed by Saraceno and colleagues (2010), harmful drinking at age 13 was constructed using a Principal Component Analysis (PCA) approach. PCA extrapolates components with mean of 0 and standard deviation of 1

AUDIT: Alcohol Use Disorder Identification Test

SMFQ: Short Mood and Feeling Questionnaire

SDQ: Strengths and Difficulties Questionnaire – conduct problem subscale

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Table 4

Imputed data analyses: Total, indirect and direct effects of depressive symptoms (i.e., predictor) on harmful drinking (i.e., outcome) via deviant peers (i.e., mediator) [95% CI] (N = 2964)

	Step 0	Step 1	Step 2	Step 3	Step 4
Total Effect (depressed mood to alcohol use)	.053 [.014, .092]	.065 [.020, .111]	.049 [0, .098]	.027 [-.022, .076]	.024 [-.025, .073]
Indirect effect (relationship via deviant peers)	.048 [.028, .068]	.057 [.037, .077]	.051 [.029, .073]	.034 [.014, .054]	.033 [.013, .053]
Direct effect (relationship adjusted for deviant peers)	.005 [-.034, .044]	.008 [-.039, .055]	-.002 [-.051, .047]	-.007 [-.056, .042]	-.009 [-.039, .059]
Effect proportion mediated (indirect/total ratio)	91%	87%	96%	83%	79%

Step 0: Unadjusted for confounders

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

Step 2: Adjusted for background confounders + SDQ conduct problems

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

Step 4: Adjusted for background confounders + SDQ conduct problems + earlier mediator at age 13 (i.e., deviant peers) + earlier outcome at 13 (i.e., alcohol use)

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Table 5

Imputed data - measure invariance across monitoring scales

	Measurement invariance				
	Models	**χ^2 (df)**	**RMSEA (95%CI)**	**CFI**	**TLI**
Monitoring	*Unconstrained*	1526.7 (498)	.037 (.035, .039)	.963	.959
	Constrained	1581.8 (535)	.036 (.034, .038)	.963	.961
Control	*Unconstrained*	1609.2 (498)	.039 (.037, .041)	.961	.958
	Constrained	1661.7 (535)	.038 (.036, .040)	.962	.961
Disclosure	*Unconstrained*	1526.2 (498)	.037 (.035, .039)	.963	.959
	Constrained	1603.7 (535)	.037 (.035, .039)	.962	.961
Soliciting	*Unconstrained*	1585.3 (498)	.038 (.036, .040)	.963	.959
	Constrained	1629.0 (535)	.037 (.035, .039)	.963	.962

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Table 6

Imputed data: Multiple group comparisons of the strength of the indirect effect via peers at age 18 between adolescents who reported low vs. high parental monitoring. All analyses are adjusted for background variables, SDQ conduct problems, alcohol use and deviant peers at age 13

	Monitoring		Disclosure		Soliciting		Control	
	Estimate (se)	**Wald**	**Estimate (se)**	**Wald**	**Estimate (se)**	**Wald**	**Estimate (se)**	**Wald**
Path a								
Low	.08 (.01, .16)	0.3,p=.98	.07 (-.07, .21)	-0.5,p=.64	.10 (.01, .20)	0.3, p= .79	.13 (.05, .21)	1.2, p = .22
High	.07 (.01, .15)		.10 (.01, .20)		.09 (.01, .19)		.06 (-.02, .14)	
Path b								
Low	.42 (.28, .56)	1.9, p=.06	.39 (.25, .53)	0.8, p=.45	.44 (.30, .58)	2.8,p=.005	.46 (.32, .60)	2.4,p =.006
High	.26 (.12, .40)		.33 (.19, .47)		.23 (.11, .35)		.23 (.11, .35)	