

Development of Substance Dependence in Two Delinquency Subgroups and Nondelinquents From a Male Twin Sample

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ABSTRACT

Objective: The effect of delinquency subtype on the development of substance dependence symptoms was examined. It was proposed that early-onset delinquents possess characteristics that increase their likelihood of developing substance dependence problems earlier and more rapidly than late-onset delinquents and nondelinquents. **Method:** The development of alcohol, nicotine, and cannabis dependence symptoms (*DSM-III-R*) was examined over a 6-year period of adolescence (age 11–17) among 36 early-onset delinquent, 86 late-onset delinquent, and 25 nondelinquent boys from a large epidemiological twin sample. Multilevel/random coefficients models were used to compare groups on the rate of growth in number of symptoms over time. **Results:** As expected, early-onset delinquents showed an earlier onset and a faster rate of increase in the number of cannabis and nicotine dependence symptoms than late-onset delinquents and controls. Both delinquent groups had a more rapid increase in alcohol dependence symptoms than controls. **Conclusions:** The data showed that early-onset delinquency is associated with earlier onset of substance use disorder symptoms and more rapid acceleration of problems with drugs than late-onset delinquency. Treatments for boys with early-onset delinquency should account for their increased risk for drug use problems in adolescence and the potential effects of those problems on the course of antisocial behavior. *J. Am. Acad. Child Adolesc. Psychiatry*, 2002, 41(4):000–000. **Key Words:** early-onset delinquency, substance use disorders, adolescents.

Recent surveys of the literature suggest that as many as 51% of American teenagers have used alcohol, 34% have used nicotine, and 22% have tried illicit drugs by the time they graduate from high school (Weinberg et al., 1998). Epidemiological data suggest that 19% of adolescents under the age of 20 meet diagnostic criteria for alcohol abuse (Cohen et al., 1993). Teen substance use may be viewed as problematic in and of itself, but it is also associated with a host of other problem behaviors (Donovan et al., 1988) and negative consequences in and beyond adolescence (e.g., possible progression to sub-

stance abuse or dependence). This article addresses the issue of clinically significant substance use problems and how they develop differentially among boys with varying histories of antisocial behavior.

A positive association between substance use disorders (SUDs) and childhood externalizing disorders such as conduct disorder (CD), attention-deficit/hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD) has been a robust finding among both clinic-referred (Grilo et al., 1995; Hovens et al., 1994; Whitmore et al., 1997) and epidemiological samples (Cohen et al., 1993; Iacono et al., 1999). The relationship between SUD and conduct problems can be accounted for in a number of ways. Adolescents may engage in antisocial behaviors that are secondary to a SUD (Brown et al., 1996), or perhaps SUDs are just one of several negative outcomes found among adolescents with early-onset antisocial behavior (Brown et al., 1996; Moffitt et al., 1996). Thus SUDs may be a cause or a consequence of conduct problems.

The positive association between conduct problems and SUDs may arise from the influence of some third

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variable, such as peer group. Indeed, peer influence is a significant contributor to both early-onset delinquency (Patterson, 1986) and late-onset delinquency (Moffitt, 1993), especially the latter, and the association between peer factors and substance use in adolescence is robust (Kandel, 1996). It is possible that peers have an indirect effect on the development of SUDs among adolescents (especially among those with conduct problems).

Finally, it is possible that SUDs and conduct problems are both manifestations of the same underlying factor. In a recent review of the literature on SUDs in adolescents, Weinberg et al. (1998) pointed out that substance use does not typically progress to a SUD for most adolescents. However, those children at greatest risk for SUDs tend to exhibit more signs of disinhibited behavior such as poor executive cognitive functioning (e.g., poor judgment), higher levels of aggression, and higher rates of CD and ADHD (Weinberg et al., 1998). Furthermore, findings from a large epidemiological twin study suggest that substance dependence among adolescents is genetically influenced, and the transmission of SUDs from fathers to sons is mediated by a constellation of disinhibited characteristics (Iacono et al., 1999). Thus substance abuse and conduct problems may both be manifestations of an underlying (genetically mediated) tendency toward behavioral disinhibition.

Although delinquency and CD are themselves signs of disinhibition, it appears that delinquents can be further subdivided into groups that differ on disinhibited characteristics (Taylor et al., 2000). Developmental theorists have outlined two subtypes of delinquents delineated largely by differences in persistence (and age at onset) of antisocial behavior and by differences in etiology (DiLalla and Gottesman, 1989; Moffitt, 1993). Taylor et al. (2000) found that early-onset delinquent boys (early starters) showed more signs of disinhibition than late-onset delinquent boys (late starters), including significantly greater rates of ADHD and ODD and greater impulsivity. In addition, there was evidence of greater genetic influence on the early starter phenotype than on the late starter phenotype, supporting the notion of different etiologies for the two subtypes.

In addition to differences in behavioral disinhibition, early starters have more severe behavioral problems than late starters (Moffitt et al., 1996). Early starters also are more prone to severe antisocial behavior and adult offending (Farrington et al., 1990; Moffitt, 1993). This association between onset and severity is not unique in clinical

science. For example, a similar relationship has been noted in the literature on alcoholism in adults (Babor et al., 1992; Cloninger, 1987; McGue et al., 1997; Watson et al., 1997). Two subtypes of alcoholism have been identified in adults: type I and type II (Cloninger, 1987), type A and type B (Babor et al., 1992), or late-onset and early-onset. The early-onset alcoholic subtype (type II, type B) is characterized by antisocial behavior, more severe alcohol problems, greater incidence of drug use disorder, greater family history density for alcohol problems, and greater genetic influence than the late-onset type (Babor et al., 1992; Cloninger, 1987).

Early-onset delinquents possess characteristics that may increase their likelihood of developing substance dependence problems earlier and to a greater extent than their late-onset counterparts. For example, early starters score higher on indices of impulsivity (White et al., 1994) and lower on the personality dimension of constraint (Tellegen, 2000) than late starters (Taylor et al., 2000). Thus early starters tend to be less behaviorally inhibited, less concerned about the morality of their behavior, and more attracted to exciting and novel activities. Early starters also score higher on the negative emotionality personality dimension (Tellegen, 2000) than late starters (Taylor et al., 2000). Thus early starters tend to be more emotionally labile and less optimistic about how the world treats them. This same pattern of personality characteristics is associated with and appears to predate early-onset substance abuse (Cloninger et al., 1988).

Early starters also have an earlier affiliation with antisocial peers than late starters (Taylor et al., 2000), and it is reasonable to suspect that a child's peer group may influence those factors related to the development of substance dependence (e.g., frequency and severity of use). Finally, early starters are more severe in their antisocial behavior, and (by definition) they exhibit disinhibited behavior at an earlier age. As such, if substance dependence were, in part, a consequence of antisocial behavior, then early starters would again be expected to exhibit earlier signs of substance dependence and greater escalation of problems compared with late starters.

In an investigation of outcomes (including substance dependence) among subgroups of delinquents, Moffitt et al. (1996) found that early-onset severely antisocial boys and late-onset severely antisocial boys had similar rates of alcohol, nicotine, and cannabis dependence diagnoses at age 18, indicating that SUDs may be a "snare" that contributes to the persistence of antisocial behavior.

Although that cross-sectional analysis suggested that late adolescent outcomes were similar for both early and late starters, it did not shed light on differences in severity or in the *rate* of development of substance dependence problems between the two groups. To obtain a more complete understanding of the relationship between delinquency subtypes and the development of substance dependence, one must examine the development of substance dependence longitudinally during adolescence.

Part of the challenge of examining the development of clinically significant substance use problems is in their measurement. Indeed, reliance on diagnoses would likely hide differences present at the earliest stages of substance use problems given that most children and adolescents are not likely to meet full criteria for a SUD. However, children in preadolescence or mid-adolescence may evidence *symptoms* of a SUD, thus marking the initial stages of the development of a problem with drugs or alcohol.

Early-onset delinquents appear to represent a subgroup of delinquents at especially high risk for the early development of substance dependence problems. To test this hypothesis, we compared early starters (boys with onset of antisocial behavior at or before age 12), late starters (boys with onset of antisocial behavior after age 12), and nondelinquent controls (boys with no indication of antisocial behavior through age 17) for the emergence of alcohol, nicotine, and cannabis dependence symptoms over a 6-year period in adolescence. Early starters were expected to have an earlier onset and a more rapid increase in the number of substance dependence symptoms over the course of adolescence than late starters and controls. Given previous findings that late-onset delinquents are also at risk for substance dependence problems, we expected that late starters would show a faster increase in their symptom counts over the course of adolescence than nondelinquents.

METHOD

Participants

This study examined a subsample of 147 boys aged 10 to 12 years (mean = 11.32, SD = 0.51) participating in the Minnesota Twin Family Study (MTFS). Briefly, the MTFS is an ongoing, community-based study of substance abuse and related problems among same-sex, reared-together twins and their parents. Families are recruited for follow-up visits at 3-year intervals. Parents of 11-year-old boys participating in the MTFS had, on average, a high school degree (or its equivalent) and 13.77 (SD = 1.89) years of education. Consistent with the demographics in Minnesota at the time the twins were born, 98% of the sample were white. Participants who were 18 years old at the time of their second follow-up assessment gave written informed consent to participate in the study. Those younger than 18 gave written informed

assent, and one of their parents gave written informed consent for the son's participation at each assessment. Although this study was not concerned with hypotheses related to genetic influence, the MTFS data set offered distinct advantages, including the availability of data on clinically significant substance use problems (assessed systematically with structured interviews) from a clinically relevant period of development (adolescence) over multiple time points, which made it well suited for this investigation. In addition, rates of delinquency and related disorders (ADHD, ODD) in this twin sample (see Taylor et al., 2000) were comparable with rates reported for nontwin samples (e.g., Cohen et al., 1993; Moffitt, 1993), indicating that the results from this sample can reasonably be generalized to nontwin samples.

The sample for this study consisted of 36 early starters (boys who evidenced at least three of the following before age 12: aggression at school, CD diagnosis, contact with the police, or a score greater than 1 SD above the mean on the Delinquent Behavior Inventory, a 36-item delinquency checklist), 86 late starters (boys with at least one of the indicators noted above occurring after age 12), and 25 nondelinquent controls (boys with no signs of antisocial behavior through age 17). (For more detail on the measures used to select groups, see Taylor et al., 2000.) Age 12 was selected as the cut point for onset on the basis of research on hazard rates for delinquency (Farrington et al., 1990). Information about substance use or SUD was *not* used in defining groups.

All but one boy from the present sample (an early starter) completed the first follow-up assessment, and 93% completed the second follow-up. Of the 10 boys with missing data at age 17, 70% (6 early starters, 1 late starter) refused the assessment and the rest (3 late starters) were as yet unable to be scheduled for a visit.

Diagnostic Interview and Procedures

Symptoms of alcohol, nicotine, and cannabis dependence were assessed at three time points (age 11, 14, and 17) using *DSM-III-R* (American Psychiatric Association, 1987) criteria. When the MTFS began, *DSM-III-R* was the standard diagnostic manual in psychiatry. *DSM-III-R* criteria were used for this analysis because they were consistently applied to all three assessments.

Symptoms were assessed using structured clinical interviews administered independently to each boy and his mother by trained interviewers (each of whom had a bachelor's or master's degree in psychology). Lifetime occurrence of substance dependence symptoms was assessed at ages 11 and 14 via a modified version of the Diagnostic Interview for Children and Adolescents-Revised, child version (DICA-R-C) (Herjanic and Reich, 1982; Reich and Welner, 1988). Age-at-onset information was checked to ensure that symptoms reported at age 14 had occurred since the intake assessment. At age 17, boys were administered the expanded Substance Abuse Module (Robins et al., 1987) from the Composite International Diagnostic Interview (Robins et al., 1988) to assess substance dependence symptoms occurring since the previous visit (at age 14). At each assessment, mothers reported on their son's symptoms (for similar time periods noted above) via a modified version of the DICA-R, parent version (DICA-R-P) (Reich and Welner, 1988). Symptoms were assigned by consensus of two or more clinical psychology graduate students (in their second year of study or beyond) using all available information. Separate alcohol, nicotine, and cannabis dependence symptom counts were tallied from each assessment by summing (via computer algorithm) the number of *DSM-III-R* symptoms met in full by either informant's report.

Analyses

The goal of this study was to examine individual change in substance dependence symptoms over time and to examine how differences in change related to group membership. Thus the data were

analyzed by means of a multilevel/random coefficients or hierarchical linear model (HLM) (Bryk and Raudenbush, 1992). The 147 subjects in the present study comprised 34 twin pairs and 79 individual twins distributed among the three groups. Individual assessments thus were inherently clustered within two higher-order levels: individual subjects' data consisted in repeated assessments, and each such vector of repeated assessments in turn was nested within twin pairs. To the extent that individuals of the same age growing up together in the same family are likely to be similar with respect to substance dependence symptoms, their symptom counts will be correlated, which violates the basic assumption of many statistical procedures that each observation is independent of all others, and therefore results in biased tests. In contrast, HLM can explicitly incorporate such groupings of subjects. We therefore adopted a three-level HLM, with assessments constituting the first level, individuals the second, and twin pairs the third (Bryk and Raudenbush, 1992).

The symptom count for each substance (alcohol, nicotine, cannabis) was log-transformed and modeled separately. Given the variability in symptom counts across subjects, a random coefficients regression approach was used to model the log-transformed symptom counts as a function of time. In essence, the random coefficients approach simultaneously estimates regression curves for each individual and for the sample as a whole. In addition, in the present study, regression curves were also estimated for each family or twin pair. Therefore, the model produces estimates of intercept and slope for the whole sample (the sole focus of traditional regression approaches) and for each twin pair and each individual within a twin pair. The individual-specific regression coefficients are treated as representing random samples normally distributed around the family-specific coefficients, which in turn are random samples normally distributed around the sample average—hence the term random coefficients or random effects. The sample regression coefficients are invariant across individuals and thus represent fixed effects.

In addition to being able to accommodate clustering of observations, the approach offers another very practical advantage: it permits inclusion of subjects who do not have complete data available; the procedure "borrows" from those who do by using the whole-sample variance-covariance matrix of regression coefficients in estimating individual coefficients. This allowed us to include subjects who had not completed either or both follow-up assessments, rather than having to exclude them as required by traditional approaches such as repeated-measures analyses of variance. This was a particular advantage in the present study given that several early starters were missing data at age 17, which could have presented a threat to the reliability of traditional repeated-measures analyses.

To model the tendency for symptom counts to increase more rapidly later in adolescence, a quadratic term was included in the model, and the log-transformed symptom counts were estimated as a linear and nonlinear (quadratic) function of time. For each individual, the actual time from the intake visit, which averaged 3 and 6 years for the two follow-up assessments, was used. The time variable was "centered" such that it equaled 0 for the intake visit and represented years since that assessment for each follow-up visit. Thus the fixed effects in each model were the log-transformed number of symptoms at the age 11 assessment (the intercept), the linear and quadratic functions of time since intake, the group variable (early starter, late starter, or control), and group \times linear change and group \times quadratic change interactions. We included individual-specific slope parameters as a random effect in each model. Random intercepts were not included because only a few boys had symptoms at the intake assessment (age 11). The model thus estimated individual differences in the rate of change in log-transformed symptoms but not in the initial level. We included linear random effects for all three models and a quadratic random effect for alcohol and nicotine dependence (this parameter yielded variance estimates close to zero for cannabis dependence and was dropped from the model). The covariance matrix of random effects was freely estimated and all model parameters were derived by means of restricted maximum likelihood estimation procedures.

By virtue of the manner in which the model was expressed, the statistical software used (SAS PROC MIXED) produced significance tests of the regression parameters for each group, as well as F tests for group differences in these parameters. We examined overall growth curves for symptoms of substance dependence by considering intercept and both linear and quadratic slope coefficients simultaneously, and we contrasted each pair of groups with respect to these growth curves. Similar contrasts of the overall rate of increase in symptom count were obtained by considering linear and quadratic coefficients simultaneously.

RESULTS

Table 1 presents the (raw) means and standard deviations for substance dependence symptom counts at age 14 and 17 (very few boys had symptoms at age 11). Late starters averaged approximately one symptom for each substance at age 17 (consistent with a substance abuse diagnosis), whereas early starters averaged two to three

TABLE 1
Symptom Count Means and Standard Deviations at Each Assessment for Each Group

	Control		Late Starter		Early Starter	
	Mean	SD	Mean	SD	Mean	SD
First follow-up (age 14)	<i>n</i> = 25		<i>n</i> = 86		<i>n</i> = 35	
Alcohol dependence	0.00	0.00	0.19	0.56	0.60	1.72
Nicotine dependence	0.00	0.00	0.48	1.25	1.20	2.14
Cannabis dependence	0.00	0.00	0.13	0.72	0.69	1.98
Second follow-up (age 17)	<i>n</i> = 25		<i>n</i> = 82		<i>n</i> = 30	
Alcohol dependence	0.12	0.44	1.37	1.78	2.27	2.48
Nicotine dependence	0.04	0.20	1.59	2.01	3.13	2.36
Cannabis dependence	0.04	0.20	1.15	2.04	3.13	3.15

Note: Early starters had the only symptoms at age 11: alcohol (mean = 0.03; SD = 0.17); nicotine (mean = 0.33; SD = 1.10).

symptoms for each substance at age 17 (consistent with a more severe clinical presentation such as a substance dependence).

Figure 1 presents growth curves for (a) alcohol, (b) cannabis, and (c) nicotine dependence symptoms in the three groups over 6 years. The mean log-transformed symptom count for each group is presented along with the estimated growth curve, reflecting the increase in symptoms from age 11 to 17. Table 2 presents estimates of the regression coefficients for the fixed parameters in each of the substance dependence models for each group. As indicated in Table 2, the quadratic change parameter was significant for alcohol, cannabis, and nicotine dependence for both delinquency groups, indicating a tendency for problems with drugs and alcohol to accelerate in later adolescence for both early- and late-onset delinquents. Remarkably, the control boys did not show even a linear increase in SUD symptoms over the course of adolescence, as evidenced by all nonsignificant parameters for that group in Table 2 (and the flat growth curves in Figure 1a–c).

Figure 1a illustrates the group differences in rate of increase of problems with alcohol ($F_{3,144} = 13.32, p < .001$). As expected, group contrasts revealed that the quadratic change was significantly greater for late starters than for controls ($F_{1,44} = 4.60, p = .034$). Group contrasts that simultaneously considered the intercept and both slope terms, and thus constituted contrasts of the overall growth of alcohol problems, were significant for the controls versus early starters and late starters ($F_{3,144} = 5.25, p = .002$ and $F_{3,144} = 3.69, p = .013$, respectively). However, as illustrated by similar growth curves in Figure 1a, early starters and late starters showed only a trend (in the expected direction) toward a significant difference in overall growth rate in alcohol problems ($F_{3,144} = 1.84, p = .142$). As expected, the overall increase in alcohol symptoms was significantly greater for both early starters and late starters relative to controls ($F_{2,144} = 6.50, p = .002$ and $F_{2,144} = 5.51, p = .005$, respectively).

Growth curves for cannabis dependence for each group are presented in Figure 1b. Once again, symptom count showed an accelerating trend over time, reflected by a significant quadratic effect among groups ($F_{3,278} = 26.58, p < .001$). The quadratic term was significantly greater for both early and late starters than for controls ($F_{1,278} = 15.91, p = .001$ and $F_{1,278} = 6.00, p = .015$, respectively). As expected, contrasts of overall growth were significant for all pairwise comparisons ($F_{3,278} = 5.56, p = .001$ for early starters versus late starters, $F_{3,278} = 11.60, p < .001$ for early starters

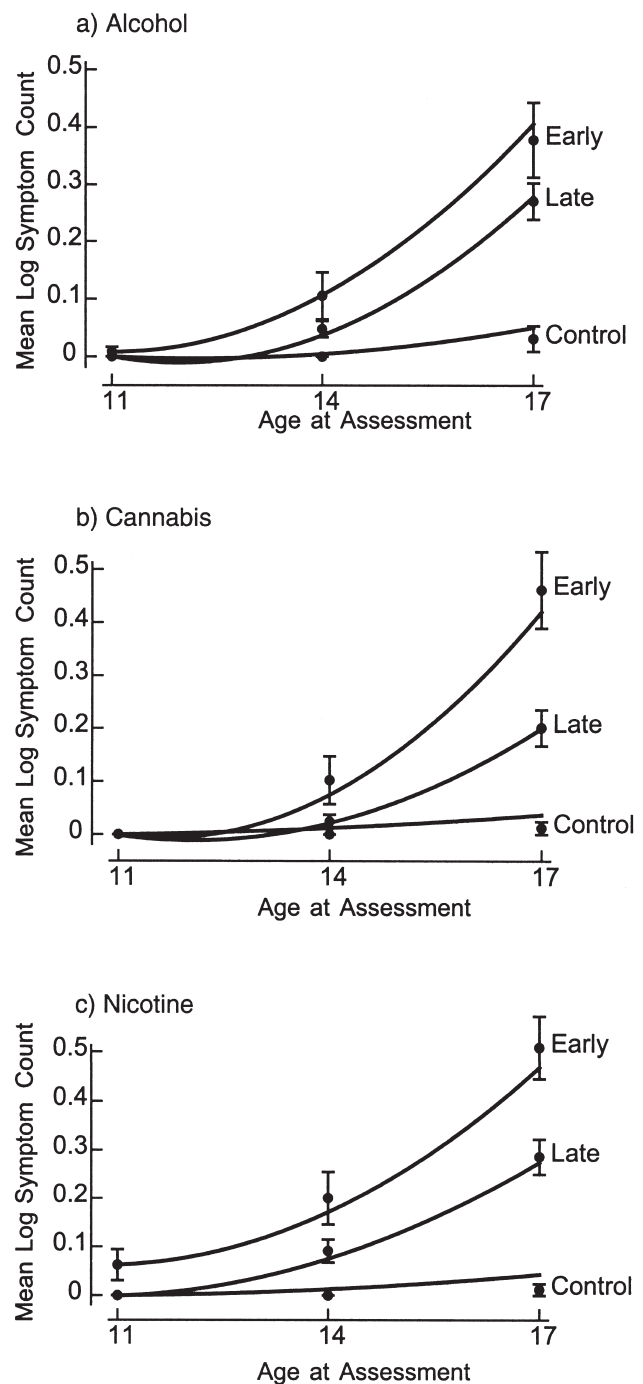


Fig. 1 Growth curve in mean log-transformed alcohol, cannabis, and nicotine dependence symptom count for each group. Note: The group mean of log-transformed symptom counts is given at each assessment and the associated (\pm) standard error is represented with a vertical line and cross bars; Early = early starter; Late = late starter. Curves were obtained from the estimated intercept and linear and quadratic slope coefficients for each group using 0, 3, and 6 years, respectively, as the value of the time variable at each visit. Curves do not pass through all points primarily because a second-degree polynomial is necessarily limited in its ability to fit three data points.

TABLE 2
Estimates of Regression Fixed Effects for Alcohol, Cannabis, and Nicotine Dependence Models

Effect	Alcohol		Cannabis		Nicotine	
	<i>B</i>	(SE)	<i>B</i>	(SE)	<i>B</i>	(SE)
Early starters						
Initial level	.008*	(.004)	.001	(.016)	.063***	(.016)
Linear change	-.009	(.017)	-.021	(.017)	.005	(.025)
Quadratic change	.011**	(.004)	.015***	(.002)	.011*	(.004)
Late starters						
Initial level	-.000	(.003)	-.002	(.011)	-.001	(.010)
Linear change	-.022*	(.011)	-.018	(.011)	.005	(.016)
Quadratic change	.011***	(.002)	.009***	(.002)	.007**	(.003)
Controls						
Initial level	-.000	(.005)	.000	(.021)	-.000	(.019)
Linear change	-.005	(.019)	.002	(.020)	.001	(.029)
Quadratic change	.002	(.004)	.001	(.003)	.001	(.005)

Note: The “initial level” estimate corresponds to the *y*-intercept, which reflects the modeling estimate of the log-transformed symptom count at time 0 (age 11). The regression equation that best fit the data estimated a few of the initial level estimates at slightly above or below 0; these estimates can be read simply as 0 (indicating no symptoms at age 11). “Linear” and “quadratic change” represent the two slope coefficients. *B* gives the restricted maximum likelihood estimate of the regression coefficient associated with each effect and (SE) gives its standard error.

* $p < .05$; ** $p < .01$; *** $p < .001$. Significance level is for the test of the difference of *B* from zero.

versus controls, and $F_{3,278} = 3.66$, $p = .013$ for late starters versus controls), indicating that all groups differed significantly in the rate of increase of problems with cannabis from age 11 to 17. Consistent with our hypothesis, the early starters showed a significantly greater increase overall in cannabis dependence symptoms relative to controls ($F_{2,278} = 17.37$, $p < .001$) and late starters ($F_{2,278} = 8.31$, $p < .001$). Late starters and controls also differed significantly ($F_{2,278} = 5.41$, $p = .005$), as expected. (Note that estimates of the degrees of freedom for the test statistics are a function of the number of random effects in the model; hence the difference between denominator degrees of freedom for tests associated with cannabis dependence versus alcohol or nicotine dependence.)

Finally, as illustrated in Figure 1c, nicotine symptom count also showed an accelerating trend over time as the quadratic term differed significantly among groups ($F_{3,144} = 4.50$, $p = .005$). However, none of the pairwise contrasts for nonlinear change in nicotine symptoms was significant. As foreshadowed by the estimates reported in Table 2, the intercept (initial level) for nicotine symptoms differed significantly among groups ($F_{3,144} = 5.23$, $p = .002$), indicating that group membership was significantly related to level of nicotine symptoms at the intake assessment. Between-group contrasts revealed that the intercept was significantly greater in the early starters than in the controls and late starters ($F_{1,144} = 6.44$, $p = .012$ and $F_{1,144} =$

11.30 , $p = .001$, respectively). Contrasts of overall growth were significant for early starters versus controls and late starters ($F_{3,144} = 10.27$, $p < .001$ and $F_{3,144} = 6.77$, $p < .001$, respectively). Late starters also differed significantly from controls ($F_{3,144} = 3.50$, $p < .017$). As expected, both early and late starters had a significantly greater overall increase in nicotine symptoms than controls ($F_{2,144} = 8.42$, $p < .001$ and $F_{2,144} = 4.95$, $p = .008$). The difference between early and late starters was not significant, but it was in the expected direction ($F_{2,144} = 1.82$, $p = .165$).

DISCUSSION

This was one of the first investigations of the development of substance dependence in two well-defined delinquency subgroups. The use of longitudinal data allowed for an examination of changes in alcohol, nicotine, and cannabis dependence symptom counts from preadolescence to mid-adolescence to late adolescence among boys classified as early-onset delinquents, late-onset delinquents, or nondelinquent controls. Given the characteristics differentially associated with early-onset delinquency (e.g., higher prevalence of behavioral disorders, greater affiliation with antisocial peers, etc.), early starters were expected to be at greater risk for the development of substance dependence problems than late starters and controls. These expectations were largely supported by the data.

The results were consistent with the notion that boys who are disinhibited, associate with antisocial peers, and possess certain psychological characteristics (e.g., low constraint) are more prone to the development of early-onset substance dependence than boys who lack or have lower levels of these characteristics. Moreover, the results were consistent with the notion that early-onset substance dependence may be mediated by disinhibited characteristics such as those possessed by early starters (Iacono et al., 1999). This study cannot address questions about the biological framework for the various disinhibited characteristics observed among early starters; however, there is some evidence that the link is, in part, genetic (Iacono et al., 1999; Slutske et al., 1998).

The developmental period reported here covers only a portion of the full-risk period for the development of a SUD. However, it is possible to construct reasonable predictions that lay the groundwork for future investigations. As noted earlier, the literature on adult alcoholism suggests two types: early-onset (type II, type B) and late-onset (type I, type A). The early-onset type shows greater genetic influence, is more associated with antisocial behavior and illicit drug use, and is more persistent and severe than the late-onset type. Similarly, the taxonomy proposed for delinquency can be framed as early-onset versus late-onset. The early-onset type shows greater genetic influence and is more persistent and severe than the late-onset type. The present data, taken together with other research on delinquency, could suggest that early-onset delinquents are simply more severe than late-onset delinquents on many outcomes including substance use problems. However, these data, considered in light of the adult alcoholism literature, suggest that some early starters may go on to exhibit the type II/B alcoholism phenotype as adults. Indeed, studies such as this one may provide clues that further our understanding of certain types of adult alcoholism that may in turn lead to more effective treatments or prevention strategies.

Limitations

The strength of this study lies in the longitudinal design, the assessment of clinical symptoms of SUD using structured clinical interviews with two informants, and the use of an analytic method that compared group differences in the rate of change in SUD symptoms, thus providing a clearer picture of developmental differences between groups. The present analysis, however, also had limitations. First, the sample was racially homogeneous

and all male, and thus the findings from this study must be applied cautiously to girls and nonwhite populations. Second, the data did not cover the entire risk period for the development of SUDs, and thus it should not be assumed that the observed differences between delinquency subgroups at age 17 will extend into adulthood. Finally, this analysis considered only one variable (delinquency) related to the development of SUDs, but many other factors (e.g., peers, personality, comorbid diagnoses such as ADHD that may also be linked to disinhibition, etc.) likely influenced the development of substance dependence in this sample.

Clinical Implications

The literature provides evidence of a robust association between antisocial behavior and substance use in adolescence. The data from this study provided some new information to consider in the treatment of antisocial behavior in boys: some delinquent boys are at greater risk for the early development of clinically significant substance use problems than others. Our data showed that early-onset delinquency is associated with earlier onset of SUD symptoms and greater increase and faster acceleration of problems with drugs than late-onset delinquency. Moffitt (1993) suggested that substance use problems may contribute to the continuation of antisocial behavior from adolescence into adulthood among late-onset delinquents. It is just as easy to see the potential effect of substance use problems on the persistence of antisocial behavior among early-onset delinquents. As such, treatments for boys with early-onset delinquency should account for their increased risk for substance use problems in adolescence and the potential for those problems to further exacerbate antisocial behavior problems.

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