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The effects of general and alcohol-specific peer factors in adolescence on trajectories of alcohol abuse disorder symptoms from 21 to 33 years

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Abstract

Background—The present study examines whether general and alcohol-specific peer risk factors from age 10 to 18 are associated with longitudinal patterns of adult alcohol abuse disorder symptoms from age 21 to 33.

Methods—Using growth mixture modeling, trajectory groups of alcohol abuse disorder symptoms from age 21 to 33 were identified. We then examined the relationships between the identified trajectory groups of alcohol abuse disorder symptoms and respondents' own, a general negative peer factor, and an alcohol-specific peer factor (having drinking peers) in adolescence using pseudo-class Wald chi-square tests and multinomial logistic regressions.

Results—Four different trajectory groups of alcohol abuse disorder symptoms were identified: persister group (3%), decrease group (23%), escalator group (3%), and a no-disorder group (71%). Bivariate Wald chi-square tests indicated that adolescent binge drinking behavior and general and alcohol-specific peer factors differentiated the adult alcohol abuse trajectory groups. Multivariate multinomial logistic regression showed that the general negative peer factors distinguished those who later persisted in alcohol abuse from those who desisted (i.e., persister group vs. decrease group) during young adulthood, even after adjusting for respondents' adolescent binge drinking. On the other hand, associating with drinking peers did not distinguish these trajectories.

Conclusion—Alcohol-specific peer influences appear to influence alcohol abuse disorder symptoms in the early 20s, while general negative peer exposure in adolescence increases in importance as a risk factor for alcohol abuse disorder symptom persistence in the late 20s and the early 30s.

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Keywords

Alcohol abuse disorder symptoms; Young Adulthood; Adolescent peer factors; Conceptualizing general and outcome specific peer measures

1. Introduction

Young adulthood, a period of exploration of adult roles, is also a developmental period of increased risk for the development of alcohol problems, including alcohol abuse disorder. Results from the National Comorbidity Survey-Replication indicate that about 14% of young adults aged 18-29 experienced a lifetime alcohol abuse disorder (Kessler et al., 2005). In fact, compared to other age groups, alcohol abuse and alcohol dependence disorders are most prevalent among those aged 18-29 (Grant et al., 2004). Problematic drinking behavior among young adults has been associated with considerable losses in individual functioning and leads to negative consequences (Perkins, 2002), including injuries (Gmel et al., 2007; Mundt et al., 2009) and risky sex (Cavazos-Rehg et al., 2007).

Dynamic changes in the level or severity of drinking behavior are among the common features of alcohol problems in young adulthood. On average, alcohol use and abuse increases at the outset of young adulthood, and then steadily decreases starting at approximately age 24 (Bachman et al., 2002; Substance Abuse and Mental Health Services Administration, 2009). However, longitudinal studies have shown individual heterogeneity in patterns of change across the 20s in an array of drinking behaviors, including binge drinking and alcohol use disorder (Jacob et al., 2005; Jacob et al., 2009; Jackson and Sher, 2005; Maggs and Schulenberg, 2004; Chassin et al., 2002; Schulenberg and Maggs, 2002; Schulenberg et al., 1996). Yet, no study to date has examined predictors of individual heterogeneity in longitudinal patterns of alcohol abuse disorder symptoms among a community sample of young adults at ages 21 to 33. Thus, relatively little is known about factors that may distinguish various patterns of adult alcohol abuse disorder symptoms in the general population. Identifying specific longitudinal patterns of alcohol abuse in young adulthood, as well as their predictors, will help shed light on the course of alcohol abuse disorder development (Jackson and Sher, 2005).

The life course perspective (Elder, 1994; Mortimer and Shanahan, 2003) and other theories (Catalano and Hawkins, 1996) suggest that factors distinguishing different patterns of adult alcohol abuse disorder symptoms may stem from earlier developmental periods. Within these theoretical frameworks, it is hypothesized that experiences in childhood and adolescence may influence individuals' drinking behavior in young adulthood. Along this line, some research has shown that individual's adolescent drinking behavior predicts one's adult alcohol abuse or dependence problems (Hill et al., 2000; Guttmanova et al., 2011; Guo et al., 2000). Environmental factors in childhood and adolescence have also been linked to adult alcohol problems. Guo and colleagues (2001) examined the effect of each child and adolescent environmental precursor on adult alcohol abuse and dependence disorder at age 21. Maggs and colleagues (2008) reported that parental education increased the quantity of alcohol consumed at ages 23 and 33. Merline (2008) found that retrospective self-report of parental drinking predicted alcohol abuse at age 35. Although these prior studies shed light on the linkage between child and adolescent precursors and adult alcohol problems, they did not directly address the question of whether child and adolescent risk factors impact heterogeneity in trajectories of adult alcohol use problems. In addition, these prior studies have focused mostly on individual or family risk factors.

While literature that links child and adolescent environmental predictors to adult alcohol problems is relatively sparse, there is abundant information about the association between child and adolescent environmental factors and adolescent alcohol problems (for review, see Donovan, 2004; Hawkins et al., 1992; Maggs and Schulenberg, 2004). In particular, multiple studies have found that child and adolescent peer factors are potent predictors of adolescent substance use (e.g., Elliott et al., 1985; Curran et al., 1997; Bauman and Ennett, 1996; Fletcher et al., 1995). The most robust peer predictor of adolescent alcohol use is adolescents' association with alcohol-using (Curran et al., 1997; Bot et al., 2005; Griffin et al., 2000; Poelen et al., 2009) or delinquent (Li et al., 2002; Dishion and Owen, 2002) peers.

An emerging understanding in the literature is that the development of problem behavior likely stems from two sources: (1) general developmental risk and (2) factors that are related to the specific problem (Capaldi et al., 2008; Capaldi et al., 2009; Moffitt, 1993). It is hypothesized that general and problem-specific environmental risk factors may have different associations. Moffitt (1993) posited that exposure to general negative family environment (e.g., an adverse family-rearing context) predicts "life-course-persistent antisocial behavior," whereas an antisocial-behavior-specific peer environment (e.g., peers' antisocial behavior) is a crucial factor for predicting "adolescent-limited antisocial behavior." Such differential associations have also been reported in studies examining the impact of adolescent peer factors on adolescent alcohol problems. For example, in the analyses of adolescent boys' drinking, Capaldi and colleagues (2009) concluded that general peer environment (e.g., exposure to deviant peers) increases in importance as a risk factor for alcohol use later in adolescence, whereas alcohol-specific peer environment (e.g., associating with drinking peers) influences adolescent's early and later alcohol use in adolescence. Duncan and colleagues (2006) also reported that general peer environment (e.g., peer deviance) predicted change in adolescent alcohol use. Alcohol-specific peer environment (e.g., peer encouragement of alcohol use), on the other hand, predicted both initial level and longitudinal change, or slope, in adolescent alcohol use. These prior studies focused on the impact of adolescent general environmental influences and alcohol-specific influences on *adolescent* drinking. Building on these prior studies, the present study hypothesizes that these adolescent general and alcohol-specific peer influences may also differentially predict patterns of alcohol abuse disorder symptoms in adulthood. To our knowledge, no study to date has examined such a hypothesis.

Building on prior studies, the present study seeks to address the following four research questions: (1) What are the longitudinal patterns of alcohol abuse disorder symptoms in a community sample of young adults followed from age 21 to age 33? (2) Does adolescent binge (heavy episodic) drinking distinguish these patterns from one another? (3) Do child and adolescent negative general and alcohol-specific peer factors distinguish these patterns of adult alcohol abuse disorder symptoms over and above the influence of adolescent binge drinking? (4) Do general and alcohol-specific peer measures differentially predict these patterns?

2. Method

2.1. Sample

Data are from the Seattle Social Development Project (SSDP), a longitudinal study examining a broad range of substance use and behavior outcomes. In September 1985, all fifth-grade students ($N = 1053$) in 18 Seattle elementary schools serving children from high-crime neighborhoods were invited to join the longitudinal study, and 808 students (77%) consented to be a part of the SSDP sample. A similar acceptance rate has been reported in other studies recruiting children or adolescents (Ellickson and Bell, 1990; Elliott et al., 1981;

Thornberry et al., 1990). Analyses presented here included data collected during late childhood (age 10), adolescence (ages 11, 12, 13, 14, 15, 16, and 18), and young adulthood (ages 21, 24, 27, 30, and 33). The study was approved by the Human Subjects Review Committee at the University of Washington.

The sample is gender balanced (51% male) and ethnically diverse (47% European American, 26% African American, 22% Asian American, and 5% Native American). Fifty-two percent of study participants were eligible for the National School Lunch/School breakfast program between the ages of 10 and 13. Sample retention rates have remained high; out of those still living (23 participants were deceased by age 33), 92% ($n = 721$) of respondents participated in the age 33 interview.

2.2. Measures

2.2.1. Alcohol abuse disorder symptoms (ages 21 through 33)—Alcohol abuse at ages 21, 24, 27, 30, and 33 was assessed through computer-assisted, face-to-face interviews using the Diagnostic Interview Schedule (DIS) (Robins, 1981). Alcohol abuse disorder criteria included a failure to fulfill major role obligations due to alcohol consumption, recurrent alcohol use in hazardous situations, repeated alcohol-related legal problems, and repeated social or interpersonal problems due to alcohol consumption. An Alcohol Abuse Symptom Index was computed at each age as the sum of the number of DSM-IV criteria (American Psychiatric Association, 1994) met for alcohol abuse disorder criteria due to alcohol consumption. The measure ranges from 0 to 4 criteria; meeting one criterion is sufficient for a diagnosis of alcohol abuse disorder. For the analyses presented here, the measures were recoded down to 0 to 3 or above since very few participants exceeded this range.

2.2.2. Chronicity of binge drinking (ages 13 through 18)—Respondents' own binge (heavy episodic) drinking was assessed by students' reports of the number of times they had five or more drinks in a row in the past month at ages 13 to 16 and 18. For the analyses presented here, those having five or more drinks at least once were coded “1,” and “0” otherwise at each age. These dichotomized measures were summed into a single measure representing the number of measurement occasions that students drank five or more drinks in a row across adolescence. This summary measure ranged from 0 to 4 times (Mean = .42, SD = .79).

2.2.3. General negative peer environment (ages 10 through 18)—General negative peer environment was defined to be association with deviant or antisocial peers. Multiple items were used to assess respondents' three or four best friends' deviant/antisocial behavior from ages 10 to 16 and 18. An average of 11 items were available at each age and a total of 81 items were used to represent general negative peer environment from ages 10 through 18. Example items included: “[friend 1] did anything that could have gotten him or her in trouble with the police?”, “How often in the past year have you been asked by other teenagers to do things that could get you in trouble with your parents, the school, or the police?”, and “Have you ever been invited to join a gang?” Out of 81 items, 30 items were scored on a 4-point scale (e.g., 0 = NO!, 1 = no, 2 = yes, 3 = YES!), and 51 items were dichotomous (0 = No, 1 = Yes). All the items were standardized to ensure a common metric and equal weight across items. The standardized items were averaged to arrive at an overall scale score at each age, with means ranging from $-.01$ to $.01$ and standard deviation ranging from $.55$ to $.73$ across ages. Then the overall scale scores were averaged across ages into a single composite measure representing students' overall exposure to antisocial/deviant peers during late childhood and adolescence. For the present analyses, the combined summary measure was categorized into quartiles to facilitate interpretation.

2.2.4. Alcohol-specific peer environment (ages from 10 through 18)—Alcohol-specific peer environment was measured by the student's report of their three or four best friends' drinking behavior at ages 10 to 16 and 18. At least three items were available at each age and a total of 25 items were used to represent alcohol-specific peer environment from ages 10 through 18. Example items include; “Has [friend 1] tried beer, wine, or liquor when their parents didn't know about it?”, and “How many times in the past month has [friend 1] gotten drunk?” Three items were scored on a 4-point scale (0 = never/does not drink, 1 = once or twice, 2 = 3 or 4 times, 3 = 5 or more times), and the remaining items were dichotomous (0 = No, 1 = Yes). Items were standardized, averaged at each age, and then averaged across ages into a single composite measure representing students' average exposure to peers who drank during late childhood and adolescence. Again, the composite measure was categorized into quartiles to facilitate interpretation.

Respondents' gender was coded as 0 = female, 1 = male.

2.3 Analysis Strategy

To examine the possibility that some young adults in this sample had different trajectories of alcohol abuse disorder symptoms, we conducted a series of growth mixture models using Mplus 5.21. Growth mixture modeling (GMM), an extension of conventional growth curve modeling (Muthén and Muthén, 2000; Collins, 2006), uses iterative procedures to provide subgroup-specific estimates of growth factors (e.g., intercept and slope) and posterior probability of individuals' membership in each of the subgroups. Readers interested in more details about growth mixture modeling are referred to prior studies (see for example Muthén and Muthén, 2000; Ram and Grimm, 2009; Nagin and Odgers, 2010).

In line with prior research (Ram and Grimm, 2009), single-group models (e.g., intercept only, intercept and slope, intercept, slope, and quadratic, etc.) were first estimated to find the most parsimonious representation of change. Next, unconditional growth mixture models were estimated for two-, three-, four-, and five-class solutions (Feldman et al., 2009; Nagin and Odgers, 2010). The number of trajectory classes chosen was guided by consideration of multiple fit indices (Muthén and Muthén, 2000; Nagin and Odgers, 2010; Ram and Grimm, 2009), the conceptual interpretation, and prevalence of subgroups (Muthén and Muthén, 2000). Within-class variances were constrained to be equal across classes to define trajectories based on a pattern shared among individuals in a given class rather than being driven by variability. Statistically nonsignificant within-class variances were fixed to zero to facilitate the estimation process, when applicable. To avoid potential problems with local optima (Hipp and Bauer, 2006), the number of random starts was increased to 5000 and the second stage of optimization increased to 500. More details about fit statistics and growth mixture model-building strategies can be found elsewhere (e.g., Nylund et al., 2007; Tofighi and Enders, 2007; Muthén, 2004).

After selecting the optimal model representing trajectories of alcohol abuse in young adulthood, we examined the bivariate relationships between each predictor (respondents' adolescent binge drinking, general negative peer environment, and alcohol-specific peer environment) and the trajectory classes. Consistent with prior research (Clark and Muthén, 2009), the significance of these bivariate associations was examined using a pseudo-class Wald chi-square test. Finally, we re-estimated the model, regressing the trajectory class on all of the adolescent predictors and gender in multivariate multinomial logistic regression.

Missing data were relatively rare due to the high retention in the SSDP sample. Missingness was subsequently addressed using full-information maximum likelihood (FIML) estimation in Mplus (Schafer and Graham, 2002; Buhi et al., 2008; Acocck, 2005), and as a result,

across alcohol abuse symptoms, predictor variables, and the control variable, 91% of the original sample had available data for the final analysis.

3. Results

Single-group analyses supported a linear model for the growth mixture modeling of alcohol abuse symptoms; neither a quadratic term nor its variance was statistically significant and thus we did not include a quadratic term in following *k-class* growth mixture models. Each class trajectory model was carefully examined using the guidelines described above. In particular, we paid attention to the two-class solution for its parsimony and substantial class sizes (63% and 37%, respectively). However, a two-class model identified two different low disorder groups, having one group with a slightly higher number of alcohol abuse disorder symptoms. Neither of these two low disorder groups reported more than one alcohol abuse disorder symptom across the study points (the diagnostic threshold for alcohol abuse disorder according to the DSM-IV) (American Psychiatric Association, 1994), which led us to conclude that the substantive interpretation of these two classes is unclear and less interesting. A four-class trajectory model (see Table 1) was selected as the best fitting, conceptually meaningful model. This model presented the lowest adjusted BIC (SABIC) and an entropy value of 0.721, which represented an entropy value between medium (0.6) and high (0.8) entropy (Clark and Muthen, 2009). As described above, the initial four-class solution was examined that allowed the within-trajectory-group variability in symptom count to be estimated. The analysis showed that, while there was some variability in symptoms within classes at each time point, the variability in the longitudinal trend within classes was not significantly different from zero. Thus the final model was run fixing the within-class variance to zero.

Figure 1 shows the four unconditional-class trajectory groups. A large proportion of the sample had virtually no symptoms of alcohol abuse disorder (no-disorder, 71%), while a relatively small group started with virtually no symptoms of alcohol abuse disorder, but then showed development of symptoms (escalator group, 3%); at age 33, this group was estimated to have more than one alcohol abuse disorder symptom. Another group started with more than one alcohol abuse disorder symptom at age 21 and persisted in having more than one symptom (persistor group, 3%). The fourth group started with almost one alcohol abuse symptom and then reduced symptoms over time (decreaser group, 23%). Descriptive analyses were conducted to further understand the characteristics of each trajectory group. Lifetime abstainers (i.e., never said “yes” to “Other than a sip or two, have you ever drunk beer, wine, wine coolers, whiskey, gin or other liquor?” from ages 10 to 33), comprising 2.4% of the analysis sample, belonged fully to the no-disorder group. At age 21, none of the escalators reported any alcohol abuse symptoms and 84.6% of the persistors met at least one alcohol abuse disorder symptom. All the persistors met at least one alcohol abuse disorder symptom for at least three study points, and 80.8% of the persistors met at least one alcohol abuse disorder symptom for four or five study points. 77.5 % of the decreasers met at least one alcohol abuse disorder symptom from ages 21 to 24.

The second set of analyses examined predictors of the symptom group membership with focus on four comparisons across specific trajectory groups: (1) the no-disorder group vs. the persistor group, (2) the no-disorder group vs. the decreaser group, (3) the no-disorder group vs. the escalator group, and (4) the persistor group vs. the decreaser group.

We first tested for whether the symptom-group-specific means in average adolescent binge drinking were different from each other at a bivariate level, using pseudo-class Wald chi-square tests. The no-disorder group had the lowest mean in the adolescent binge drinking measure and the persistor group had the highest, while the decreaser and escalator groups

fell in between (See Table 2). The no-disorder group showed a lower mean in adolescent binge drinking than the persistor group and the decreaser group. However, the no-disorder group and the escalator group did not differ from each other in their average level of adolescent binge drinking. Finally, the persistor group reported a higher mean than the decreaser group in respondents' binge drinking.

We also examined whether adolescent general and alcohol-specific peer influences distinguished these groups in bivariate analyses, using pseudo-class Wald chi-square tests. The no-disorder group reported lower levels of general negative peer environment, and lower levels of alcohol-specific peer environment than the persistors, decreasers, and escalators. The persistor group reported a higher mean than the decreaser group across general negative peer environment and alcohol-specific peer environment.

In sum, bivariate analyses indicated that students' binge drinking behavior and peer factors during late childhood and adolescence were associated with the adult alcohol abuse trajectory classes. All of the differences were significant as expected with one exception: respondents' binge drinking during adolescence did not differentiate the escalator group from the no-disorder group at a bivariate level.

It is possible that these general and alcohol-specific adolescent peer influences simply operated through their influence on adolescent binge drinking. Thus, the next set of analyses examined whether general and alcohol-specific peer measures predicted adult alcohol abuse problems above and beyond respondents' adolescent binge drinking, and if so, whether general and alcohol-specific peer measures differentially predict these patterns of adult alcohol abuse trajectory. To examine these questions, all the main predictors and the gender variable were entered into a multivariate multinomial logistic regression as predictors of the trajectory classes where the latent trajectory classes were simultaneously formed.

As shown in Table 3, in the multivariate analyses, chronicity of binge drinking in adolescence elevated odds of being in the persistor group, relative to the no-disorder group (Odds Ratio = 2.56). Chronicity of binge drinking in adolescence was associated with a trend-level increased risk for being in the decreaser group, compared to the no-disorder group (Odds Ratio = 1.49). Chronicity of students' adolescent binge drinking lowered the odds of being in the decreaser group, compared to the persistor group (Odds Ratio = .58).

Importantly, over and above the influence of their own adolescent binge drinking, students' adolescent peer experiences were also shown to differentiate the trajectory groups of adult alcohol abuse disorder symptoms. The general negative peer measure was associated with increased risk for being in the persistor group, compared to the no-disorder group (Odds Ratio = 3.96), and lowered the odds of being in the decreaser group, compared to the persistor group (Odds Ratios = .29).

Even after adjusting for adolescent binge drinking, the alcohol-specific peer measure was also associated with increased risk for being in the persistor group, compared to the no-disorder group (Odds Ratio = 2.56), and for being in the decreaser group, relative to the no-disorder group (Odds Ratios = 1.8).

4. Discussion

This study examined the longitudinal patterns of alcohol abuse disorder symptoms during young adulthood (age 21 to 33) and linked the identified adult patterns to adolescent peer experiences as well as respondents' adolescent binge drinking behavior. We found four different longitudinal patterns of alcohol abuse disorder symptoms in a community sample of young adults: persistors, decreasers, escalators, and no-disorder group. Our findings

suggest that for many young adults, alcohol abuse disorder symptoms have either not been an issue in young adulthood or have been a temporary issue in their early 20s. However, for some young adults, symptoms are persistent or increase during young adulthood. Each of these three alcohol abuse groups may arise from different etiologies. Factors distinguishing the decreasers from those who do not desist are particularly important.

As expected, a young adult's own adolescent binge drinking behavior emerged as a strong predictor of his or her alcohol abuse symptom trajectory class. Yet, importantly, child and adolescent peer factors were also related to the adult alcohol abuse trajectories, even after adjusting for the respondents' adolescent binge drinking. We found that the general and alcohol-specific measures were differentially associated with the particular longitudinal patterns of alcohol abuse disorder symptoms. General involvement with antisocial peers distinguished those who persisted in alcohol abuse disorder from those who were more developmentally limited. On the other hand, associating with drinking peers predicted elevated alcohol abuse disorder symptoms in the early 20s (shared characteristics of persistor and decreaser groups), but it did not predict later divergence in this early heightened level. Taken together, these findings suggest that adolescent drinking peers have proximal influences on young adults' alcohol abuse disorder symptoms and that being exposed to deviant peers in general across adolescence increases in importance as a risk factor for alcohol abuse disorder symptoms for the later part of young adulthood.

These findings are consistent with Moffitt's theoretical discussion (1993) about the etiology of "life-course-persistent" and "adolescent-limited" antisocial behavior. The current findings found that the adolescent alcohol-specific measure did not predict later change in the early heightened alcohol abuse symptoms, which appears to be in contrast to prior studies examining alcohol involvement in adolescence (Capaldi et al., 2009; Duncan et al., 2006). However, as noted above, the prior studies examined alcohol use in adolescence. The difference in findings may stem from the difference in the developmental period that the current study focused on and/or measures that the current study investigated. Thus, the current findings are not necessarily in contrast to prior findings concerning adolescent alcohol use.

Surprisingly, none of the child and adolescent risk factors included in our analysis distinguished the no-disorder group from escalators. One possibility is that other child and adolescent factors not examined here, for example, general and alcohol-specific family factors or impaired functioning in cellular and molecular systems (Witt, 2010), such as disruption in brain activity due to alcohol use (Volkow et al., 2008; Zhu et al., 2004), may contribute to later onset alcohol abuse problems. A second possibility is that the triggers for newly elevated drinking in young adulthood may be more a function of that developmental period. For example, perhaps factors such as social influences and stressful life events experienced in young adulthood may be more predictive of later onset of alcohol abuse disorder symptoms than the child and adolescent factors examined here. Although the analysis is intentionally focused on the peer domain, these other potential predictors could be fruitful in future studies examining later onset alcohol abuse disorder.

We note two main limitations of our study. First, all the measures included in the present analysis relied on respondents' self-report. In particular, respondents' report on the behavior of their peers may be less accurate than peer self-reports. Second, growth mixture modeling is data driven. Generalization of growth mixture findings should be carried out with caution (Sher et al., 2011). In particular, small subgroup sizes for persistors and escalators may raise concerns about over-extraction of classes (Bauer and Curran, 2004). Statistically nonsignificant associations in analyses of the escalator group may be due to a small number of participants in that group. On the other hand, the persistor and escalator groups are

expected to be less common, given that they show elevated alcohol abuse disorder symptoms beyond the normative “peak” developmental period of alcohol problems in young adulthood. As these patterns of psychopathological behavior are expected to be unusual (Moffitt, 1993), we might naturally expect relatively few participants to belong to the corresponding classes. Also, the persister group is the same size and predictors were significant in a meaningful way. In sum, we acknowledge the potential power issue related to the small sample size of the escalator group. Keeping these study limitations in mind, these results should be replicated in similar longitudinal studies.

This study makes two main contributions to the existing literature. First, it capitalizes on prospective longitudinal data spanning multiple developmental periods from ages 10 through 33 and describes longitudinal patterns of alcohol abuse disorder symptoms among a community sample. Second, our study expands the literature by investigating differential impacts of earlier general and alcohol-specific peer measures on the identified patterns of adult alcohol abuse disorder symptoms. Using these data, the current study found that alcohol-specific peer influences appear to influence alcohol abuse disorder symptoms in the early 20s, while general peer environment risk increases in importance as a risk factor for alcohol abuse disorder symptoms in the late 20s and the early 30s.

Researchers should consider the possibility that the developmental course of alcohol abuse disorder symptoms may vary between individuals. An important next step in this line of research is to identify concurrent adult predictors distinguishing different patterns of alcohol abuse disorder symptoms. Our findings provide a somewhat hopeful message to practitioners: problematic alcohol involvement in young adulthood, particularly a persistent alcohol abuse problem, can be prevented by nurturing healthy peer networks among adolescents. Prevention efforts that focus on the overall health of friendships among adolescents are needed to promote healthier adult alcohol involvement and forestall the development of persistent alcohol problems among young adults.

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Figure 1.
Trajectory groups of alcohol abuse disorder symptoms.

Table 1

Fit indices for growth mixture models, with the selected four-class model indicated by shading.

No. of classes	BIC ^a	SABIC	Entropy	No. of parameters	Log likelihood	Adjusted LMRT ^{bc}	BLRT ^{bd}
1 class	4254.40	4238.53	--	5	-2110.52	--	--
2 classes ^e	4234.16	4208.76	0.512	8	-2090.39	<0.001	<0.001
3 classes	4236.69	4211.29	0.729	8	-2091.66	-- ^f	-- ^f
4 classes	4238.7	4203.77	0.721	11	-2082.65	<0.001	<0.001
5 classes	4255.71	4211.26	0.519	14	-2081.15	0.0560	^e ^g

Note

^a Decrease greater than 6 indicates "strong" improvement in fit (Raftery, 1995).

^b P-value

^c Adjusted Lo-Mendell-Rubin Likelihood Ratio Test — lower p-value indicates that the *k*-class model is preferable over the *k-1* class model.

^d Bootstrapped Likelihood Ratio Test — lower p-value indicates that the *k*-class model is preferable over the *k-1* class model.

^e Within-class variances were statistically significant so that they were not constrained to 0.

^f These two tests were not applicable, since within-class variances were specified differently across the 2-class model and the 3-class model. Within class variances for the 3-class model were fixed to 0 since they were not statistically significant.

^g Not all the bootstrap draws converged properly.

Table 2

Means of child and adolescent risk factors and the trajectory classes of adult alcohol abuse disorder symptoms.

Child and adolescent predictors	Trajectory classes			
	No-disorder group	Persistor group	Decreaser group	Escalator group
Chronicity of binge drinking	0.30	1.26 ^a	0.62 ^{a,b}	0.48
General negative peer environment	1.29	2.34 ^a	1.81 ^{a,b}	1.89 ^a
Alcohol-specific peer environment	1.31	2.25 ^a	1.80 ^{a,b}	1.91 ^a

^aWald tests indicated that the mean difference between the no-disorder group and each trajectory class was statistically significant at the .05 level.

^bWald tests indicated that the mean difference between persistors and decreasers was statistically significant at the .05 level.

Table 3

Multinomial logistic regression predicting the adult alcohol abuse trajectory classes.

Panel A: Alcohol abuse trajectories compared to no-disorder group ^a				Panel B: Decreaser group vs. persistor group ^a			
Trajectory groups	Exp(β)	95% CI ^b		Exp(β)	95% CI ^b		
		Lower bound	Upper bound		Lower bound	Upper bound	
Persistor group							
Chronicity of binge drinking	2.56 *	1.54	4.25	Decreaser group	0.58 *	0.37	0.90
General negative peer factor	3.96 *	1.42	11.09		0.29 *	0.10	0.83
Alcohol-specific peer factor	2.56 *	1.27	5.17		0.70	0.34	1.44
Decreaser group							
Chronicity of binge drinking	1.49 †	1.00	2.23	Escalator group			
General negative peer factor	1.17	0.87	1.56				
Alcohol-specific peer factor	1.80 *	1.31	2.47				
Escalator group							
Chronicity of binge drinking	0.95	0.29	3.18				
General negative peer factor	3.04	0.77	12.10				
Alcohol-specific peer factor	1.70	0.42	6.86				

^a Adjusted for gender.

^b CI: confidence interval for odds ratios.

* p<05.

† <10.