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Childhood Cumulative Contextual Risk and Depression Diagnosis Among Young Adults: The Mediating Roles of Adolescent Alcohol Use and Perceived Social Support

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Abstract

This study examined associations between cumulative contextual risk in childhood and depression diagnosis in early adulthood, testing two adolescent mediating mechanisms, alcohol use and perceived social support from family and friends, while accounting for the stability of internalizing problems over time and examining possible gender moderation. Multiple group mediation analyses were conducted using parent- and adolescent-report as well as hospital records data from the Northern Finland Birth Cohort 1986 ($N = 6,963$). Our analyses demonstrated that the association between cumulative contextual risk in childhood and depression diagnosis in adulthood is mediated by adolescent alcohol use and perceived social support both for boys and girls. The findings highlight potentially malleable mediating mechanisms associated with depression in vulnerable youth that could be targets in selective depression preventive interventions.

Keywords

cumulative contextual risk; adolescence; adulthood; alcohol use; social support; depression; gender

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Introduction

Research has documented an increased prevalence of depression among individuals exposed to contextual risk factors in childhood (Duncan, Brooks-Gunn, & Klebanov, 1994; Elovainio et al., 2012; Mossakowski, Codescu, Neuhaus, & Kutz, 2013), particularly when those risks are cumulative (e.g., Gerard & Buehler, 2004). Although it is well documented that the sheer number of contextual risk factors in a child's life increases the likelihood of adverse outcomes (Evans, Li, & Whipple, 2013), the degree to which the association of cumulative contextual risk in childhood with depression diagnosis in adulthood is mediated by potentially malleable risk and protective processes in adolescence has received less attention. One line of evidence suggests that a risk factor for depression is adolescent substance use, including alcohol (Mason et al., 2008; Trim, Meehan, King, & Chassin, 2007), representing a possible *risk pathway*. Another line of evidence suggests that a diverse and supportive social network may reduce risk for depression (Platt, Keyes, & Koenen, 2014), representing a possible *protective pathway*. The current study addresses key gaps by using longitudinal survey data from the Northern Finland Birth Cohort 1986 (NFBC1986) to examine associations between cumulative contextual risk in childhood and depression diagnosis in early adulthood, testing two adolescent mediating mechanisms, alcohol use and perceived social support from friends and family, while accounting for the stability of internalizing symptoms over time and examining possible gender moderation.

Past research has demonstrated positive associations between exposure to cumulative contextual risks in childhood and depression symptoms later in life. According to *social causation theory*, exposure to cumulative contextual risks is associated with a higher number of stressors and fewer coping mechanisms, which in turn lead to depressive symptoms (Pearlin, 1989; Pearlin, Menaghan, Lieberman, & Mullan, 1981). Guided by social causation theory, several studies have investigated the associations between contextual risk factors in childhood and depressive symptoms in adolescence and adulthood. For example, Elovainio et al. (2013) examined associations between socioeconomic status and the developmental trajectory of depressive symptoms from childhood through adulthood. The results provided evidence that lower socio-economic status as well as negative emotionality in childhood were associated with higher risk of depressive symptoms in adolescence. Also, higher initial levels of depression were recorded among girls, and were associated with a slower decrease in depressive symptoms in early adulthood. Similarly, Mossakowski et al. (2013) examined the effect of poverty on depression in early adulthood using data from the National Longitudinal Survey of Youth (NLSY). The results suggested that chronic exposure to poverty as well as female gender and prior mental health issues were predictive of depressive symptoms in adulthood. Of note, findings from different samples across different countries report a similar pattern of gender differences indicating a higher prevalence of depression in girls than boys (Van de Velde, Bracke, & Levecque, 2010). Other studies have documented associations between different markers of contextual risks (e.g., socioeconomic adversity, socioeconomic disadvantage, low socioeconomic status) in childhood and the lifetime risk of depression (Culpin, Stapinski, Miles, Araya, & Joinson, 2015; Gilman, Kawachi, Fitzmaurice, & Buka, 2002; Goosby, 2013; Jackson & Goodman, 2011; McLaughlin et al., 2011; Najman et al., 2010). Taken together, the evidence highlights the

need for examining potential mediating mechanisms that can represent developmental pathways leading from cumulative contextual risks in childhood to depression diagnosis in early adulthood. Adolescence may be an important period to examine such associations given that the total incidence rate of depression doubles in adolescence compared to childhood, and almost twice as many girls than boys become depressed (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998).

Alcohol Use and Supportive Social Network: Possible Mediators

There is a need for examinations of the potentially malleable risk and protective processes in adolescence that might mediate the association of childhood cumulative contextual risk with subsequent depression in young adulthood. In an extensive literature review, Zhou and colleagues (2014) indicated that depression and substance use disorders are the most common mental illnesses in adolescence. Indeed, alcohol use, which typically emerges and increases during the teen years (Miech, Johnston, O'Malley, Bachman, & Schulenberg, 2016), often co-occurs with depressive symptoms and depression diagnoses among young people (Costello, Erkanli, Federman, & Angold, 1999; Mason, Chmelka, Howard, & Thompson, 2013). Thus, adolescent alcohol use might represent a *risk pathway* leading from childhood cumulative risk to young adult depression. Although it is well documented that depression and alcohol use co-occur among youth, the nature of this association remains unclear. According to the *self-medication hypothesis* (Khantzian, 1997), individuals use substances, including alcohol, to alleviate the symptoms associated with mental illnesses, such as depression, suggesting that depression precedes and predicts alcohol involvement. There is at least some support for the self-medication hypothesis, particularly among adults (Jerez-Roig et al., 2014; Robinson, Sareen, Cox, & Bolton, 2009; Swendsen et al., 2000). However, among adolescents, research tends to show the reverse, that alcohol use typically precedes and predicts depression (Mason et al., 2008; Trim et al., 2007; Miettunen et al., 2014), although the reasons for this association are uncertain (e.g., pharmacological effects of alcohol; alcohol leading to failures in psychosocial functioning that lead, in turn, to depression) and exceptions to this pattern do exist (Mason et al., 2009; Wymbs et al., 2014). Prior research also has documented positive associations between cumulative contextual risk and adolescent alcohol use. For example, in an analysis of data from the NFBC1986, cumulative contextual risk in early childhood was a positive predictor of alcohol and other substance use in mid-adolescence (Mason et al., 2016; January et al., 2016). Taken together, findings from prior research suggest that childhood cumulative contextual risk might be related to subsequent depression through adolescent alcohol use, although this remains to be tested.

Additionally, researchers working along the lines of *the social causation theory* proposed that a supportive social network of family and friends is one of the key coping sources for adolescents struggling with alcohol use and mental health disorders (Ary, Duncan, Duncan, & Hops, 1999; Matlin, Molock, & Tebes, 2011; McFarlane, Bellissimo, & Norman, 1995). Thus, supportive social networks in adolescence might represent an additional *protective pathway* from cumulative risk in childhood to depression diagnosis in adulthood. From the perspective of *social support theory*, social support is the perception that one has available assistance from other people or is a part of the supportive social network (Pearlin, 1989).

Social support can be measured quantitatively (e.g. the size and type of the one's social network) and qualitatively (e.g. one's perception of available social support) (Platt et al., 2014). However the literature is not clear as to what extent different sources of support in adolescence contribute to developmental outcomes. While one line of evidence suggests that family support, such as participation in family routines and eating dinner together, is associated with lower rates of depressive symptoms in adolescents (Eisenberg, Olson, Neumark-Sztainer, Story, & Bearinger, 2004; Fulkerson et al., 2006; Goldfarb, Tarver, Locher, Preskitt, & Sen, 2015; Neumark-Sztainer, Larson, Fulkerson, Eisenberg, & Story, 2010); other emphasize the importance of positive peer relationships and close friendships (Sullivan, 1953; Brendgen et al., 2013; Choukas-Bradley & Prinstein, 2014). Given that sources of social support among adolescents can vary considerably due to the quality of support they provide, it has been suggested that a *diverse social network*—one that captures social support across different groups - is potentially more protective against depressive symptoms than the perceived availability of social support within one group (Platt et al., 2014). However the literature in this area is scant. Our study will address this gap by examining the protective role of combined social support from the social network of friends and family in associations between cumulative contextual risk and depression.

Of course, one of the major contributing factors to depression diagnosis in adulthood is previous history of internalizing problems. Past research established predictive links between early internalizing symptoms, such as inhibited fearful behavior, anxiety, and worries, and internalizing problems in adolescence and adulthood (Rubin & Coplan, 2010). Longitudinal studies revealed that prolonged, persistent experiences of internalizing problems throughout childhood are strongly associated with depression symptoms in adolescence (See Zahn-Waxler, Klimes-Dougan, & Slattery, 2000 for extensive review). It has been suggested that repetitive experiences of negative emotions, such as fear, anxiety, or sadness, may lead to the development of certain patterns of responding to social events that sets up a stage for the development of depression. For these reasons, it is important to control for previous history of internalizing problems when examining the origins or developmental trajectory of depression in samples of adolescents.

Taken together, these findings suggest that developmental pathways to depression may begin in early childhood with exposure to cumulative contextual risks that set the stage for susceptibility to depression later in life. The association between cumulative contextual risk and depression may be mediated by adolescent alcohol use (a risk factor) or perceived social support from family and friends (a protective factor). The possible moderating influence of gender also is an important consideration. Although the associations between cumulative contextual risk and depression are well documented, less is known about whether or not these associations and hypothesized developmental pathways are moderated by gender. The vulnerability-stress model (Cyranowski, Frank, Young, & Shear, 2000) specifically suggests that girls are more vulnerable to depression compared to boys because of their higher affiliation needs, which places them at a higher risk for depression in the presence of low levels of support (Kendler, Myers, & Prescott, 2005). While these findings do not explain all gender differences in the prevalence of depression and depressive symptoms, they do suggest potential gender differences in pathways to depression diagnosis, especially through the social support.

Purpose of This Study

The purpose of this study was to extend the current research literature by examining associations between cumulative contextual risk in childhood and depression diagnosis in early adulthood, testing two adolescent mediating mechanisms, alcohol use (a risk pathway) and perceived social support from friends and family (a protective pathway), while accounting for the stability of internalizing symptoms over time and examining possible gender moderation. More specifically, in our study we tested two main hypotheses:

1. First, we hypothesized that alcohol use and perceived social support from family and friends would mediate the association of cumulative contextual risk in childhood with subsequent depression diagnosis in early adulthood. As noted above, while previous studies reported positive associations between alcohol use and depression, and negative associations between perceived social support and depression, the joint processes by which these two mediating mechanisms operate *together* are not yet well understood.
2. Second, we hypothesized that the pathways from cumulative contextual risk to depression diagnosis through alcohol use and social support would differ by gender. Because it has been proposed that females are more sensitive to low levels of social support, we expected that the mediating pathway from cumulative contextual risk to depression diagnosis through social support would be stronger for adolescent girls than boys. In terms of alcohol use, there is inconclusive evidence for gender moderation in either the environmental risk factors for or the psychological consequences of adolescent alcohol use. Thus, our examination of potential gender differences in mediating pathways through alcohol use was exploratory.

Method

Participants

Data came from the Northern Finland Birth Cohort 1986 (NFBC1986) study on health and well-being (with the original purpose of studying the emergence of diseases that can be based on genetic, biological, social or behavioral risk factors). For the current study, the data were drawn from a prenatal survey of mothers, a health survey at age 7 (parent reports), a learning disability survey at age 8 (teacher reports), adolescent survey of health and well-being at age 16 (adolescent self-reports), and the Finnish Hospital Discharge Register data.

Of the 9,479 initially recruited NFBC1986 participants, 8,755 provided a consent form to use the data for research, and 6,963 had data on cumulative contextual risks. For a case to be included in the statistical analysis, data on a major predictor variable (e.g., cumulative contextual risk) is required. Thus, the analysis sample consisted of 6,963 participants (73% of the original birth cohort). Forty-nine percent of the participants in the analysis sample were male, with the mean age of 16.0 at the time of adolescent data collection, ranging from 14.58 to 16.96. Additional details regarding the NFBC1986 data collection are available elsewhere (Hurtig et al., 2007; Järvelin, Hartikainen-Sorri, & Rantakallio, 1993).

Measures

Cumulative Contextual Risk—The prevalent approach to measuring cumulative risk is to construct an index by adding together multiple dichotomous risks affecting family life (Evans et al., 2013). The cumulative risk approach proposes that accounting for the *number* of risk factors improves validity of the cumulative risk measure (Sameroff, 1979), because family risk factors often co-occur with one another. Therefore, consistent with the current literature, the cumulative contextual risk index was created by summing scores from eight dichotomous parent-reported risk factors that prior research has shown are associated with depressive symptoms in adolescence and adulthood (socio-economic disadvantage; Elovainio et al., 2013; Mossakowski et al., 2013; single parent, low maternal education, maternal alcohol use, Horan & Widom, 2015). These risk factors represent maternal characteristics while pregnant (e.g., teenage mother, smoking while pregnant, drinking while pregnant), family's socioeconomic disadvantage at age 7 (e.g., unemployed mother, unemployed father, less than 9 years of comprehensive school for mother, less than 9 years of comprehensive school for father), and family structure at age 7 (e.g., single parenthood). Each indicator was coded 1 to indicate the presence of risk or 0 to indicate the absence of this risk factor, as described below.

Teenage mother was coded 1 if the mother gave birth to the participating child at age 19 years or younger. *Smoking while pregnant* was coded 1 if the mother smoked cigarettes after the first trimester of pregnancy. *Drinking while pregnant* was coded 1 if the mother consumed alcohol at any time during pregnancy. *Unemployed mother/father* was coded 1 if the mother/father self-identified as “unemployed, receives benefits” in a health survey at child age 7. *Less than 9 years of comprehensive school for mother/father* was coded 1 if the mother/father self-reported less than 9 years of comprehensive school in a health survey at child age 7. *Single parent* was coded 1 if the main caregiver self-reported being a single parent in a health survey at child age 7.

Anxious-fearful behavior—Anxious-fearful behavior in childhood (at age 8) was measured with the Rutter Children Behavior Questionnaire for teachers, a widely used measure for determining children's behavioral and emotional problems (RCBQ; Elander & Rutter, 1996; Rutter, 1967). Teachers were asked to rate how well each item described child behavior on a 3-point Likert-type scale: 0 (does not apply), 1 (applies somewhat), or 2 (certainly applies). For this study we used the *anxious-fearful subscale* (8 items, $\alpha = .75$) that measures children's displays of anxiety, fear, sadness and worries (e.g. “Is often worried”). Higher scores indicate greater problems.

Internalizing problems—Internalizing problems in adolescence (at age 16) were measured with the Achenbach Youth Self Report (Achenbach & Rescorla, 2001). Youth were asked to rate how well each item applies to their behavior on a 3-point Likert-type scale: 0 (not at all true), 1 (somewhat true), 2 (very true or often true). For the current study we used the Internalizing scale, which is created as a sum of withdrawn (7 items), somatic complaints (9 items), and anxious/depressed subscales (15 items). The version available to us had one question omitted (e.g. “I feel sad”), so the internalizing scale consisted of 31 items (e.g. compared to the 32 items of the full scale) and represented a broad range of

internalizing problems (e.g. “Unhappy or depressed”). Higher scores indicate greater problems ($\alpha = .88$).

Alcohol use—Alcohol use in adolescence (at age 16) was based on three items from adolescent self-report survey referring to the *frequency* (How many times during the past 12 months have you had at least one drink of alcohol?), *intensity* (How many times in the past 12 months have you been drunk?), and *heavy episodic drinking* (Alcohol heavy episodic drinking past 30 days), measured on the 7-point scale ($\alpha = .91$).

Perceived social support—Perceived social support was based on five items from the adolescent self-report survey (administered at age 15). Three items referred to the *frequency of socialization* with friends and family with the response options ranging from “hardly ever” to “daily” 1) How often do you meet friends; 2) How often do you spend time with family?; 3) How often do you have meals with your parents/other family members? Two items referred to *perceived social support*: 4) Do you have a close friend with whom you can confidentially discuss your matters? (ranged from “I have no close friends” to “I have several close friends”); and 5) Are your parents interested in your school, hobbies, and other things you consider important? (ranged from “never” to “almost always”) ($\alpha = .43$).

Depression diagnosis—Depression diagnosis was obtained from the Finnish Hospital Discharge Register data that contains official medical records of diseases and related health problems through the end of year 2013 (approximate age 28). Depression diagnoses were recorded using the International Classification of Diseases (ICD): ICD-9 during 1987-1995, and ICD-10 since 1996. The official hospital discharge register data had the dates of the first depression diagnosis for every participant, and were dichotomized to indicate the presence or absence of depression diagnosis.

Analyses

Mediation analyses were conducted with the full analysis sample ($N = 6,963$) using multivariate path analysis in Mplus 7.11 (Muthén & Muthén, 1998 - 2010) with the weighted least squares mean- and variance (WLSMV) adjusted estimator due to the dichotomous nature of the depression diagnosis outcome variable. The WLSMV estimator does not require dependent variables to be normally distributed, and provides the best estimation for models with categorical or ordered outcomes, and works well in models with a large number of factors (Muthen & Muthen, 1998-2010). In Mplus, pairwise deletion is used with categorical outcomes estimated with the WLSMV estimator. There were no missing data on a primary predictor (i.e., cumulative contextual risk), primary outcome (i.e., depression diagnosis) or gender. The missing data on the other variables contributing to the model (i.e., alcohol use, perceived social support, and internalizing problems in adolescence; anxious-fearful behavior in childhood) was low, ranging from 6 % to 11%.

Attrition analyses showed that the analysis sample had participants with slightly higher cumulative risk ($M = 0.77$, $SD = 0.97$) compared to the birth cohort ($M = 0.74$, $SD = 0.96$), $t(16,440) = 1.97$, $p = 0.048$, even though this difference was small (Cohen's $D = 0.03$). The analysis sample also had children with lower ratings of anxious-fearful behavior at age 8 (M

= 1.03, $SD = 1.74$) compared to the birth cohort ($M = 1.11$, $SD = 1.83$), $t(14,966) = 2.72$, $p = .006$); this difference also was small (Cohen's $D = 0.05$). The analysis sample did not differ from a birth cohort in the rates of depression diagnosis (7.1 % v. 7.4 % $X^2(1, N = 16,442) = 0.78$, $p = 0.378$), gender ratio (51% v. 51.6% female, $X^2(1, N = 16,442) = 1.11$, $p = 0.29$), ratings of internalizing problems at age 16 ($t(13,339) = 0.16$, $p = 0.87$), adolescent alcohol use ($t(13,498) = 0$, $p = 1.00$), and perceived social support ($t(13,498) = 0$, $p = 1.00$).

A fully saturated path model with observed variables was estimated. This model was just identified and, therefore, had perfect fit (Chi-square = 0, Degrees of Freedom = 0); thus, similar to a regression analysis, the focus was on evaluating the statistical significance of the path coefficients and indirect effects, and testing possible gender differences. To examine mediation, bias-corrected bootstrapped 95% confidence intervals were computed to determine the statistical significance of the indirect effects generated by Mplus based on 5,000 bootstrap samples (MacKinnon, Lockwood, & Williams, 2004). Bootstrapping uses computer intensive resampling from the original sample to obtain more accurate estimates of the confidence intervals. Confidence intervals that do not include zero are statistically significant.

Multiple group structural equation modeling was further used to test possible gender differences in all of the path coefficients, with particular attention to the indirect effects from cumulative contextual risk to depression diagnosis. The DIFFTEST command in Mplus was used to test for group differences in the path coefficients via series of parameter constraints. Specifically, each path was tested in turn, forcing the parameter estimate to take on the same value for males and females and comparing model fit with that from a model in which the same parameter was allowed to differ across groups. A statistically significant chi-square difference test indicates a group difference. The indirect effects from cumulative contextual risk to depression diagnosis for females and males were evaluated for significance using bootstrapped confidence intervals. Further, in order to evaluate if the indirect effects for females and males were statistically different from each other, the NEW parameter option in the MODEL CONSTRAINT command in Mplus was used to create two new parameters: a) parameter 1 was calculated as the difference in indirect effects from cumulative contextual risk to depression diagnosis *through alcohol use* between females and males, and b) parameter 2 was calculated as the difference in indirect effects from cumulative contextual risk to depression diagnosis *through the perceived social support* between females and males. Then the new parameters were evaluated for significance using bootstrapped confidence intervals.

Results

Descriptive Statistics

Table 1 presents descriptive information about the study variables (mean, range, and standard deviation) for females and males. It is notable that depression diagnosis rates in females are approximately twice as high as in males (e.g., 9.3 % and 4.8% respectively). Also, overall, the sample has low rates of anxious - fearful behavior at age 8 and internalizing problems at age 16. Estimated correlations between study constructs are reported in Table 2. As expected, cumulative contextual risk is positively associated with

depression diagnosis, alcohol use, and anxious-fearful behavior, and negatively associated with perceived social support from family and friends.

Multiple Group Gender Moderation Analyses

Analyses tested gender moderation by conducting a two-group model test treating gender as a grouping variable. The chi-square difference test between the two models was non-significant ($\chi^2 = 14.47$, $df = 11$, $p = .21$), indicating no significant differences between males and females for any of the paths in the model. Next, the focus of the analyses was on identifying gender differences in the indirect effects from cumulative contextual risk on depression diagnosis through alcohol use and perceived social support. These analyses also did not reveal any significant gender differences, thus reported below are analyses with the full sample.

Mediation Analyses

Figure 1 presents the path analysis results for the full sample with unstandardized and standardized parameter estimates. Although not depicted in the figure, gender was included as a covariate, with paths leading to each mediator and outcome measure. Also not depicted are covariances among exogenous predictors and among the residual variances of the mediators. Cumulative contextual risk in childhood was significantly positively associated with the adolescents' alcohol use ($\beta = .13$, $p < .001$) and negatively associated with the perceived social support ($\beta = -.06$, $p < .001$). However the associations between the cumulative contextual risk and internalizing problems in adolescence ($\beta = .01$, $p = .519$) or depression diagnosis ($\beta = .04$, $p = .052$) were not significant. Further, indirect effects from the cumulative contextual risk to depression diagnosis through perceived social support ($b = .01$ [.01, .02]) and alcohol use ($b = .02$ [.01, .03]) were significant (see Table 3). Although not the focus of analyses, anxious-fearful behavior in childhood was positively associated with internalizing problems in adolescence ($\beta = .06$, $p < .001$), which in turn were associated with the lifetime depression diagnosis ($\beta = .19$, $p < .001$). Coefficients for gender in the model show that being female increases risk for internalizing problems in adolescence ($\beta = -5.07$, $p < .001$) and depression diagnosis over the lifetime ($\beta = -.20$, $p < .001$). Overall, the general pattern of results indicates the presence of risk and protective pathways from cumulative contextual risk to depression diagnosis. First, the analyses of the hypothesized risk pathway in our study provided evidence that exposure to cumulative contextual risks in childhood is associated with higher alcohol use in adolescence, which is in turn associated with higher risk of depression diagnosis in adulthood. Second, the analyses of the hypothesized protective pathway through the perceived social support indicated that cumulative contextual risk in childhood is negatively associated with the perceived social support in adolescence, possibly attenuating the protective role of perceived social support in depression diagnosis.

Discussion

Although childhood cumulative contextual risk has been shown to predict later depression in adolescents and adults (Duncan et al., 1994; Elovainio et al., 2012; Mossakowski et al., 2013), the potentially pliable mediating mechanisms involved in this long-term association

remain understudied and unknown. Previous research indicated that one possible risk factor for depression is adolescent alcohol use (Mason et al., 2008; Trim et al., 2007), thus suggesting a possible *risk pathway*. Research has also established that a diverse and supportive social network may reduce risk for depression, suggesting a possible *protective pathway* (Platt et al., 2014). Our study was designed to address two questions. First, do alcohol use and social support in adolescence mediate the association between childhood cumulative contextual risk and adult depression diagnosis? Second, do the mediating pathways from cumulative contextual risk to depression diagnosis through alcohol use and social support differ by gender? Our analyses revealed that the indirect effects of cumulative contextual risk on depression diagnosis through alcohol use and perceived social support were significant and did not differ by gender, confirming the hypothesized risk and protective pathways respectively.

Findings from the hypothesized risk pathway supported the hypothesis that adolescent alcohol use would serve as a mediator linking childhood cumulative contextual risk with adult depression diagnosis. Prior research (Hawkins, Catalano, & Miller, 1992), including prior analyses with this sample (January et al., 2016; Mason et al., 2016) have demonstrated a link between cumulative risk exposure and later alcohol involvement. This link could be due to either genetic (e.g., family history of alcohol abuse) or environmental (e.g., parental modeling) influences, or a combination of both. In turn, adolescent alcohol use has been shown to precede and predict later depressive symptoms and depression diagnosis (Mason et al., 2008; Trim et al., 2007; Miettunen et al., 2014). To our knowledge, this is the first study to test and find support for the full mediational chain leading from child cumulative risk to adult depression through adolescent alcohol use. The fact that this pathway was comparable for boys and girls is consistent with research showing both a closing gender gap in levels of alcohol use and little consistent evidence for gender moderation in the environmental causes and psychological consequences of adolescent alcohol use (e.g., Keyes, Grant, & Hasin, 2008).

Findings from the hypothesized protective pathway indicated that cumulative contextual risk in childhood is negatively associated with the perceived social support in adolescence, possibly contributing to the weaker protective effect of perceived social support in depression diagnosis. The *social support theory* that guided our analyses emphasizes social support from parents and peers as a one of the key coping mechanisms for at-risk adolescents struggling with alcohol use and mental health disorders (Ary, Duncan, Duncan, & Hops, 1999; Matlin, Molock, & Tebes, 2011; McFarlane, Bellissimo, & Norman, 1995). The lack of significant gender differences in this mediating pathway was somewhat non expected, because in accordance with the *vulnerability-stress model* (Cyranowski et al., 2000) we originally hypothesized that in the presence of low levels of social support adolescent girls would face higher risks for depression compared to boys, because of their higher needs for affiliation. A potential explanation for the non-significant gender differences in the indirect effect through perceived social support may come from the rather limited (e.g., consisting of five items) measurement of social support available to us in the current dataset. It has been shown that studies reporting significant gender differences in the mean levels of perceived social support in adolescence tend to utilize multiple measures that capture several types of social support (e.g., emotional, appraisal, informational, and

instrumental) in each domain of adolescent life (see Rueger, Malecki, & Demaray (2010) for a full review). Thus, whether or not the associations between cumulative contextual risk and social support are moderated by gender requires further investigation.

Limitations

It is important to recognize the study's limitations. Thus, due to the secondary data source, we had certain measurement limitations, including a limited number of items to test our research questions. Cronbach's alpha for the perceived social support scale was relatively low ($\alpha = .43$), which might be due to the small number of relevant items. The small number of available items did not allow us to create a multidimensional construct of perceived social support. It has been speculated that low reliability in the mediating variable may lead to underestimation of the mediation effect in analyses with the observed variables (particularly in small samples, which does not apply here) (Hoyle & Kenny, 1999). In addition, observed variables include measurement error and other variance, not related to the "true" score. By contrast, latent variables are free of measurement error, which may attenuate the estimation of standardized regression coefficients in path analyses with unreliable observed variables (Raykov & Marcoulides, 2012). For this reason, it is generally advised to use latent variable approaches, when possible, to account for measurement unreliability. So, it could be useful in future studies to use latent variables of the hypothesized model as well as include additional measures of perceived social support from other sources, such as school, community, religion, etc.

Further, the fact that our findings are based on the data from Finland can be interpreted as both a strength and limitation. Our primary predictor, cumulative contextual risk, may have different implications in Finland, compared to the U.S. and other country contexts. In fact, 51% of our sample had zero contextual risks, and 29% of the sample had indicated only one risk – so, overall, approximately 80% of the sample can be classified as low risk. The welfare model, adopted by the Nordic countries, including Finland, provides means of social support equally to all people in need, including access to health care, education, and family support services (e.g., Nordic Center for Welfare and Social Issues, 2016). For these reasons, the differences in the social classes in Nordic states are not as dramatic, compared to the U.S. The profiles of families with multiple contextual risks in Nordic states might differ compared to those in the western countries. Nonetheless, using data from Finland allowed us to rely on the comprehensive data collection on a birth cohort to assess pathways to depression symptoms using data from multiple informants and developmental periods (e.g., teacher reports in childhood, self-reports in adolescence, and register data in adulthood).

Future Directions

The current study made an important step in examining the influence of adolescent alcohol use and perceived social support in the association between cumulative contextual risk in childhood and depression diagnosis in adulthood. Research with the more established measures of social support (e.g., the Child and Adolescent Social Support Scale; Malecki, Demaray, Elliott, & Nolt, 1999) might help to unfold gender differences in associations between the perceived social support and depression diagnosis. Research is also needed to examine other relevant contexts that may provide potential sources of support in adolescence

(e.g., peer relationship). In addition, future research should pursue examination of additional, potentially gender sensitive mediating mechanisms that place youth in more disadvantaged position in developing of depression (e.g., cognitive vulnerabilities, interpersonal factors).

Conclusion

In our study, we elaborated on the putative causal pathways from cumulative contextual risks in childhood to depression diagnosis in early adulthood through adolescent alcohol use (a risk pathway) and perceived social support (a protective pathway). To explain how adolescents who were exposed to higher levels of contextual risk might be more vulnerable to depression, we started with a general path model, and then attempted to determine specific indirect effects from cumulative contextual risk to depression diagnosis. Further, in order to examine gender differences in the indirect effects from cumulative contextual risk to depression diagnosis, we compared those indirect effects between boys and girls. Our analyses demonstrated that associations between cumulative contextual risk in childhood and depression diagnosis in adulthood are mediated by adolescent alcohol use and perceived social support from family and friends both for boys and girls, thus providing evidence for the hypothesized risk and protective pathways. A unique contribution of this study was the use of a long-term longitudinal design with a large birth cohort, which provided us with the advantage of testing the proposed mediating mechanisms during an important developmental period – adolescence- which is characterized by the rapid growth of depression symptoms. Our findings indicate a need for programs that increase parental and peer social support and effectively reduce depressive symptoms in adolescents. It has been reported that interventions that teach children and adolescents the skills required for maintaining a diverse supportive social network have shown promising results for building up the resilience mechanisms against depression (Southwick & Charney, 2012). Future research should examine the developmental appropriateness and critical periods for these interventions and their relevance for adolescents.

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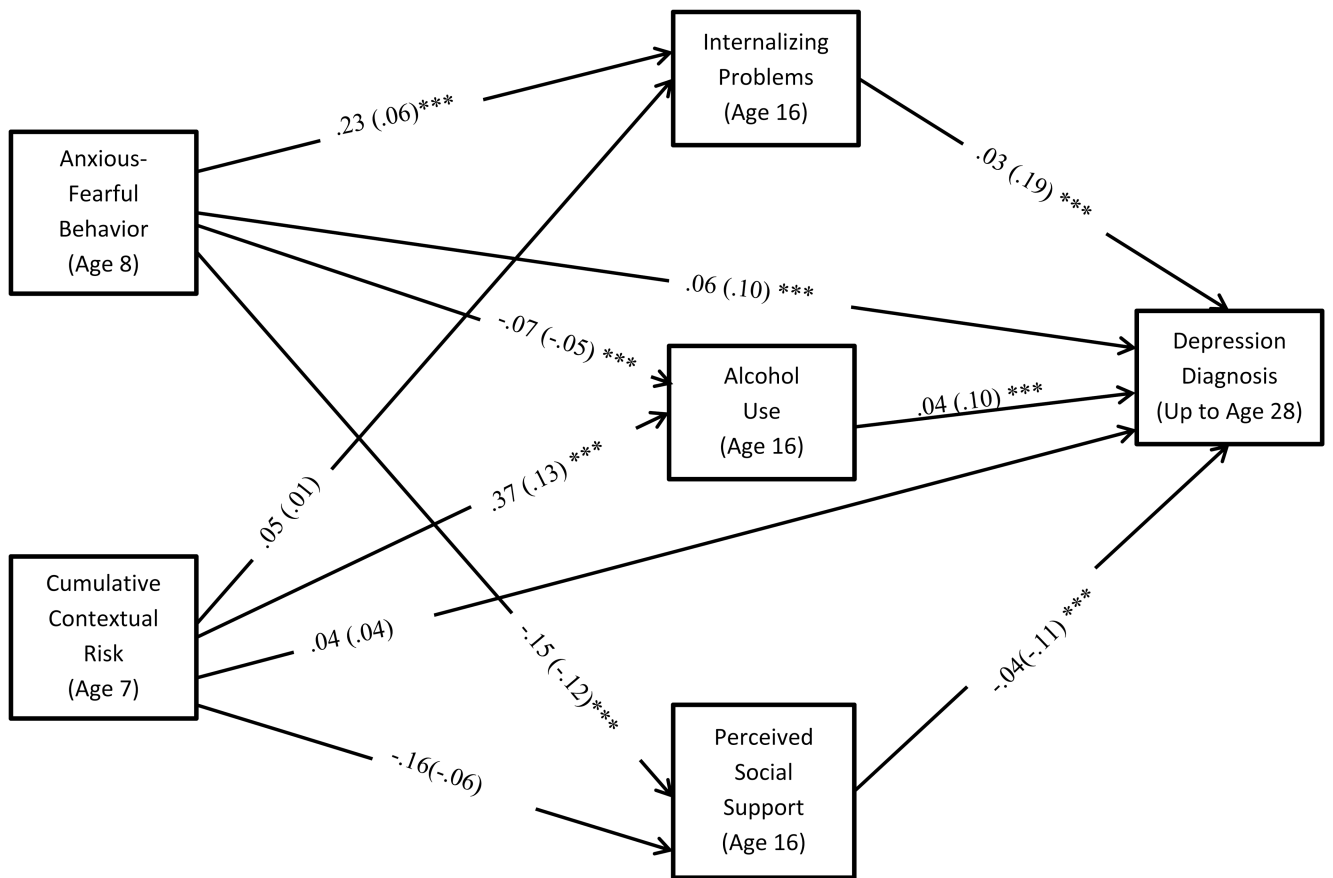


Figure 1. Final fully saturated model showing estimated (standardized) coefficients in the full analysis sample (N = 6,963).
 Note: *p < .05, **p < .01, ***p < .001

Table 1

Descriptive Statistics of Analytic Variables

Variable	Overall Sample			Female		Male	
	Mean/%	Range	SD	Mean/%	SD	Mean/%	SD
Depression Diagnosis (Register)	6.4%	0-1		9.3%		4.8%	
Anxious-Fearful (age 8)	1.03	0-16	1.74	0.94	1.67	1.12	1.80
Internalizing Problems (age 16)	40.03	23-81	7.15	42.52	7.63	37.30	5.52
Alcohol Use (age 16)							
At least one alcohol drink in the past 12 months	2.18	0-6	1.85	2.24	1.84	2.12	1.86
Being drunk in the past 12 months	1.67	0-6	1.71	1.73	1.71	1.61	1.72
Heavy episodic drinking past 30 days	0.74	0-5	1.08	0.74	1.07	0.73	1.09
Social Support (age 16)							
Meet friends	4.60	1-5	0.73	4.62	0.70	4.59	0.75
Spend time with family	4.20	1-5	1.19	4.26	1.15	4.13	1.23
Have meals with your parents	4.44	1-5	1.01	4.31	1.10	4.57	0.87
Do you have a close friend	3.10	1-4	0.98	3.22	0.88	2.96	1.07
Parent interest in your school/hobbies	2.85	1-3	0.38	2.85	0.38	2.85	0.37
Sex (male)	49%	0-1					
Cumulative Contextual Risk							
Teenage Mom (<20) (Prenatal)	3.8%	0-1					
Smoking while pregnant (Prenatal)	12.9%	0-1					
Drinking while pregnant (Prenatal)	11.3%	0-1					
Single Parent (age 7)	6.6%	0-1					
Unemployed Mother (age 7)	12.1%	0-1					
Unemployed Father (age 7)	10.7%	0-1					
Father education less than 9 yrs (age 7)	12.6%	0-1					
Mother education less than 9 yrs (age 7)	6.4%	0-1					

Table 2

Correlations of Study Constructs

Variable	1	2	3	4	5	6	7
1. Male							
2. Cumulative contextual risk	-.002						
3. Anxious-Fearful (age 8)	.051**	.072**					
4. Internalizing Problems (age 15)	-.363**	.012	.039**				
5. Social Support (age 15)	-.026*	-.064**	-.102**	-.231**			
6. Alcohol Use (age 15)	-.026*	.127**	-.037**	.100**	-.045**		
7. Depression Diagnosis	-.087**	.040**	.073**	.176**	-.104**	.072**	

Note:

**
p < .001

Table 3
Standardized Total, Direct, and Indirect Effects from Cumulative Contextual Risk to Depression Diagnosis through Social Support and Alcohol Use

Path	<i>b</i>	Bootstrap 95% CIs <i>b</i>	
		Lower	Upper
<i>Social Support Mediation</i>			
CCR → Social Support	-.057	-.082	-.032
Social Support → Depression	-.113	-.153	-.073
Indirect Effect	.007	.003	.011
<i>Alcohol Use Mediation</i>			
CCR → Alcohol Use	.131	.105	.157
Alcohol Use → Depression	.100	.052	.145
Indirect Effect	.013	.007	.021
Direct Effect CCR → Depression	.041	-.004	.083
Total Indirect CCR → Depression	.022	.013	.031
Total Effect CCR → Depression	.019	.063	.104

Note. CCR = Cumulative Contextual Risk; CI = confidence interval