Published in final edited form as:

J Abnorm Child Psychol. 2019 June ; 47(6): 1065–1074. doi:10.1007/s10802-018-0501-z.

Adolescent Depression and Substance Use: The Protective Role of Prosocial Peer Behavior

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Abstract

Adolescents with depression disorders have higher rates of substance use. In order to advance contextually relevant mental health interventions, basic research is needed to test social ecological mechanisms hypothesized to influence adolescent depression and substance use. Accordingly, we conducted growth curve modeling with a sample of 248 urban adolescents to determine if depression's effect on substance use was dependent upon peer network health (sum of peer risk and protective behaviors) and activity space risk (likelihood of high-risk behaviors at routine locations). Results showed that peer network health moderated the effects of depression on substance use, but this effect was not altered by activity space risk. These findings suggest the importance of peer network health relative to depression and substance use, particularly for young adolescents.

Keywords

Urban adolescents; Depression; Peer networks; Substance use; Activity space

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Disclosure of potential conflicts of interest:

The authors declare that they have no conflict of interest

Statement of human rights:

We followed all ethical guidelines in conducting this research as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed consent:

Informed consent was obtained from all individual participants included in the study

Approximately half of all lifetime psychiatric disorders begin by age 14 and three-quarters by age 24, signifying that childhood mental health must be fully evaluated and addressed, as the presence of these disorders further complicate and worsen mental health outcomes when not addressed (Kessler et al., 2007). Adolescent depression and its association with substance use is a complicated public health issue. For example, adolescents with depression engage in more substance use to alleviate interpersonal problems, through participating in substance use with peers (Rudolph, Flynn, & Abaied, 2008) and adolescents with more depression symptoms have more friends who smoke, drink alcohol, and use marijuana (Siennick et al., 2015). However, research has also shown that improvement of specific protective factors such as peers and neighborhood context, can positively affect a child's developmental trajectory and decrease risk for psychiatric and substance use disorders (Scott, Wallander, & Cameron, 2015). Neighborhood context, such as the objective features and characteristics within a defined location and the subjective interpretation of these places, is an important domain to address in adolescent research (Gutman & Sameroff, 2004; Mason, Mennis, Way, & Zaharakis, 2015; Mennis, Stahler, & Mason, 2016). Understanding these dynamic relationships among depression, peers, and neighborhood context is important in developing ecologically sensitive substance use prevention and treatment models. The current study focuses on the interactive effects of these constructs associated with trajectories of young urban adolescent substance use.

Depression

The prevalence of depression disorders among adolescents varies by gender, race/ethnicity, and by substance use. For example, 12.5% of U.S. adolescents ages 12 to 17 are estimated to have experienced a major depressive episode (MDE) during 2015, including 5.8% of boys and almost four times as many girls (19.5%) (Center for Behavioral Health Statistics and Quality, 2016). Adolescents who identify with two or more races are most likely to have experienced a MDE (15.6%) in 2015, followed by Whites (13.4%), Hispanic or Latino (12.6%), Asian (9.7%), and Black or African American (9%) adolescents (Center for Behavioral Health Statistics and Quality, 2016). Adolescents who have experienced a MDE use illicit drugs at twice the rate compared to those adolescents without MDE (31.5% to 15.3%), are twice as likely to misuse prescription medication (12.2% to 4.9%), twice as likely to be daily cigarette users (1.8% to 0.7%), and binge alcohol users (1.7% to 0.8%) (Center for Behavioral Health Statistics and Quality, 2016). Adolescents with psychiatric disorders also have higher rates of substance use disorders (SUD), with rates varying according to the type of mental health diagnosis. Depression has been the most frequently examined, with most studies suggesting a range of 20% to 30% comorbidity of SUDs and depression (Mason et al., 2016). Research on the role of peers in the development of depression disorders provides insight into how peer context can be uniquely leveraged to address adolescent depression. For example, research has found that adolescents with involved, prosocial and supportive peers, experience less depression (Fredricks & Eccles, 2008; Seidman et al., 1999), while those with more negative peers (using drugs, stealing,) experience more depression in young adulthood (Gutman & Sameroff, 2004).

Peers and Health Outcomes

The peer group provides a critical social influence for adolescents in the initiation of substance use (Huang, et al., 2014; Sieving, Peery, & Williams, 2000) and depression (Brendgen, Vitaro & Bukowski, 2000; Mason, Hitchings, & Spoth, 2008). The role of peers influencing risk and protective health behaviors is thought to peak during mid adolescence (Pesola et al., 2015). These important peer networks (close friendships) establish group norms that define peer group culture, for both prosocial as well as antisocial behavior. Primary socialization theory proposes that these group norms of behavior are transmitted through social contexts such as family, school, and peers. This theory states that the adolescent's mental health and personality traits do not directly cause substance use, but instead, affect this outcome when the individual interacts with the primary socialization source (Oetting, Deffenbacher, & Donnermeyer, 1998). For example, individual factors such as depression, influence the interaction with particular deviant peers, and this social interaction increases the likelihood of substance use, thereby contributing to the primary socialization processes. In this example, the primary socialization context is risk exacerbating, but the individual's risk behavior is only expressed within the setting of the deviant peer interaction (Oetting, Donnermeyer, & Deffenbacher, 1998). This model is unique in that it locates the health behavior within the context of interpersonal relations, instead of completely within the individual.

Extensive research has shown that peer context predicts tobacco, alcohol, and drug use (Bauman & Ennett, 1996; Knecht et al., 2011; Light et al., 2013; Valente et al., 2005). However, much less evidence is available for prosocial effects of peers. Supportive friendships have been studied as a moderator or protective influence against psychological and behavioral problems, often associated with peer rejection (Lansford et al., 2007) or with negative experiences within families (Bolger et al., 1998), both of which are linked to substance use uptake. Adolescents who are rejected by their normative peer group due to depression or substance use gravitate towards a deviant peer group to reduce isolation (Brendgen, Vitaro & Bukowski, 2000; Petraitis, Flay, & Miller, 1995). Subsequently, the adolescent adopts the new group's norms through social processes such as imitation of high-risk behaviors, and receives positive reinforcement, providing belonging and identity formation.

Activity Space

Place-oriented researchers have used the construct of activity space as a broad measure to understand the spatial dimensions of how place influences and interacts with individuals' lives. Activity space can be defined as an index that comprises all the locations that an individual has direct contact with as a result of his or her daily activities (Miller, 1991). Activity spaces are the manifestation of our spatial lives, representing the routine locations and all the accompanying psychological, social, and health-related experiences of these places (Golledge, 1997). Multiple approaches to measuring activity space have been used to capture individual location data within a specific time-frame such as travel diaries (Goodchild & Janelle 1984), structured interviews (Mason, Cheung, & Walker, 2004), and using location tracking technologies such as Global Positioning Systems (GPS) (Kwan,

2013; Mennis et al., 2016). This geographically informed research has shown that youth, and urban adolescents in particular, spend their time in a variety of spatially dispersed activity spaces that are not delimited by conventional geographic boundaries, such as census tracts, zip codes, political wards, or even home neighborhood (Browning & Sober, 2014). Research on activity spaces suggests that the places a person frequents outside the home exposes him or her to a variety of psychological, social, and geographic factors that influence issues such as substance use that are not apparent when observations are limited to only the home neighborhood (Mason et al., 2015; Mennis and Mason, 2011; Wong & Shaw, 2011; Zenk et al., 2011).

Most peer relations research does not include place as a construct of interest, ignoring individuals' subjective meaning of places, and the accompanying dynamic between place and peers. However, peer relations are not aspatial, but rather are embedded within places. Adolescents' experiences of place occur through social interactions that ensue at particular locations. Indeed, an adolescent's perception of a place is closely tied to the people with whom the adolescent interacts and their associated attitudes, values, and behaviors that are constituted at particular locations (Korpela, 2012; Smaldone et al., 2008). Understanding these dynamic social-spatial processes could advance the knowledge base of adolescent depression and substance use.

The Current Study

The present study contributes to the literature by incorporating activity space, peer network health (sum of peer risk and protective behaviors), depression, and substance use over a 24-month study with young urban adolescents. Our goal was to analyze the individual trajectories of substance use involvement over time and the associated moderating effects of depression and peer network health. We tested two hypotheses. First, we hypothesized that peer network health would moderate the effect of depression on substance use, such that more protective peer networks would suppress the effect of depression on substance use. Second, we hypothesized that adolescents with less risky activity spaces would experience increased protective effects from their peer networks, thereby buffering the influence of depression on substance use.

Methods

Participants

This study utilizes data from the Social-Spatial Adolescent Study, a two-year longitudinal investigation of the interacting effects of peer networks, urban environment, and substance use. Participants were recruited between November 2012 and February 2014. The majority of participants (72%) were recruited from an urban adolescent medicine primary care clinic at Virginia Commonwealth University Medical Center, in Richmond Virginia. Age-eligible (age 13 or 14) adolescents presenting to the clinic for routine or acute care were approached and invited to participate in this study by a research assistant. Other participants were recruited from a city health district satellite clinic, located within a subsidized housing development. These participants were recruited by referral to the study team from the primary Patient Advocate at the satellite clinic. Over 400 adolescents and parents were either

approached at the outpatient hospital clinic or referred from the satellite clinics, of these, 57% enrolled in the study (N=248). Enrollment and data collection procedures were the same across sites. Written informed consent was obtained from all parents and adolescent participants prior to conducting any research activities. The first author's university and the Richmond City Health Department's institutional review boards approved the research protocol, and the study received a federal Certificate of Confidentiality from the National Institutes of Health.

All participants completed the 30-minute baseline survey on a study laptop in a private room separate from parents and any clinic staff. Participants completed follow-up web surveys upon receiving a text message and email, with an imbedded URL link to the survey. Follow-up surveys were conducted every six for 24 months, and the majority of participants (84%) completed through the 24-month follow-up survey. Subsequent independent t-tests revealed no significant differences between completers and non-completers on key variables such as substance use, peer network health, family history, neighborhood disorder, age, race, and gender ($p > 0.05$). Participants were provided a study phone with service at enrollment and received up to \$140 for completing baseline and follow-up surveys every six months for two years. Chi-square tests revealed no significant differences in age, sex, or race of participants between the two recruitment sites.

Measures

Demographics.—Participants reported age, gender, race and ethnicity during the baseline survey.

Neighborhood Disorder.—Neighborhood disorder was measured at the block group level for each participant in order to capture an ecological picture of participant's home environments. We used the U.S. Census Bureau and crime data to construct an index of neighborhood disorder incorporating percent living below the poverty line, percent single parent households, percent housing that is vacant, percent housing that is not owner occupied, and an index of the rate of criminal assaults. Using 2013 estimates (EASI, 2016) for the 103 block groups in the Richmond, Virginia metropolitan area that contain subject residences, the index was calculated as the mean of the z-scores of the variables (Cronbach's $\alpha = .84$). Each participant was assigned a SES/neighborhood disorder value based on the block group within which they resided at enrollment.

Family History and Attitudes Towards Substance Use.—Family substance use history and family attitudes toward substance use were assessed using the 7-item scale from the Communities that Care survey (Arthur, Hawkins, Pollard, Catalano, & Baglioni, 2002). Prior research has documented favorable reliability (Cronbach's $\alpha = 0.78$). Items assess substance use problems within the family and the adolescent's perceptions of the parents' attitudes about the adolescent's use of substances. Items were summed to create a total score. Higher scores indicate higher levels of family risk related to substance use.

Depression.—Depression was measured using the scale from the Behavior Assessment System for Children, second edition (Reynolds & Kamphaus, 2006), a twelve item measure

that assesses traditional symptoms of depression, including sadness, hopelessness, loneliness, and inability to enjoy life. The scale has an acceptable Cronbach's alpha reliability coefficient of 0.83. Items were summed to create a total scale score, with scores ranging between 0 and 20. Higher scores indicate greater levels of depression symptomatology.

Peer Network Health.—Peer network health data were gathered using the Adolescent Social Network Assessment (ASNA; Mason, Cheung, & Walker, 2004). The ASNA captures information on each subject's close personal contacts, which constitute their personal or egocentric friend network. Because our study focused on the influence of close friend networks, we limited the number of nominated close friends to three as this is close to the national average of nominated friends (2.54; Ali et al., 2011). These selected friends were those with whom participants indicated they spent the most time with on average. Researchers investigating peer effects on substance use have often used perceptions of the index individual on their peers' substance use. This approach is known as an ego-centric network design where individuals identify his or her close peers and their relationships. This design obtains estimates of peer effects without conducting a full social network analysis and is widely used (Deutsch, Chernyavskiy, Steinley, & Slutske, 2015; Mason, 2014; Valente et al., 2013).

Adolescents were asked to think of up to three friends and to provide information about each of their friend's substance use, influence on behavior, and types of activities. Specifically, participants were asked about negative/risky activities of each nominated peer such as substance use, daily substance use, and participation in illegal, violent, or dangerous behaviors. The ASNA also asks whether the participant has been influenced to use or not to use substances by each friend. In addition, participants were asked about positive/protective activities with their friends such as receiving help with school or transportation (instrumental support), or by talking through problems (emotional support). These items create a total score for each friend and are based upon a weighted scoring procedure, with scores ranging from -14 to 14. Weights are based upon previous research that has shown, for example, that risk for substance use increases over 4-fold when adolescents' close peers engage substance use and that this behavior produces medium effect sizes (Mason, 2009). Further, protective dimensions of peer behaviors such as engaging in positive or prosocial activities has shown a 3-fold reduction in risk for substance use with medium effect sizes (Mason, 2009). Given these data, we developed the following weighted scoring procedures that assigns weights to both risk and protective peer behavior: substance user = -1, daily user = -3, high-risk activity = -4, influence to use = -6; and non-substance user = 4, absence of high-risk activities = 4, influence not to use = 6. Each peer's score is summed. Assuming three peers per participant, total peer network health scores can range from -42 to 42. Higher scores indicate greater peer network health, and lower scores indicate increased behavioral risk. The ASNA has favorable internal reliability (Cronbach's alpha = .84) and correlates significantly in the expected direction with self-reported measures of substance use (any alcohol, marijuana or other substance, $r = -.64$), alcohol use ($r = -.66$) and marijuana use ($r = -.54$; Mason, Mennis, & Schmidt, 2011).

Activity Space Risk.—Activity space risk was measured with the Ecological Interview (Mason, Cheung, & Walker, 2004). The Ecological Interview is a measure that produces a geographical listing of the participant's routine activity locations, as well as evaluative descriptions of these various environments which are used to characterize their geography of risk and protection. The Ecological Interview produces accurate and valid geographic data with previous studies successfully identifying (geocoding specific locations) 90% of the collected location data (Mason et al., 2004). For this study, participants were asked to "Think of your typical week and about the places you go, excluding your home." Participants then selected types of places they frequent most often during a typical week from a list of eight common places including an option for other places not listed. The types of places are: school, friend's home, city places (city streets/corners, stores, restaurants), religious site, park/nature, recreation/sports center, work/job, other place. The participant then rated the likelihood that they would use tobacco, alcohol, and marijuana at each of their selected locations. The ratings were coded on an eight-point scale, from 1 = *Not at all likely* to 8 = *Very likely*. Participants identified types of locations and the corresponding rating of each location's level of risk to use particular substances. An activity space risk score is a summation of their ratings at each location. A participant could select up to eight places producing scores ranging from 3 to 192 (e.g., one location rated as 1 for each substance = 3, to eight locations rated as 8 for each substance = 192). Total scores are then divided by the number of locations, producing a mean activity space risk value. Higher scores indicate a cumulative risk of substance use incorporating exposure to all the locations that an adolescent typically frequents.

Substance Use.—The Adolescent Alcohol and Drug Involvement Scale (AADIS, Moberg and Hahn, 1991) was used to measure alcohol and drug use severity (including alcohol, marijuana/hashish, hallucinogens, cocaine, barbiturates, PCP, heroin and other opiates, and tranquilizers). The AADIS captures risk factors for substance use disorders. The scale has possible values ranging between 2 and 69, with higher scores indicating greater substance use severity. A score of 2 indicates never having used a substance (abstinence), and scores greater than 36 indicate a likely substance use disorder. The AADIS has excellent internal consistency (Cronbach's alpha 0.94) and is highly correlated with self-reported measures of substance use ($r = 0.72$), clinical assessments ($r = 0.75$), and subjects' perceptions of the severity of their own drug use problem ($r = 0.79$).

Statistical Analysis

Growth modeling of substance use severity.

We conducted growth modeling analyses in a multilevel modeling (MLM) framework to understand (a) the baseline level and rate of change in substance use severity across the 24 months in the study and (b) how baseline levels of depression, peer network health, and activity space risk affected these growth parameters. Equation 1 shows the unconditional growth model.

$$\begin{aligned}
 SU_{it} &= \beta_{0i} + \beta_{1i}(t_{it}) + \varepsilon_{it} \\
 \beta_{0i} &= \gamma_{00} + u_{0i} \\
 \beta_{1i} &= \gamma_{10} + u_{1i}
 \end{aligned} \quad (1)$$

Substance use severity at time t for adolescent i (SU_{it}) is modeled as a function of the baseline level for adolescent i (β_{0i}), a linear slope describing its change over time for adolescent i (β_{1i}), and a residual term (ε_{it}). Time was scaled (t = months since baseline / 24) to have a minimum of 0 (at baseline) and a maximum of 1 (at 24 months). The fixed effects, or the means of the baseline level and slope, are represented by γ_{00} and γ_{10} , respectively. Individual-level differences from these means are represented by u_{0i} and u_{1i} , respectively. The model captures the variances for these individual differences in the baseline level and slope via the parameters τ_{00} and τ_{11} , respectively, and the covariance between the baseline level and slope is captured by τ_{10} . Variance of the ε_{it} residuals is captured by σ_{ε}^2 ; residual variance was held constant across all measurement waves. We ran both a linear growth model (as specified in Equation 1) as well as a quadratic (which included a t_{it}^2 term) and compared the fit of these using likelihood ratio tests of the deviance, or -2 times the log likelihood ($-2LL$) in order to identify the best-fitting functional form of substance use trajectories over time.

Equation 2 shows a conditional growth model wherein the individual growth parameters (the intercept (β_{0i}) and the time slope (β_{1i})) are predicted by baseline depression and peer network health (PNH), as well as their interaction.

$$\begin{aligned}
 SU_{it} &= \beta_{0i} + \beta_{1i}(t_{it}) + \varepsilon_{it} \\
 \beta_{0i} &= \gamma_{00} + \gamma_{01}(DEP_i) + \gamma_{02}(PNH_i) + \gamma_{03}(DEP_i \times PNH_i) + u_{0i} \\
 \beta_{1i} &= \gamma_{10} + \gamma_{11}(DEP_i) + \gamma_{12}(PNH_i) + \gamma_{13}(DEP_i \times PNH_i) + u_{1i}
 \end{aligned} \quad (2)$$

In this model, we are testing whether baseline depression (DEP_i), PNH (PNH_i), and their interaction ($DEP_i \times PNH_i$), are significant predictors of adolescents' initial levels of substance use (β_{0i}) and their rate of change in substance use (β_{1i}). PNH and depression were Z-scored ($M = 0$, $SD = 1$) for easy interpretation, and the model adjusted for effects of sex, age, black (versus non-black) and family history, all of which were mean centered, on both the intercept and slope terms. Significant interactions were unpacked using model estimated simple effects of the initial level and rate of change. These were obtained by substituting high ($M + SD$) and low ($M - SD$) values for baseline depression and peer network health, and using postestimation commands to generate predicted values along with their standard errors and 95% confidence intervals. For example, using Equation 2, the predicted initial value of SU for an adolescent with high depression and high PNH is $\hat{\beta}_{0i} = \gamma_{00} + \gamma_{01}(1) + \gamma_{02}(1) + \gamma_{03}(1 \times 1) = \gamma_{00} + \gamma_{01} + \gamma_{02} + \gamma_{03}$, and their predicted rate of

change is $\check{\beta}_{1i} = \gamma_{10} + \gamma_{11}(1) + \gamma_{12}(1) + \gamma_{13}(1 \times 1) = \gamma_{10} + \gamma_{11} + \gamma_{12} + \gamma_{13}$. For an adolescent with high depression and low PNH, however, model predictions are $\check{\beta}_{0i} = \gamma_{00} + \gamma_{01}(1) + \gamma_{02}(-1) + \gamma_{03}(1 \times (-1)) = \gamma_{00} + \gamma_{01} - \gamma_{02} - \gamma_{03}$ and $\check{\beta}_{1i} = \gamma_{10} + \gamma_{11}(1) + \gamma_{12}(-1) + \gamma_{13}(1 \times (-1)) = \gamma_{10} + \gamma_{11} - \gamma_{12} - \gamma_{13}$ for the initial level and rate of change, respectively. These linear combinations were generated using ESTIMATE statements in SAS PROC GLIMMIX.

Equation 3 shows a three three-way interaction model wherein baseline depression, baseline PNH, and baseline activity space risk (ASR; also Z-scored with M=0, SD=1) predict the baseline level and rate of change in substance use severity.

$$\begin{aligned}
 SU_{it} &= \beta_{0i} + \beta_{1i}(t_{it}) + \varepsilon_{it} \\
 \beta_{0i} &= \gamma_{00} + \gamma_{01}(DEP_i) + \gamma_{02}(PNH_i) + \gamma_{03}(ASR_i) \\
 &\quad + \gamma_{04}(DEP_i \times PNH_i) + \gamma_{05}(DEP_i \times ASR_i) + \gamma_{06}(PNH_i \times ASR_i) \\
 &\quad + \gamma_{06}(PNH_i \times PNH_i \times ASR_i) + u_{0i} \\
 \beta_{1i} &= \gamma_{10} + \gamma_{11}(DEP_i) + \gamma_{12}(PNH_i) + \gamma_{13}(ASR_i) \\
 &\quad + \gamma_{14}(DEP_i \times PNH_i) + \gamma_{15}(DEP_i \times ASR_i) + \gamma_{16}(PNH_i \times ASR_i) \\
 &\quad + \gamma_{16}(PNH_i \times PNH_i \times ASR_i) + u_{1i}
 \end{aligned} \tag{3}$$

This model also included sex, age, black (versus non-black) and family history, all of which were mean centered, as covariates on both the intercept and slope terms. All growth models were conducted in SAS PROC GLIMMIX with an identity link and assuming normal distribution. Models used full maximum likelihood estimation and Huber-White robust standard errors.

Results

Descriptive Statistics & Correlations.

The sample was 57% female, 88% African American, with an average initial age of 13.4 years. Less than one-third of the sample (28%) moved at least once within a one-year period of the study. Table 1 provides correlations among the key variables. As expected, depression was positively correlated with substance use and activity space risk. Peer network health was negatively correlated with substance use and activity space risk.

Growth modeling of substance use severity.

Linear and quadratic unconditional growth models were run to determine the functional form of change. The variance of the quadratic term had to be set to zero in order for the quadratic model to achieve convergence, thus the quadratic model differed from the linear only by 1 degree of freedom. The deviances of these models were compared using a likelihood ratio test (LRT) with one degree of freedom. Although the smaller deviance for the quadratic model ($-2LL = 8146.22$) suggested a better fit than the linear model ($-2LL =$

8149.20), this difference was not statistically significant, $\chi^2_{(df = 1)} = 2.98, p = 0.084$, suggesting that the additional complexity of the quadratic model was not supported by the data. Thus, the linear model was retained.

Table 2 shows the results of the unconditional growth model. The fixed effects are the parameters show for the average of the individual growth curves. The average baseline level of substance use severity (γ_{00}) is 4.8 ($p < 0.0001$), and its average rate of change from 0 to 24 months (γ_{10}) is an increase of 3.2 units ($p = 0.0012$). The variances of the intercept (τ_{00}) and the time slope (τ_{11}) are both significant, suggesting that there is significant variation in the baseline level and rate of change in substance use severity across adolescents. However, the covariance between the intercept and slope (τ_{10}) is not significant, suggesting that one's initial level of substance use severity is not associated with their rate of change over time.

Figure 1 shows a spaghetti plot of the model-estimated growth curves. The black line shows the average of the growth curves, generated using the fixed effects parameters for the initial level (γ_{00}) and the rate of change (γ_{10}). The gray lines show the individualized growth curves, generated using the fixed effects parameters and the random deviations for each adolescent for both the initial level ($\gamma_{00} + u_{0i}$) and the rate of change ($\gamma_{10} + u_{1i}$). Figure 1 shows tremendous individual-level variability in the initial level and the rate of change over time, as suggested by the large and highly significant random effects for both parameters.

Table 3 shows the results of the conditional growth model testing the interaction between baseline depression and PNH on the initial level and rate of change. Table 3 shows a significant interaction between depression and PNH in predicting the initial level of substance use severity ($b = -1.80, SE = 0.82, p = 0.0281$) but no significant effects on the rate of change were found. Simple effects analyses showed that PNH appeared to buffer the effect of depression on substance use severity. Model predictions showed that for adolescents with low PNH ($M - SD$), depression was significantly associated with increased levels of substance use severity at nearly all assessment waves ($p < .05$ at months 0, 6, 12, 18 and $p = 0.06$ at month 24). For adolescents with high baseline PNH ($M + SD$), depression was not significantly associated with substance use severity at any of the assessment waves, suggestive of a buffering effect. Figure 2 shows the substance use severity trajectories by depression and PNH, illustrating the buffering effect of high PNH.

Finally, we tested the three-way interaction between baseline depression, baseline PNH, and baseline activity space risk. This three-way interaction did not significantly predict the initial level ($p = 0.11$) nor the rate of change ($p = 0.42$), suggesting that the buffering effect of PNH on depression was not altered by activity space risk.

Discussion

The present study provides insight into the developmental effects of several risk and protective factors for substance use among young urban adolescents. A unique contribution of this study is the examination of close peers and meaningful places across 24 months with an understudied population, young urban African-American adolescents. Identifying the long-term effects of depression on substance use and the role of close peer networks and

activity space risk, is useful in examining the complex processes associated with adolescent substance use.

The finding that depressions' effect on substance use at age 13 is dependent upon the adolescents' peer network health has important clinical implications. It appears that adolescents experiencing heightened levels of depression symptoms entered our study using substances at a much higher rate, compared to adolescents with higher levels of peer network health. Having higher levels of peer network health buffered higher levels of depression's influence on substance use for these young adolescents. This finding supports previous research in this area with similar samples (Fredricks & Eccles, 2008; Seidman et al., 1999). It is reasonable to assume that adolescents in the current study who had a protective set of close friends, i.e., friends who are engaged in prosocial activities and who provided instrumental and emotional support, experienced a buffering effect of their peer network health against depression and substance use.

It may be that the adolescents with low levels of peer network health are using substances to cope with their depressive symptoms. This would be consistent with previous research with adolescents and would support the self-medication hypothesis (Tomlinson & Brown, 2012). It is plausible that these adolescents have more peers that are using substances, and who are more depressed, thus providing a model for dealing with depressive symptoms. This interpretation would be consistent with primary socialization, where the substance use is expressed within the context of the peer relations (Oetting, Donnermeyer, & Deffenbacher, 1998).

The clinical implications for these results could include focusing on the importance of health promoting peer groups, perhaps prior to when children enter middle school. Based on these findings, screening for peer network health is warranted. This type of screening needs to be sensitive to children's strong protective feelings of their peers, particularly if these screens are only focusing on risk behaviors. Including pro-social behavioral assessment as part of a general health screening, would reduce potential resistance and may provide more accurate data (Mason, 2014). In terms of clinical approaches, peer-focused interventions such as Peer Network Counseling, has shown promising outcomes with adolescents (Mason et al., 2016) by leveraging the importance of peers.

Contrary to our second hypothesis, we did not find a significant effect of activity space risk on peer network health. While previous research that adolescents' perceptions of risk for substance use at particular places is closely tied to the people with whom the adolescent interacts (Korpela, 2012; Mason, 2010; Smaldone et al., 2008), for this sample, the three-way interaction was not significant. This finding may provide support that young adolescents are less mobile than their older counterparts. Having less access to places that provide risk and or protection from substance use may have contributed to the damping of the effect of these places. Access to transportation options such as driving or public transportation, typically increases during middle to late adolescence. In other words, it may be that during early adolescence, peers exert a stronger effect on mental and behavioral health compared to activity places.

There are several study limitations to consider when interpreting these results. First, the sample was an urban, primarily African American sample and therefore our findings may not apply to other populations. While this is an important population to study due to historic underrepresentation within research studies on adolescents, replications with more diverse ethnic and geographic populations are needed. Second, our measure of peer network health was self-report, possibly limiting the accuracy of the data collected regarding friends' antisocial and prosocial activities. However, some research has suggested that an adolescent's perceptions of their friends' antisocial behaviors are more predictive of engagement in risk behavior than collecting data directly from friends (e.g., Deutsch, Chernyavskiy, Steinley, & Slutske, 2015). Further, we limited our friend network measure to collect data on each participant's three closest friends. While this is in line with data on the average number of nominated close friends among adolescents (Ali et al., 2011), findings may differ for a more expanded inclusion of friendships. Future studies could examine more extensive friend networks to determine if these findings vary with the size of an adolescent's friend network.

The present study results provide novel insight into the interactive effects of peers, depression, place, and substance use trajectories. This study contributes to literature on the role of peers in relation to mental and behavioral health. Very little is known about the interactive effects of peer network health on depression's influence on substance use with young, urban adolescents. Given these findings, research examining these associations among younger adolescents could provide important developmental insights into how adolescents may be coping with depressive symptoms by utilizing their peer network.

Acknowledgments

Funding: This research was supported by a Grant No. 1R01 DA031724-01A1, from the National Institute on Drug Abuse to the first author. The findings and conclusions are those of the authors and do not necessarily represent the views of the National Institute on Drug Abuse, or National Institute of Health.

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Figure 1. Spaghetti plot showing model-estimated growth curves for the sample average (black line) and for each adolescent in the study (gray lines).

Figure 2.
Growth curves of substance use severity by depression and PNH.

Table 1.

Correlations of Key Variables at Baseline

	1	2	3	4
1. Substance Use	-			
2. Depression	.216 [*]	-		
3. Peer Network Health	-.333**	-.132 [*]	-	
4. Activity Space Risk	.575**	.272**	-.208**	-

^{*}
= $p < 0.05$;

^{***}
= $p < 0.01$

Table 2.

Unconditional linear growth model of substance use severity

<i>Fixed Effects</i>	Estimate	SE	p	95% LCL	95% UCL
Intercept (γ_{00})	4.77	0.72	<.0001	3.36	6.19
Time Slope (γ_{10})	3.17	0.97	0.0012	1.26	5.08
<i>Random Effects</i>	Estimate	SE	p	95% LCL	95% UCL
Intercept (τ_{00})	69.64	11.99	<.0001	51.04	100.71
Intercept & Time Slope Covariance (τ_{10})	-2.03	13.21	0.8776	-27.92	23.85
Time Slope Variance (τ_{11})	60.17	22.45	0.0037	32.47	147.54
Residual Variance (σ^2_{ϵ})	88.24	17.02	<.0001	78.89	99.3472

SE = standard error, LCL = lower confidence limit, UCL = upper confidence limit.

Table 3.

Conditional linear growth model of substance use severity

<i>Fixed Effects</i>					
Predicting the Intercept	Estimate	SE	p	95% LCL	95% UCL
Depression	1.67	0.72	0.0214	0.25	3.09
PNH	-2.60	0.74	0.0005	-4.05	-1.15
Depression x PNH	-1.80	0.82	0.0281	-3.41	-0.20
Age	1.03	1.24	0.4053	-1.41	3.48
Sex	1.49	1.31	0.2581	-1.10	4.07
Black	-0.21	2.11	0.9195	-4.37	3.95
Family History	2.64	0.79	0.001	1.08	4.21
Constant	4.51	0.61	<.0001	3.31	5.71
Predicting the Time Slope	Estimate	SE	p	95% LCL	95% UCL
Depression	0.10	1.09	0.9294	-2.05	2.24
PNH	1.60	1.02	0.1172	-0.40	3.60
Depression x PNH	1.08	1.21	0.3726	-1.29	3.45
Age	-0.34	1.91	0.8593	-4.09	3.41
Sex	-0.21	1.97	0.9141	-4.07	3.65
Black	0.02	2.71	0.9938	-5.29	5.34
Family History	-1.32	0.98	0.1789	-3.25	0.61
Constant	3.36	0.96	0.0005	1.47	5.24
<i>Random Effects</i>					
	Estimate	SE	p	95% LCL	95% UCL
Intercept	36.87	9.19	<.0001	23.87	64.38
Intercept & Time Slope Covariance	13.57	11.15	0.2236	-8.29	35.43
Time Slope Variance	51.95	21.54	0.0079	26.48	144.37
Residual Variance	88.31	5.19	<.0001	78.97	99.43

SE = standard error, LCL = lower confidence limit, UCL = upper confidence limit.