Is it Selection or Socialization? Disentangling Peer Influences on Heavy Drinking and Marijuana Use Among Adolescents Whose Parents Received Brief Interventions

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Sara J Becker[1], Kristine Marceau[2], Lynn Hernandez[1] and Anthony Spirito[3]

[1]Center for Alcohol and Addiction Studies, Brown University School of Public Health, Providence, RI, USA. [2]Department of Human Development & Family Studies, Purdue University, West Lafayette, IN, USA. [3]Department of Psychiatry & Human Behavior, The Warren Alpert Medical School, Brown University, Providence, RI, USA.

ABSTRACT: This study attempted to disentangle the effects of peer selection and socialization on heavy drinking and marijuana use among adolescents whose parents received 2 distinct brief interventions (BIs). It also examined whether the two BI models—Family Check-Up and Psychoeducation—had differential effects on peer processes. Parents were randomized to BI conditions and their adolescents (61% male, age 12-19 years) completed self-report measures of days of heavy drinking, days of marijuana use, and perceived peer substance involvement at baseline, 6 months, and 12 months. Separate cross-lagged panel models revealed evidence of selection and socialization for both heavy drinking and marijuana over the first 6 months and evidence of only selection over the subsequent 6 months. Consistent with prior studies, a less robust pattern of peer processes was found when simultaneously controlling for both heavy drinking and marijuana. Results highlight the need to examine multiple substances simultaneously and suggest that the BIs may have had protective effects on peer influences over time.

KEYWORDS: adolescent substance use, selection, socialization, marijuana, alcohol

Peer influences are among the strongest and most consistent factors associated with adolescent substance use (SU).[1,2] Adolescents are particularly susceptible to peer influences given their developmental stage and the importance of peer networks in adolescent life.[3] Although the association between peer influences and adolescent SU has been widely documented,[4,5] mechanisms underlying this relationship are not well understood.

There are 2 predominant theories to explain the influence of peers on adolescent SU: selection and socialization.[6] Selection refers to the tendency of adolescents to actively seek out peers with similar beliefs, attitudes, and behaviors toward SU. Socialization pertains to the tendency for adolescents' beliefs, attitudes, and behaviors to be influenced by peers due to modeling and pressure to conform. In community-based adolescent samples, the relative importance of selection and socialization has been evaluated using both cross-sectional and longitudinal approaches.[7-9] Such investigations have suggested that peer influences vary for the two most commonly used drugs among adolescents[10]: alcohol and marijuana.

For alcohol, numerous community-based studies have found consistent evidence of a bidirectional, longitudinal relationship between adolescent and peer drinking, indicating significant transactional influences of both socialization and selection.[8,11] Community-based studies focused specifically on the longitudinal relationship between adolescent and peer marijuana use have produced less consistent results. One recent community-based study found evidence of both peer selection and socialization effects on adolescent marijuana use,[4] another found more robust evidence of socialization than selection,[12] and a third did not find unique effects of either selection or socialization on marijuana use.[13] Earlier studies examining composite measures of adolescent SU (including marijuana) have similarly produced mixed findings.[11,14,15] These inconsistent results demonstrate the need to examine the unique effects of peer processes separately on drinking and marijuana use in adolescents.

A key limitation of prior longitudinal research on peer socialization and selection has been the focus on community-based adolescent samples.[11-15] By targeting community-based samples, these longitudinal studies have predominantly provided information on factors related to adolescents' SU *initiation and SU maintenance in the absence of intervention*. Studies of treated samples are needed to elucidate the extent to which peer influences persist after receiving targeted intervention to reduce SU. Considering the well-established effects of parenting processes on both peer affiliation and SU, it would also be valuable to study the effect of parenting interventions on peer processes over time.

To our knowledge, only one study has specifically examined the relative influence of peer processes among treated adolescents, and no studies have examined peer processes among

adolescents whose parents have received targeted intervention. Becker and Curry[7] followed 106 adolescents who received a brief outpatient intervention for 12 months. The focal brief intervention (BI) was motivation enhancement therapy/cognitive behavioral therapy-5 sessions,[16] which was hypothesized to protect against both selection (via social support skills) and socialization (via peer refusal skills). Adolescents reported significant decreases in both marijuana and alcohol use over the 12-month study. Regarding peer influences, there was evidence of both selection and socialization for alcohol use, but only evidence of selection for marijuana use: effects were small in magnitude. One possible interpretation of these findings is that the BI was more effective in protecting against peer socialization than selection for marijuana use. However, it was not possible to determine whether the BI had a significant effect on peer processes without another treatment or no-treatment comparison group.

This study aimed to replicate Becker and Curry[7] in a sample of adolescents whose parents were randomized to receive 1 of 2 BIs (1 session each): the Family Check-Up (FCU),[17,18] a parent motivational intervention that provided parents with feedback about their adolescent's level of risk and specific parent factors linked to adolescent alcohol and drug use, and a time-matched parent psychoeducational (PE) comparison. The FCU was selected as the experimental condition, because it specifically targeted parent risk and protective factors associated with adolescent alcohol and drug such as communication and monitoring.[19,20] Unexpectedly, the parent trial did not find any differences between adolescents in the FCU and PE conditions[21] and neither condition resulted in reduced drinking or marijuana use across the three follow-up points.

Despite the lack of significant differences in the parent trial, the data remained conducive to testing the relative influence of peer processes among adolescents whose parents received targeted intervention. We extended prior work, and thus provide novel contributions to the literature, in 3 ways. First, we conducted the first empirical test of whether longitudinal peer processes varied as a function of the specific parenting intervention received. Because improvements in parenting have been shown to be protective against affiliation with deviant peers and because the FCU specifically targeted parental monitoring and communication,[19,20] we expected effects of peer selection and socialization to be weaker among adolescents who received FCU relative to those who received PE. Second, in addition to testing alcohol and marijuana in separate models, we conducted an exploratory test controlling for both substances in the same model. Simultaneously controlling for the effects of both substances is important, given that separate models may inflate estimates of selection and socialization when a high degree of adolescents use multiple substances.[13]

Consistent with Becker and Curry,[7] we hypothesized that separate models would find evidence of both selection and socialization for alcohol, but only evidence of selection for marijuana. In addition, we expected that there would be a less robust pattern of peer socialization and selection effects in the combined model. Finally, we had an exploratory hypothesis that FCU would reduce socialization and selection effects over time relative to PE, given that parents received more training in skills (eg, parental monitoring, parental communication) that should theoretically interrupt both socialization and selection mechanisms. We tested hypotheses controlling for covariates that have been found to influence SU patterns and response to intervention across at least 1 randomized trial (ie, Hispanic ethnicity,[22] age,[23,24] biological sex[23,24]). We focused on heavy drinking days as our measure of alcohol use because it was highly correlated with days of any alcohol use at all three timepoints (r's = 0.79-0.97, P's < .001) and because heavy drinking is associated with a greater risk of later SU disorders.[25]

Method

Participants

This analysis used data from a randomized trial of 2 parenting BIs for adolescent SU.[21] Research staff gave presentations about the study at local high schools. In addition, advertisements were placed in community settings including family and truancy courts, emergency departments, and mental health agencies. To qualify, adolescents had to be between 12 and 19 years of age and report alcohol or marijuana use within the past 30 days. A primary goal of the original trial was to determine if the parent BIs affected sibling outcomes[21]; as such, adolescents also had to have a sibling age of 12 to 21 and a legal guardian willing to receive a BI. Data from siblings are not included in this analysis. Table 1 presents the characteristics of the 109 adolescents (61% male, 28% Hispanic, mean age = 15.94, range 12-19 years) who comprised the analysis sample.

Procedures

Consistent with procedures approved by the University and Hospital Institutional Review Boards, adolescents and parents provided written assent/consent for participation. Enrolled adolescents and parents completed a comprehensive baseline assessment, prior to being randomized to condition. Urn randomization was used to balance on 2 variables: scores above or below the clinical cutoff on the externalizing subscale of the Child Behavior Checklist (score ≥ 65)[26,27] and scores above or below the clinical cutoff on the Adolescent Drinking Index (scores ≥ 16).[28] Condition assignments were revealed at the end of the baseline assessment via opaque envelopes. Parents and adolescents repeated assessment measures at 6- and 12-month follow-ups.

Intervention conditions

The FCU is a parent motivational BI that consists of an initial interview, a multi-method assessment, and a family feedback

Table 1. Sample characteristics, means, and standard deviations of study variables.

VARIABLE	UNITS	ASSESSMENT		
		BASELINE	6 MONTHS	1 YEAR
Study variables				
Heavy drinking days	Mean (SD)	3.08 (5.20)	4.52 (8.90)	4.66 (8.87)
	[min, max]	[0, 30]	[0, 39]	[0, 40]
Marijuana days	Mean (SD)	22.06 (29.68)	24.58 (32.05)	26.84 (33.85)
	[min, max]	[0, 90]	[0, 90]	[0, 90]
Perceived peer substance				
Involvement	Mean (SD)	1.83 (0.97)	2.04 (1.06)	1.90 (0.96)
Peer substance tolerance	Mean (SD)	2.45 (0.66)	2.59 (0.75)	2.64 (0.68)
Covariates				
Age	Mean (SD)	15.85 (1.30)		
Sex	Female (%)	39.45		
	Male (%)	60.55		
Ethnicity	Hispanic (%)	28.44		
	Non-Hispanic (%)	71.56		

There were no significant changes in any of the study variables (ie, heavy drinking days, marijuana days, perceived peer substance involvement) over time.

session.[17,29] Families completed the baseline self-report measures plus a 1-hour video-taped family assessment task (FAsTask) during which parents and adolescents discussed household expectations and rules around SU, how limits are set in the home, and how parents monitor their adolescents' behavior. Two independent coders rated these tasks and provided an individualized parent feedback report. Within 2 weeks, parents returned for a feedback session. Goals of the feedback session were to educate parents about risk for alcohol and other drug use among adolescents, support appropriate parental monitoring and limit setting, and motivate change in ineffective parenting behaviors using a motivational interviewing style.

Families receiving PE completed the same baseline self-report measures. Two weeks later (matching the FCU feedback), parents returned for an educational session during which a counselor reviewed a uniform set of informational material on alcohol and other drugs.

Measures

Days of heavy drinking and marijuana use. Number of standard drinks and quantity of marijuana consumed each day over the past 90 days was assessed during baseline, 6-, and 12-month follow-ups using the Timeline Followback method.[30] Daily responses were coded to indicate the total number of days the adolescent had engaged in heavy drinking (defined as 4+ drinks for girls and 5+ drinks for boys) and any marijuana use. Total heavy drinking and marijuana use days demonstrated good convergent validity with adolescent report on the Adolescent Drinking Questionnaire[31] and Drug Use Questionnaire[32] (r's > 0.90, P's < .001), respectively.

Perceived peer substance involvement. Perceived peer substance involvement (PPSI) was assessed via a measure developed by Chassin et al[33] containing 7 items about perceived peer SU and 7 items about perceived peer tolerance of use. This measure was used in the Becker and Curry[7] study that we aimed to extend. On a 6-point scale, adolescents reported how many of their friends (1 = *none* to 6 = *all*) engaged in occasional and regular heavy drinking and use of alcohol, marijuana, or other drugs. Adolescents also rated on a 6-point scale whether their close friends would strongly disapprove (1) to strongly approve (6) of their engaging in these same behaviors. The scales have demonstrated strong internal consistency in prior samples.[33,34] In the current sample, internal consistency (alpha) of the two sets of items was ⩾0.83 and ⩾0.80, respectively, across all three timepoints. Of note, the specific items assessing PPSI in heavy drinking and marijuana use were highly correlated at the 6- and 12-month assessments (r's > 0.90, P's < .001), indicating that separate examination of these items would not be justified. Thus, consistent with the initial validation study,[33] the two scales were standardized and averaged to obtain a composite score of PPSI.

Covariates. Adolescent age, sex, and Hispanic ethnicity were assessed at baseline and included as covariates.

Analysis plan

Primary hypotheses were tested using 2 cross-lagged panel models in Mplus V7.4 9[35] assessing 2 distinct SU outcomes: heavy drinking days and marijuana use days. We began with an a priori model including 3 sets of hypothesized associations: (a) stability paths over each interval (eg, we expected baseline days of use to predict 6-month days of use and baseline PPSI to predict 6-month PPSI); (b) concurrent associations at each timepoint (eg, we expected days of use and PPSI to be associated at baseline, 6 months, and 12 months); and (c) cross-lagged paths (eg, we expected baseline days of use to predict 6-month PPSI and baseline PPSI to predict 6-month days of use). In addition, we examined bivariate correlations between covariates (eg, age, sex, and ethnicity) and the outcome variables (eg, days of use and PPSI at all three timepoints) and added paths accounting for any significant associations. We also tested for the presence of indirect effects from baseline to 12-month measures (eg, from baseline to 12-month days of heavy drinking) using the MODEL INDIRECT command with bootstrapped standard errors. To improve model fit, we examined modification indices using the MODINDICES command and added any paths that exceeded 3.84 (corresponding with a 1-degree-of-freedom chi-square test). These adjustments led to the best-fitting model retaining all hypothesized paths.

Next, we conducted an exploratory multiple-group analysis with treatment condition as the grouping variable. This analysis examined whether any path estimates differed across the two treatment groups. Specifically, we first tested a model wherein all path estimates were allowed to vary across the FCU and PE groups. Then, we constrained all paths to be equal across groups. A significant decrease in fit according to a chi-square test would indicate significant differences in groups, and each path would be tested individually to confirm which paths differed. If no differences were found, we concluded that there were no differences by treatment condition and returned to the prior, more parsimonious model analyzing the entire sample.

As a final exploratory analysis, we tested the unique longitudinal associations between heavy drinking days and marijuana use days with PPSI in a single model. Following the same procedure as the separate models, we systematically tested stability, concurrent, and cross-lagged associations, while controlling for covariates. Across all analyses, significant paths from adolescent days of use to PPSI were viewed as evidence of peer selection, whereas significant paths from PPSI to adolescent days of use were viewed as evidence of peer socialization.

Attrition. Retention rates over the study were good at 6-month (n = 90; 86%) and 12-month (n = 86; 81%) assessments. Adolescents who completed versus dropped out by 6 or 12 months did not differ on baseline heavy drinking days, marijuana use days, PPSI, age, sex, or ethnicity. Missing data were hence treated as missing at random and accommodated using full information maximum likelihood. Heavy drinking and marijuana use days were log transformed to normalize distributions. Replication of the analyses using robust standard errors instead of transforming variables (MLR option in Mplus) yielded an identical pattern of results regarding effect sizes and significance.

Results

Table 2 depicts the associations among the outcome variables over time. Significant stability, concurrent, and cross-lagged associations were found among the study variables (eg, heavy drinking days, marijuana use days, PPSI) over time, supporting the need to control for these effects in the cross-lagged panel model.

Heavy drinking days and PPSI

The a priori model containing stability, cross-lagged, and concurrent associations, as well as putative covariates, demonstrated an adequate fit to the data: $\chi^2(8) = 13.25$, $P = .10$, root mean square error approximation (RMSEA) = 0.08, comparative fit index (CFI) = 0.98, Tucker-Lewis index (TLI) = 0.92. This model contained several paths to account for significant bivariate associations between the covariates and key outcome variables: (a) paths from age to 6-month heavy drinking, 6-month PPSI, 12-month heavy drinking, and 12-month PPSI; (b) paths from sex to 6-month PPSI; and (c) paths from ethnicity to 6-month heavy drinking, 6-month PPSI, and 12-month PPSI. No modification indices greater than 3.84 were found so we did not add any further paths.

We used this model as the basis for testing our exploratory hypothesis that results would differ by treatment group. When we constrained path estimates to be equal across treatment groups, there was no change in fit relative to the model that allowed all path estimates to freely vary, $\Delta\chi^2(25) = 20.82$, $P = .70$. Thus, we concluded that there were no significant differences by treatment condition and moved forward with the a priori model described above. As a final step, we dropped all paths that did not approach significance ($P < .10$) from the model. The final, most parsimonious model (see Figure 1) fit the data relatively well, $\chi^2(17) = 22.52$, $P = .17$, RMSEA = 0.06, CFI = 0.98, TLI = 0.96.

Marijuana use days and PPSI

The a priori model containing stability, cross-lagged, and concurrent associations, as well as putative covariates, demonstrated an adequate fit to the data: $\chi^2(7) = 10.79$, $P = .15$, RMSEA = 0.07, CFI = 0.99, TLI = 0.94. Paths in this model accounting for the effects of covariates included (a) paths from age to 6- and 12-month PPSI and (b) paths from ethnicity to 6-month marijuana days, 6-month PPSI, 12-month marijuana days, and 12-month PPSI. In addition, modification indices suggested that we add paths from sex to 6-month PPSI and 12-month marijuana days.

We used this model as the basis for testing our hypothesis that effects would vary by treatment condition. As with the

Table 2. Associations among study variables over time.

	BASELINE			6-MONTH FOLLOW-UP			12-MONTH FOLLOW-UP		
	HEAVY DRINKING DAYS	MARIJUANA DAYS	PPSI	HEAVY DRINKING DAYS	MARIJUANA DAYS	PPSI	HEAVY DRINKING DAYS	MARIJUANA DAYS	PPSI
Baseline									
Heavy drinking days	1								
	109								
Marijuana days	0.14	1							
	109	109							
PPSI	0.45*	0.17†	1						
	109	109	109						
6-Month follow-up									
Heavy drinking days	0.65*	0.25*	0.45*	1					
	90	90	90	90					
Marijuana days	0.31*	0.72*	0.32*	0.44*	1				
	90	90	90	90	90				
PPSI	0.44*	0.26*	0.45*	0.56*	0.39*	1			
	89	89	89	89	89	89			
1-Year follow-up									
Heavy drinking days	0.49*	0.22*	0.40*	0.83*	0.36*	0.52*	1		
	86	86	86	86	86	85	86		
Marijuana days	0.23*	0.70*	0.25*	0.40*	0.86*	0.32*	0.37*	1	
	86	86	86	86	86	85	86	86	
PPSI	0.29*	0.36*	0.42*	0.46*	0.37*	0.54*	0.49*	0.43*	1
	85	85	85	85	85	84	85	85	85

Abbreviations: PPSI, perceived peer substance involvement.
Pearson correlation is presented on the top row and N on the bottom.
*$P < .05$; †$P < .10$.

model for heavy drinking, there was no change in fit when all path estimates were constrained to be equal, $\Delta\chi^2(26) = 16.45$, $P = .92$, leading us to conclude that there were no differences between treatment conditions. As a final step, we dropped all paths that did not approach significance ($P < .10$). The final, most parsimonious model fit the data well, $\chi^2(23) = 23.07$, $P = .19$, RMSEA = 0.04, CFI = 0.99, TLI = 0.98 (Figure 2).

Heavy drinking days, marijuana days, and PPSI

The a priori model with both heavy drinking days and marijuana use days that included all hypothesized stability, cross-lagged, and concurrent associations fit the data reasonably well, $\chi^2(21) = 29.03$, $P = .11$, RMSEA = 0.06, CFI = 0.98, TLI = 0.95. This model contained all of the paths from covariates described

in the models above. Examination of modification indices did not lead to any additional paths.

As with the prior models, there was no change in fit when constraining path estimates to be equal across treatment groups, $\Delta\chi^2(31) = 32.13$, $P = .41$, indicating failure to confirm our exploratory hypothesis that there were differences between treatment conditions. Unlike the prior models, model fit was poor after dropping paths that did not approach significance ($P < .10$). We therefore retained all non-significant paths. The final best-fitting model is depicted in Figure 3.

Discussion

This study extended prior literature on the longitudinal relationship between adolescent SU and peer substance involvement. Whereas prior studies have predominantly focused on
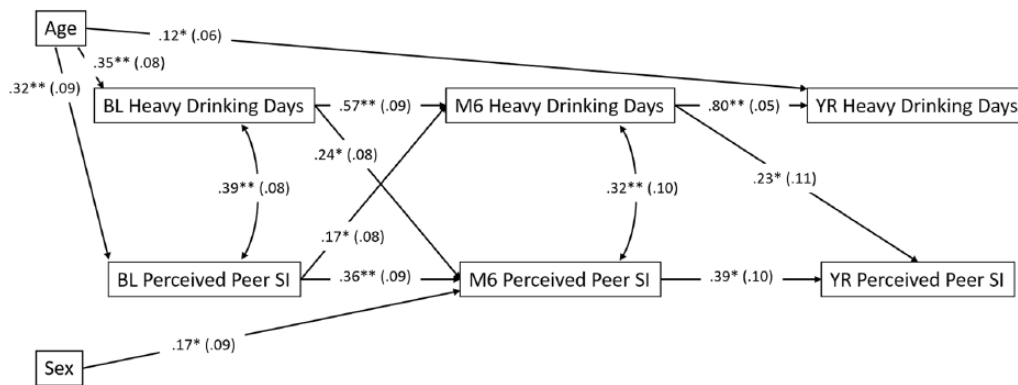
Figure 1. Cross-lagged model estimating the longitudinal relationship between adolescent heavy drinking days and perceived peer substance involvement. Significant direct paths from the best-fitting model are depicted. Parameter estimates are standardized values. Covariates (adolescent age, sex, ethnicity) were assessed at baseline. SI indicates substance involvement; BL, baseline; M6, 6-month follow-up; YR, 12-month follow-up.
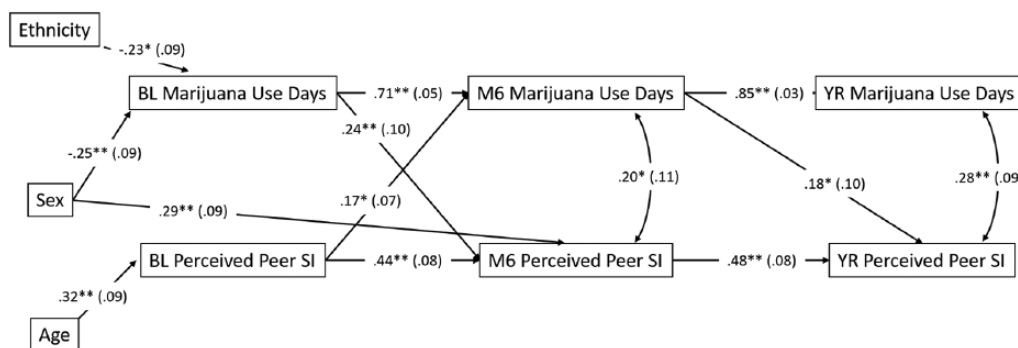N = 109.
*P ≤ .05; **P < .01.

Figure 2. Cross-lagged model estimating the longitudinal relationship between adolescent marijuana days and perceived peer substance involvement. Significant direct paths from the best-fitting model are depicted. Parameter estimates are standardized values. Covariates (adolescent age, sex, ethnicity) were assessed at baseline. SI indicates substance involvement; BL, baseline; M6, 6-month follow-up; YR, 12-month follow-up.
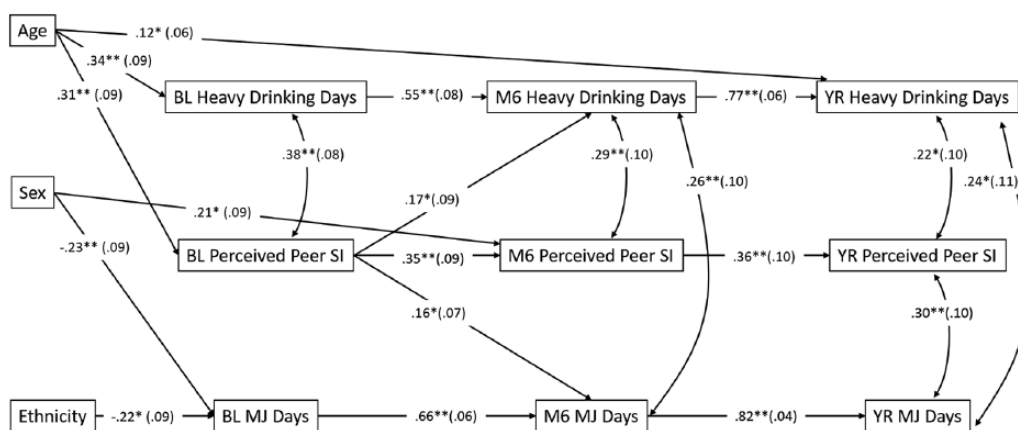N = 109.
*P ≤ .05; **P < .01.

Figure 3. Cross-lagged model estimating the longitudinal relationship among perceived peer substance involvement, adolescent marijuana days, and adolescent heavy drinking days. Significant direct paths from the best-fitting model are depicted. Parameter estimates are standardized values. Covariates (adolescent age, sex, ethnicity) were assessed at baseline. SI indicates substance involvement; BL, baseline; M6, 6-month follow-up; YR, 12-month follow-up.
N = 109.
*P ≤ .05; **P < .01.

community-based samples and interventions targeted solely toward adolescents, this study assessed longitudinal peer processes in a randomized clinical trial testing 2 brief parenting interventions for adolescents with SU.

Based on the only prior study of treated adolescents,[7] we expected to find evidence of both socialization and selection over time for alcohol, but only evidence of selection for marijuana. Our results partially supported this hypothesis. As

expected, for both heavy drinking and marijuana use, we detected consistent evidence of peer selection across the study period (from baseline to 6 months and 6 months to 1 year). By contrast, we only detected peer socialization processes immediately following the intervention (from baseline to 6 months). Counter to our hypotheses, we found no differences in peer processes by substance (eg, for marijuana vs heavy drinking). We also found no differences between the two treatment conditions on peer processes over time.

The divergence between this study and the only prior longitudinal study of perceived peer processes in treated adolescents could reflect a myriad of factors such as the differential effectiveness of the interventions (eg, the motivational enhancement therapy/cognitive behavioral therapy-5 session protocol in the prior study was associated with significant reductions in SU while there was no reduction in this study), the duration of the treatment protocol (eg, 5 sessions vs 1-2 sessions), and the testing of heavy drinking and marijuana in separate models. Another explanation for our divergence from prior work could be our focus on parenting interventions. An interesting direction for future research would be a comparison of adolescents who receive BI versus those whose parents receive BI, to determine the extent to which the treatment recipient affects peer socialization and selection processes.

This study also took the important step of controlling for both heavy drinking and marijuana use in the same model. Consistent with hypotheses, we found a less robust pattern of selection and socialization effects when simultaneously controlling for both heavy drinking and marijuana, relative to when examining each substance separately. In the composite model, only the early socialization effects remained—none of the selection effects were retained. This pattern is consistent with prior investigations suggesting that separate models overstate peer processes when adolescents use multiple substances.[13] Our results also extend prior literature by suggesting that this overstatement of results may apply particularly to the role of peer selection.

Finally, our inability to find differential peer processes as a function of treatment condition was disappointing. Although not entirely surprising given the lack of significant treatment differences in the original trial,[25] the lack of differences by treatment condition render it impossible to make causal inferences about the effect of treatment on peer processes. Nonetheless, our finding that there was a drop-off in peer processes over time (no significant peer processes between 6 and 12 months in the composite model) could indicate that both BI conditions—FCU and PE—had a protective effect on both peer selection and socialization. Our results also suggest that socialization effects were more resistant to treatment than selection effects. In other words, it is possible that the two BIs were more effective in addressing selection processes (eg, addressing adolescents' affiliation with peers) than socialization processes (eg, addressing adolescents' conformity to peers) in

the short term, and effective in protecting against both processes over the longer term. This interpretation requires replication in future work.

Limitations

This study had several limitations. It has recently been argued that cross-lagged panel designs assume that peer networks are stable and independent of observations, which could potentially confound socialization and selection processes.[36] Future work with larger samples should seek to employ social network analysis to examine peer effects to adequately disentangle socialization and selection.[37,38] In addition, as discussed in the primary outcome paper,[21] the sample size was limited due to the challenges of recruiting adolescents with problematic SU who had both a sibling and parent willing to participate. Our ability to detect significant effects within a small sample attests to the strength of the observed associations; however, the sample size may have limited our ability to detect differences by treatment condition.

Another limitation was reliance on adolescent self-report of perceived peer SU. Studies have found evidence that young people tend to overestimate their peer's SU at any given timepoint.[39] It is worth noting, however, that recent reviews[39] and commentaries[40] have argued that concerns about adolescent self-report of peer substance involvement are likely overstated. In the same vein, a recent study found that effect size estimates of socialization and selection effects were similar over time when using adolescent-reported versus peer-reported peer substance involvement,[36] suggesting that reliance on adolescent-reported PPSI would not necessarily change the interpretation of results.

Finally, the PPSI scale represented a composite of multiple substances and did not tease out specific effects for marijuana and heavy drinking. We could not test substance-specific measures in this study due to extremely high correlations between the peer heavy drinking and marijuana use items at later timepoints. It is feasible that substance-specific measures would have enabled us to detect different significant associations.

Conclusions

Despite these limitations, our results extend prior research by elucidating the longitudinal association between adolescent frequency of use and PPSI among treated youth. This was the first study to evaluate peer influences on adolescent SU within the context of a randomized clinical trial, thereby enabling us to explore whether treatment condition differentially affected peer processes over time. We were also able to extend prior research by testing both marijuana and heavy drinking in the same model to account for the high degree of adolescents who use multiple substances. Counter to expectations, our results revealed a similar pattern of peer influences on both heavy drinking and marijuana use over time and did not indicate

significant differences across treatment conditions. The lack of differences between treatment conditions limited our ability to make causal conclusions about the effect of treatment on peer processes. Nonetheless, our pattern of results suggested that the BIs may have been effective in protecting against peer processes over time.

Clinically, our findings indicated that, following receipt of BI, peer socialization processes directly after the intervention were more robust than peer selection processes. A clinical implication of this finding is that BIs for parents and adolescents might benefit from a more explicit focus on skills designed to disrupt peer socialization, such as substance refusal skills. Methodologically, our approach demonstrated the value of testing for multiple substances in the same model, as a means of preventing overly inflated estimates of significance, particularly regarding selection effects. Future studies of treated adolescent substance users with a no-treatment control condition, with putative covariates, and with measures of multiple substances are needed to determine whether BIs can disrupt peer processes and improve adolescent SU outcomes over time.

Author Contributions

Sara Becker generated the study hypotheses, led the manuscript writing process, and was responsible for synthesizing manuscript components. Kristine Marceau conducted the analyses and drafted the analysis plan, tables, and results sections. Lynn Hernandez was a Co-Investigator on the parent study, oversaw study data collection, and drafted the discussion section. Anthony Spirito was the Principal Investigator of the parent study, oversaw study procedures, and reviewed all manuscript components. All authors reviewed multiple drafts of the manuscript and approved the final version.

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