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Early Onset Problem Behaviors and Alcohol, Tobacco, and Other Substance Use Disorders in Young Adulthood

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Abstract

Objective—Ten early onset problem behaviors were used to prospectively predict alcohol, tobacco, cannabis, and cocaine disorders in young adulthood (mean age=28.6 yrs.) for a U.S. community sample of 671 participants.

Method—Data from a longitudinal study of participants who were recruited from high schools during adolescence and followed into young adulthood were used to evaluate prospective associations. The relationship between early onset problem behaviors, reported when participants were age 16 years, and psychiatric diagnoses assessed in young adulthood was tested. Structural equation models were used to evaluate both generality and specificity hypotheses regarding relationships between early onset problem behaviors and young adult disorders.

Results—Findings supported the specificity hypothesis in that “like” early onset problem behaviors significantly predicted “like” young adult outcomes (e.g., early cocaine use predicted cocaine disorders). Furthermore, eliminating such “like” predictors in regression equations resulted in a 36% reduction in the amount of variance accounted for by the equation. The generality hypothesis was also supported in that a larger number of early onset problem behaviors strengthened the prediction of young adult disorders beyond the “like” attribute, and a dose-response pattern indicated that additional early onset problem behaviors increased the probable occurrence of a young adult disorder.

Conclusions—A comprehensive framework relating early onset problem behaviors to young adult substance disorders will require the integration of both generality and specificity hypotheses, and a developmental orientation focused on the unfolding of mediating and moderating processes. Early screening of multiple, rather than single, early onset problems is also discussed.

Keywords

early-onset; young adulthood; problem behaviors; substance use disorders

1. Introduction

Early onset problem behaviors in childhood and early adolescence, such as alcohol use, tobacco use, and delinquent behaviors (e.g., stealing, truancy), have been commonly associated with the subsequent development of alcohol and other substance abuse disorders,

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mental health disorders, and criminality in early adulthood (Breslau et al., 1993; Grant and Dawson, 1997; Sampson and Laub, 1993). For example, Grant and Dawson (1997) reported that individuals who began drinking before age 15 were four-times more likely to subsequently become alcohol dependent than those who did not drink alcohol before age 21. Similarly, increased risk for nicotine dependence has been reported for early onset tobacco use (Breslau et al., 1993). Although the causal versus "marker" role of early onset problem behaviors remains indeterminate (Chen et al., 2009), research findings concur that early onset problem behaviors are among our most commonly identified risk factors for a range of outcomes related to subsequent substance abuse and mental health problems. Furthermore, these early onset problem behaviors are often the focus of prevention programs to delay initiation of problem behaviors (Kandel and Yamaguchi, 2002; Spoth et al., 2009).

With the exception of the considerable research conducted on childhood antisocial behaviors as prospective predictors of adolescent and adult alcohol and other substance use and abuse and criminality (Windle, 1990; Zucker, 2006), the vast majority of substance use research on early onset problem behaviors has been substance-specific (e.g., early onset alcohol use and subsequent alcohol disorders; early onset tobacco use and subsequent nicotine dependence). Although valuable, the substance–specific approach may also constrain the nature of other valuable research questions for several reasons. First, similar to problem behaviors observed in adolescence and young adulthood (Donovan and Jessor, 1985), it is possible that early onset behaviors tend to covary, thereby forming more of a syndrome than a substance-specific occurrence, and the syndrome poses a generalized risk for a range of adverse outcomes rather than a substance-specific risk. McGue and Iacono (2005) used this more generalized formulation and reported that a single latent variable of early onset problem behaviors prospectively predicted externalizing disorders at ages 17 and 20 years. Conversely, it is possible that early onset behaviors are highly specific and only predict subsequent behaviors similar to the early onset behavior (e.g., early onset cannabis use only predicts subsequent cannabis disorders). Second, it is possible that early onset behaviors in one domain (e.g., truancy) may impact subsequent behaviors in other domains (cannabis use) by influencing, and being influenced by, common factors (e.g., deviant peers). Third, it is possible that collectively (i.e., summed across behaviors), early onset problem behaviors may function in a dose-response manner such that increases in the number of early onset behaviors is associated with increases in the probable occurrence of subsequent disorders and/or the severity of symptoms.

The nature of the relationship between early onset problem behaviors and prospective substance abuse is of importance both for etiology and intervention. If the data are more supportive of substance–specific findings (e.g., early alcohol onset and alcohol dependence) rather than a generalized pattern, then probable causal (etiologic) factors and prevention-intervention targets should correspond with the specificity hypothesis. By contrast, if the data are more supportive of a generalized pattern, then probable causal and prevention-intervention targets may need to focus more on common risk factors (e.g., behavioral under-control, peer deviance) that influence multiple substance abuse outcomes. The examination of dose-response relationships would also be informative as to whether the number, or density, of early onset problem behaviors contributes more to the prediction of prospective outcomes than does knowledge of single early onset indicators. If a dose-response relationship is supported, then "early onset" screening procedures could be usefully applied to identify and intervene with those children/early adolescents at highest risk for the development of substance abuse problems and disorders.

The objectives of this study were to use relatively long-term prospective data (from childhood to age 28) to study both the specificity hypothesis (i.e., that early onset childhood behaviors principally predict "like" behaviors by age 28) and the generality hypothesis (i.e.,

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that early onset behaviors co-vary and jointly predict multiple substance abuse outcomes by age 28). Note that our reference to the specificity hypothesis refers to disaggregated, individual early onset problem behaviors (e.g., marijuana use, stealing property) rather than higher-order aggregated constructs such as early onset substance use and early onset antisocial behaviors. We focus on the disaggregated scores to test the specificity hypothesis because of the neurobiological literature suggesting the specificity of substance-receptor relationships (e.g., tobacco with nicotinic acetylcholine receptors; Koob and Le Moal, 2006) and the delinquency/antisocial behavior literature suggesting multiple subtypes of antisocial behavior (e.g., property versus aggression offenses; Loeber and Hay, 1997). We believe the study to be unique in providing a prospective assessment of *multiple* rather than single early onset problem behaviors and their prospective influences on *multiple* rather than a single disorder outcome. Such findings on multiple early onset influences and multiple outcomes may more closely parallel the nature of the etiology of substance use disorders and be more relevant to what clinicians see in their daily practice.

To address these hypotheses required the measurement of both multiple early onset problem behaviors (in this study ten distinct behaviors), and multiple "outcome" variables that are "like" with regard to the early onset variables (e.g., early cocaine use and cocaine disorders). To accomplish this we measured lifetime disorder status for alcohol, tobacco, cannabis, and cocaine disorders, as well as the severity of symptoms for these four disorders because much of the recent literature, and preliminary information of DSM-V (Regier et al., 2009), indicate the value of conceptualizing and measuring psychiatric phenotypes in both discrete (i.e., diagnostic status) and continuous (dimensional) forms (Widiger and Samuel, 2005). Due to its flexibility and ability to specify and test a range of models, we used structural equation modeling to evaluate both the specificity and generality hypotheses for early onset problem behaviors (McGue and Iacono, 2005). We also evaluated whether the number of early onset problem behaviors functioned in a dose response manner in their associations with age-28 lifetime substance abuse outcomes.

2. Methods

2.1 Participants

The data used in this study were collected as part of a larger, multi-wave panel design focused on risk factors and adolescent substance use and mental health. We refer to the study by the acronym LAT, which stands for Lives across Time: A Prospective Study of Adolescent and Adult Development. The initial principal objective of the LAT was to assess the onset, escalation, maintenance, and continuation (or termination) of alcohol and other substance use among 1,205 teens during the high-school years (with four waves of assessment at six-month intervals) in relation to a range of risk factors (e.g., temperament, peer substance use, family history of alcoholism). Data were collected within the adolescents' high school settings and the overall student participation rate was 76%. The sample consisted of high school sophomores (53%) and juniors (47%) recruited from two homogeneous, non-denominational suburban public high school districts (a total of three high schools) in Western New York, the average age of the respondents at the first occasion of measurement was 15.54 years (SD = 0.66), the sample was roughly equal with regard to males and females, and 98% were white. Sample retention across the first four waves of measurement was uniformly high, in excess of 90%.

There was approximately a six-year gap between the Wave 4 assessment in adolescence and the Wave 5 data collection that occurred when the average age of the young adults was 23.5 years. The Wave 5 assessment was modified from Waves 1–4 in that data collection changed from a large group, in-school survey format to individual interviews of the young adults and their mothers and fathers in their homes. Greater detail on the Wave 5 assessment

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is provided elsewhere (Windle et al., 2005), but briefly, at least one of three possible participants from 940 households participated at Wave 5, including 827 young adults. A similar protocol was used at Wave 6 (mean age of 28.7 yrs.) as Wave 5 and retention rates were similar as for Wave 5; note, some participants (approximately 70) participated at Wave 6 but did not participate at Wave 5 and vice-versa—participants were not excluded from further participation in the study if they missed a wave of data collection. Extensive multivariate and univariate attrition analyses were also conducted at Wave 5 and Wave 6 regarding the participants versus non-participants. For example, participants versus non-participants in this study did not differ significantly with regard to variables assessed during adolescence (e.g., family income, parents' educational level, adolescent's alcohol, cannabis, or other drug use) or at Wave 5 (e.g., young adult's income, prevalence of alcohol, cannabis, or other substance abuse disorders). Our conclusion was that minimal systematic differences existed among the retained sample used in this study, relative to the original sample. Inclusion in this study required participants to have completed the early onset problem behavior measure at Wave 2 and to have provided lifetime psychiatric data; this resulted in a sample size of 671.

2.2. Procedure

During the adolescent phase (i.e., Waves 1–4), subsequent to receiving informed consent both from a parent and the target adolescent, a trained survey research team administered the survey to adolescents in large groups (e.g., 40–50 students) in their high school setting at each wave. The survey took about 45–50 minutes to complete and subjects received $10.00 for their participation. Confidentiality was assured with a U.S. Department of Health and Human Services Certificate of Confidentiality. The young adult interviews at Waves 5 and 6 were conducted via one-on-one interviews either in the subjects' homes or at the host institute of the investigators. Subjects were paid $40 to complete an interview that lasted approximately two hours. Computer-assisted personal interviews were used to collect data.

2.3 Measures

Early onset problems—Ten early onset problems were used in this study and included age when first got drunk, smoked first cigarette, first tried marijuana or hash, first tried cocaine or crack, first had contact with police due to a delinquent act, first deliberately damaged public or private property, first stole something from a store, first started a fist fight, first skipped school, and first ran away from home overnight. Adolescents were asked to indicate whether they had ever participated in each of the ten problem behaviors and, if an affirmative answer was given to a question, respondents were then asked to specify the age at which they first participated in the behavior. Similar to other studies (McGue and Iacono, 2005), for nine of the ten problems we used below age 15 as the cut-off for early onset; due to low base rates and a typically later onset, we used below age 17 as the cut-off for early onset of cocaine use.

Alcohol and other substance abuse disorders—*DSM-IV* disorders were derived in the young adulthood interviews via the World Health Organization Composite International Diagnostic Interview (WHO-CIDI; WHO, 1997). Reliability data for the WHO-CIDI have been reported (WHO, 1997) and the disorders used in this study included alcohol, marijuana, and cocaine disorders. This interview instrument, or ones similar to it with minor modifications, have been used extensively in epidemiologic studies in the United States (Kessler et al., 2005) and internationally (Andrade et al., 2003).

Young adult tobacco dependence—The Tobacco Dependence Screener (TDS; Kawakami et al., 1999), a 10-item questionnaire for the screening of tobacco/nicotine dependence, was used to estimate lifetime symptoms of tobacco/nicotine dependence and a

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provisional diagnosis. The reliability and validity of the TDS were assessed in three samples of smokers in Japan (Kawakami et al., 1999). Cronbach's alpha for the TDS ranged from .74 to .81 among the samples and receiver operator characteristic analyses indicated that the TDS had a better screening performance for *ICD-10*, *DSM-III-R*, and *DSM-IV* diagnoses than did the Fagerstrom Tobacco Questionnaire. The TDS score significantly and positively correlated with severity of the nicotine dependence diagnoses, carbon monoxide levels, number of cigarettes smoked per day, and years of smoking.

2.4 Data Analyses

To test the generality-specificity relationships, structural equation models (SEMs) were specified, estimated, and evaluated with the Mplus Program (Muthén and Muthén, 1998–2008) using weighted least squares estimators for the categorical dependent variables and maximum likelihood estimators with robust standard errors (MLR) for continuous dependent variables. The MLR estimates are robust to non-normality and standard errors are computed using sandwich estimators. To evaluate model fit, we used the Root Mean Square Error of Approximation (RMSEA; Browne and Cudeck 1993) as our primary index of model fit because some (Loehlin 1998) have suggested that it is currently the best model fit index, and it also may be advantageous with the robust maximum likelihood estimator used in this study. Close fit for the RMSEA index is indicated for values less-than .05, fair fit for values ranging from .05 to .08, and poor fit for values greater than .10. A secondary fit index, the Comparative Fit Index (CFI), was also reported with values greater-than-or-equal-to 0.95 considered good fit and those between .90 and .95 considered reasonable fit (see Marsh et al., 2004; Brown, 2006). Hu and Bentler (1999) proposed a criterion of .95 for the CFI to indicate good fit; however, as noted by others (Marsh et al. 2004), this value may be too stringent for more complex models and may result in the rejection of well-fitting models, as well as impose constraints on replication.

3. Results

3.1 Prevalence of early onset behaviors

The prevalence of early onset problem behaviors indicates a range of occurrence from 6% for running away from home to 51.7% for early onset cigarette use (Table 1). Summing across all early problem behaviors, about 24% of the sample reported zero early onset behaviors, 22% reported one early onset behavior, 18% reported two early onset behaviors, 14% reported 3 early onset behaviors, and 22% reported 4 or more early onset behaviors. The prevalence of lifetime disorders for the four substances used in this study were: alcohol (40.7%), tobacco (12.8%), marijuana (21.9%), and cocaine (4.9%). Table 2 summarizes the bivariate associations between early onset problem behaviors and young adult substance disorders via odd ratios. The data in Table 2 indicate statistically significant bivariate associations among the two domains of variables, though there is some variation in the relative strength of the associations.

3.2 SEM findings

To evaluate the specificity of prediction (e.g., early onset alcohol use predicts alcohol disorders) an SEM was specified in which all ten early onset problem behaviors were used as predictors of all four substance disorders. The correlation matrix for these data is provided in Table 3. Descriptives statistics for the young adult outcomes demonstrate some deviations form normality that typifies substance use data (Table 4; Buu et al., 2011). Because the findings were highly similar for clinical diagnoses and symptom counts, only the findings for symptom counts are provided in Table 5. For each of the substance use disorders, specificity was supported because each early onset behavior significantly predicted the subsequent "like" disorder symptom count. That is, early onset alcohol use

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significantly predicted alcohol disorder symptoms, early onset cigarette use significantly predicted tobacco dependence symptoms, early onset marijuana use significantly predicted cannabis disorder symptoms, and early onset cocaine/crack use significantly predicted cocaine disorder symptoms. Furthermore, in additional analyses (not displayed), the exclusion of the early onset "like" behavior in the structural equations (e.g., removing early onset alcohol from the equation predicting alcohol disorder symptoms) resulted in an average reduction of 39% of the variance accounted for by the early onset problem behaviors on substance symptom outcomes. In addition to the specificity predictions, other early onset behaviors also predicted young adult substance disorder symptoms. For example, early onset alcohol use also significantly predicted symptoms of tobacco, cannabis, and cocaine disorders. The early onset delinquent behaviors of starting a fight and engaging in property damage were also significant predictors of alcohol disorder symptoms. Total variance accounted for across the early onset predictors for the substance disorder symptoms ranged from 9% to 16%. Thus, in general, it was the case that "like" and "unlike" early childhood substance use behaviors predicted "like" and "unlike" young adult substance use disorders. Only the early onset delinquent behaviors of property damage and starting fights significantly predicted the alcohol disorder symptoms, but not the other substance disorder symptoms. We also re-ran these analyses controlling for gender and findings were unaltered, though gender was a statistically significant predictor.

An SEM was also specified for a single latent variable (LV) of early onset problem behaviors with 10 manifest indicators predicting the four substance use disorders. Figure 1 summarizes the findings for this model which provided fair, or adequate, overall model fit (χ^2 =230.33, 71*df*, $p < .001$; *CFI*=.91, *RMSEA*=.058 with a confidence interval from .050 to .066). The parameter estimates corresponding to "factor loadings" were all statistically significant ($p < .001$), though there was some variation in the magnitude of the loadings which ranged from .39 to .89. The early onset problem behaviors LV significantly predicted all four disorders, with standardized estimates ranging in magnitude from .40 to .55. The overall amount of variation accounted for by the early onset LV was 17% for alcohol disorder, 16% for tobacco dependence, 28% for cannabis disorder, and 31% for cocaine disorder. Thus, the LV model accounted for more variation on the outcome variables than did the individual predictor model whose data were presented in Table 5. Another SEM (not included) was specified and evaluated that included the prediction of a single young adult Substance Disorder factor with manifest indicators of disorders for alcohol, tobacco, cannabis, and cocaine, and the early problem behavior LV. Factor loadings for alcohol, tobacco, cannabis, and cocaine were .68, .56, .86, and .86, respectively. This model also fit the data well with a parameter estimate of .63 ($p < .001$) between early problem behaviors and Substance Disorder, accounting for approximately 40% of the variance.

3.3. Dose-response findings

To evaluate a dose-response relationship between the number of early problem behaviors and the probability of an occurrence of a lifetime disorder, the 10 early problem behaviors were categorized from 0 to equal-to-or-greater-than 6 and cross-tabulated with the percentage of occurrence of each substance disorder. Chi-square statistics were statistically significant ($p < .001$) for all four comparisons and Figure 2 provides a bar chart for early problem behavior categories by the occurrence of alcohol disorders. Hence, for children with zero early onset problem behaviors, about 19% developed an alcohol disorder by young adulthood; in contrast, for those children with greater-than-or-equal-to (GE) 6 early onset problem behaviors, about 81% developed an alcohol disorder by young adulthood. The distribution of GE 6 was such that the vast majority (92%) had between 6 and 8 early onset problems. The general direction indicated in Figure 2 is one of linear increases in the occurrence of alcohol disorders as early onset problem behaviors increase, but a substantial

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increase occurs at the GE 6 early onset problem behaviors. Similar general, though not quite as step-like, tendencies in the dose-response relationship occurred for other substances with a higher prevalence of early problem behaviors associated with higher rates of disorder, especially in the 5-to-6 or more range.

4. Discussion

4.1 Generality-specificity

The prospective findings of this study are consistent with prior research in supporting long-term associations between early onset problem behaviors and substance abuse and dependence in young adulthood (Breslau et al., 1993; Grant and Dawson, 1997). Furthermore, findings using structural equation modeling with aggregated latent variables (LVs) of early onset problem behaviors and substance disorders were highly consistent with those of McGue and Iacano (2005). However, statistical tests of the specificity of relationships were also supported in that “like” early onset predictors (e.g., early onset cocaine use) were consistent predictors of young adult disorders (e.g., cocaine disorders). Importantly, removing these “like” early onset predictors from the regression equations resulted in a substantial decrease in the amount of variance accounted for by the early problem behaviors on substance outcomes (an average reduction of 39% in the magnitude of effects). Therefore, the specificity of these “like” early onset predictors are providing important, unique information in the prediction of young adult substance abuse disorders that is not totally accounted for by the other nine early onset problem behaviors.

This is not to suggest that the other early onset problem behaviors were not also significant predictors of the young adult substance abuse disorders. Data from both the disaggregated (individual early onset predictors) and aggregated, LV models supported the role of other predictors in addition to the “like” predictors. For instance, for the disaggregated models, early onset alcohol use was not only a significant predictor of young adult alcohol disorders, but also tobacco, cannabis, and cocaine disorders. Early onset cigarette use was also significantly associated with young adult cannabis disorders. These multiple, early onset problem behavior associations with later substance disorders are consistent with notions of the Gateway Theory of substance use progression (Kandel and Yamaguchi, 2002), though a definitive test of this theory was not provided in this article. For the aggregated LV models, it was evident that a substantially larger percentage of variance was accounted for in the substance abuse outcomes than with the disaggregated early onset problem behavior models. The common, or shared, variance of the 10 early onset problem behaviors represented by the LV may have provided greater reliability and fidelity of measurement, as well as extracting the shared features of early onset problem behaviors that predicted young adult substance disorders. Similarly, the bivariate findings indicated ubiquitous significant associations among the individual early onset problems and the young adult substance disorders.

Collectively, the findings support the importance of both the specificity hypothesis for “like” early onset problem behavior-young adult substance abuse outcomes, *and* the generality hypothesis in that the aggregated early onset problem behaviors increased prediction over the specific-predictor only model. The dose-response models also indicated a general directionality of significant increases in the occurrence of each of the disorders as the number of early onset problem behaviors increased, with a major “jump” in occurrence of alcohol and cocaine disorders for those adolescents who reported six or more early onset behaviors. Hence, based on these findings, the early onset problem behaviors may reflect *both* general underlying vulnerabilities (e.g., behavioral under-control; deviance proneness) and substance-specific vulnerabilities (e.g., intensity of reaction to alcohol). Furthermore, as indicated by the data, not all substance use disorders may have the same predictors. Such an interpretation is consistent with and parallels other research findings on substance use and

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abuse that have indicated not only common neural circuitry for the dopamine-related rewarding aspects of various substances, but also specific genes, receptors, and neurobiological mechanisms for specific substances (Koob and Le Moal, 2006). Therefore, perspectives based on the merger of *common and specific contributors* to substance abuse disorders may provide stronger explanatory frameworks not only for the specific influence of early onset problem behaviors, but in general for substance abuse disorders (Zucker, 2006).

4.2. Prevention implications

The prevention implications of the findings of this study are threefold. First, consistent with prior findings, early onset problem behaviors can be potent prognosticators of alcohol and other substance abuse disorders in young adulthood (Breslau et al., 1993; Grant and Dawson, 1997) and thereby serve as potential early behavioral targets of prevention programs. However, the findings suggest the advantages of recognizing and potentially targeting the prevention of several, interrelated early onset problem behaviors rather than a singular focus (Biglan et al., 2004). Second, the marker versus causal status of early onset problem behaviors (Chen et al., 2009) remains to be addressed through prospective intervention and etiologic studies that identify the mediators and moderators by which early onset problem behaviors increase the risk for young adult substance disorders. With enhanced knowledge of these mediators and moderators, prevention targets could focus on these intervening factors and mechanisms to prevent the expression of the early onset problem behaviors and/or disrupt developmental patterns during adolescence and young adulthood. Third, with these ten items, or a similar multi-problem early onset measure, rapid clinical screening for high risk behavior is possible. Clearly those individuals in this study with a high rate (6 or more) of early onset problem behaviors were at substantial risk of developing a substance abuse disorder in young adulthood. The use of such a composite, multi-problem early onset index that is easily administered may be of importance in screening contexts (e.g., emergency rooms, offices of pediatricians).

4.3. Limitations and conclusions

The study has limitations in that the majority of the sample was white and from middle-class income families, thereby limiting the generalizability of the findings to other racial, ethnic, and income groups. The assessment of early age of onset was also retrospective (i.e., assessed at average age 16.0 yrs.), though the shorter interval for the recall from ages 16 to childhood/early adolescence is likely to yield more reliable information than recall over longer time intervals (Parra et al., 2003). Although our below age 15 cut-off for designating risk is largely supported in the literature (McGue and Iacono, 2005), it is possible that different age cut-points (e.g., earlier age) may have yielded somewhat different findings and some of the delinquency predictors may have been more prominent predictors in the equations; it is also possible that some of these earlier onset antisocial behaviors preceded the onset of substance use, but our data did not lend itself well to an analysis of sequence data. Given the nonnormality distributions of the symptom count data, it is possible that other statistical models such as the zero-inflated Poisson regression model may have been beneficially used (Buu et al., 2011). Finally, the sample size was underpowered to extend the analyses to other early onset problem behaviors (e.g., inhalant use; heroin use) because the prevalence of these disorders in young adulthood was not large enough to permit meaningful analyses. Nevertheless, the findings of this study did support the importance both of the specificity and generality hypotheses and the need to integrate both to facilitate a more comprehensive account of early onset problem behaviors on young adult substance abuse disorders.

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Figure 1.
Structural equation model of early onset problem behaviors predicting alcohol and other substance disorders in young adulthood.

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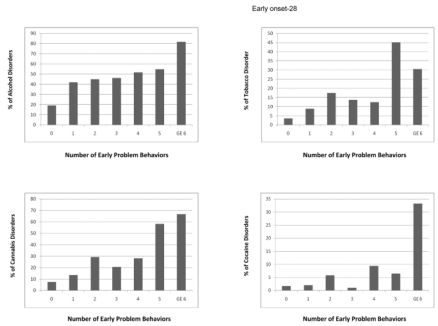

Figure 2.
Bar charts of number of early onset problem behaviors and occurrence of a lifetime alcohol, tobacco, cannabis, and cocaine disorders.

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Table 1

Prevalence of Early Onset Behaviors (*N*=671)

Early Onset Behavior	**Percentage (%)**
Alcohol	43.1
Tobacco	51.7
Marijuana	12.8
Cocaine	3.3
Police contact	5.7
Property damage	13.0
Stolen property	29.5
Fist-fight	20.0
Truant from school	14.0
Run away from home	6.0

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Table 2

Bivariate associations between early onset problem behaviors and young adult substance disorders (*N*=671)

Early onset behavior	Alcohol Disorder	Tobacco Dependence	Cannabis Disorder	Cocaine Disorder
Alcohol	2.05[c] (1.49–2.81)	3.17[c] (1.96–5.13)	3.92[c] (2.65–5.81)	4.42[c] (1.97–9.97)
Cigarettes	1.81[c] (1.33–2.48)	4.48[c] (2.57–7.81)	2.69[c] (1.81–3.98)	3.07[b] (1.36–6.90)
Marijuana	2.82[c] (1.77–4.50)	3.79[c] (2.23–6.45)	4.62[c] (2.88–7.42)	4.35[c] (2.06–9.22)
Cocaine/Crack	3.25[b] (1.31–8.07)	2.06 (0.74–5.74)	6.79[c] (2.79–16.52)	18.03[c] (7.02–46.27)
Police contact	2.98[b] (1.50–5.95)	0.79 (0.27–2.29)	2.47[a] (1.26–4.88)	6.48[c] (2.70–15.58)
Property damage	2.89[c] (1.81–4.60)	0.98 (0.50–1.93)	3.19[c] (1.99–5.12)	2.69[a] (1.21–6.00)
Stealing	2.05[c] (1.46–2.87)	1.68[a] (1.05–2.68)	2.61[c] (1.78–3.81)	2.36[a] (1.17–4.77)
Fight	2.44[c] (1.66–3.59)	1.79[a] (1.07–2.98)	1.63[a] (1.06–2.50)	3.63[c] (1.78–7.42)
Truancy	1.90[b] (1.22–2.94)	2.64[b] (1.54–4.53)	2.20[b] (1.37–3.52)	3.34[b] (1.56–7.15)
Runaway	2.06[a] (1.08–3.94)	1.77 (0.79–3.98)	2.02[a] (1.02–3.70)	1.62 (0.47–5.57)

[a] $p < .05$;

[b] $p < .01$;

[c] $p < .001$

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Table 3

Correlations among early onset problem behaviors and young adult substance disorders (*N*=671)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1.Alcohol	1.0													
2.Cigarettes	.59	1.0												
3.Marijuana	.78	.71	1.0											
4.Cocaine/crack	.67	.43	.86	1.0										
5.Police contact	.28	.14	.26	.53	1.0									
6.Property damage	.32	.20	.30	.36	.59	1.0								
7.Stealing	.28	.37	.31	.34	.57	.66	1.0							
8.Fight	.19	.16	.31	.43	.39	.54	.36	1.0						
9.Truancy	.48	.36	.50	.44	.42	.36	.37	.34	1.0					
10.Runaway	.34	.14	.20	.39	.35	.28	.27	.27	.25	1.0				
11.Alcohol Disorders	.27	.23	.34	.31	.31	.34	.27	.31	.21	.21	1.0			
12.Tobacco Disorder	.37	.45	.40	.18	−.06	−.01	.17	.18	.30	.15	.32	1.0		
13.Cannabis Disorders	.46	.34	.48	.49	.26	.37	.33	.17	.25	.20	.62	.49	1.0	
14.Cocaine Disorders	.40	.30	.39	.66	.46	.26	.24	.36	.32	.11	.63	.35	.74	1.0

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Table 4

Descriptive Statistics for Young Adult Outcomes

Outcome	*n (%)*	Mean	SD	Skewness	Kurtosis
Alcohol Disorder[a]	273 (40.7%)	.41	.49	.38	−1.86
Tobacco Dependence[a]	86 (12.8%)	.13	.33	2.23	2.98
Cannabis Disorder[a]	147 (21.9%)	.22	.41	1.36	−0.15
Cocaine Disorder[a]	33 (4.9%)	.05	.22	4.18	15.51
Alcohol Symptoms[b]	201 (30.0%)	1.90	1.82	.86	.05
Tobacco Symptoms[b]	420 (62.6%)	1.62	2.57	1.40	.65
Cannabis Symptoms[b]	475 (70.8%)	.73	1.49	2.45	5.75
Cocaine Symptoms[b]	621 (92.5%)	.25	1.03	4.77	23.13

[a]Number and percentage with disorder.

[b]Number and percentage with a score of zero

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Table 5

Individual early onset predictors of young adult substance symptoms

Early onset behaviors	Alcohol Disorder Symptoms	Tobacco Dependence Symptoms	Cannabis Disorder Symptoms	Cocaine Disorder Symptoms
Alcohol	.10[a]	.12[b]	.12[b]	.09[a]
Cigarettes	.05	.22[c]	.10[b]	.00
Marijuana	.06	.13[b]	.14[c]	.06
Cocaine/Crack	.03	−.02	.10[b]	.31[c]
Police contact	−.01	−.04	.02	.05
Property damage	.09[a]	−.07	.01	−.01
Stealing	.02	.04	.04	.01
Start Fight	.09[a]	.03	.05	.07
Truancy	.08	.05	.05	.00
Runaway	.05	.01	−.02	−.04
R-square value	.09[c]	.15[c]	.14[c]	.16[c]

[a] p < .05;

[b] p < .01;

[c] p < .001