

# Detection of Youth at High Risk for Substance Use Disorders: A Longitudinal Study

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This study extends prior research (D. Clark, J. Cornelius, L. Kirisci, & R. Tarter, 2005) by determining whether variation in the developmental trajectories of liability to substance use disorder (SUD) is contributed by neurobehavioral disinhibition, parental substance use involvement, and demographic variables. The sample, participants in a long-term prospective investigation, consisted of 351 boys, evaluated at ages 10–12, 12–14, 16, 19, and 22, whose parents either had SUD or no adult psychiatric disorder. Neurobehavioral disinhibition in childhood, in conjunction with parental lifetime substance use/SUD, place the child at very high risk for SUD by age 22 if psychosocial maladjustment progresses in severity in early adolescence. These results indicate that monitoring social adjustment during the transition from childhood to mid-adolescence is important for identifying youth at very high risk for succumbing to SUD by young adulthood.

*Keywords:* children, neurobehavioral disinhibition, substance use disorder, drug abuse, etiology

It is increasingly recognized that manifestation of substance use disorder (SUD; American Psychiatric Association, 1994) is the result of the cumulative interaction between the individual and environment (Glantz, 1992; Tarter & Vanyukov, 1994; Wills & Stoolmiller, 2002). Numerous biological and behavioral factors associated with adolescent-specific reproductive and neurologic maturation processes have been implicated to underlie substance use behavior (Spear, 2000) and SUD risk (Tarter et al., 1999). With respect to the early age onset variant of SUD, commonly referred to as *Type II* (Cloninger, Bohman, & Sigvardsson, 1981), the most salient predisposing factor is deficient inhibitory regulation that is overtly manifest as conduct problems and antisociality (Cadoret, Troughton, O’Gorman, & Heywood, 1986; Zucker, Ellis, Fitzgerald, Bingham, & Raymond, 1996). Difficult temperament in early childhood predisposes the child to both externalizing and internalizing psychiatric disorders in late childhood (Maziade, Caron, Cote, Boutin, & Thiverge, 1990) and amplifies the risk for substance abuse (Blackson, 1994; Lerner & Vicary, 1984) and SUD (Tarter, Moss, & Vanyukov, 1995).

Two comprehensive reviews of the empirical literature have concluded that the diverse pattern of psychological characteristics exhibited by youth at high risk for SUD reflect a core inhibitory disorder (Spear, 2000; Tarter et al., 1999). Physiological research involving measurement of the P300 component of the event-related potential (Begleiter & Porjesz, 1999), electrodermal activation (W. Iacono, Carlson, Taylor, Elkins, & McGue, 1999), and

voluntary control of saccadic eye movement (Blecker et al., 2002; M. Iacono, Carlson, & Malone, 2000; W. Iacono et al., 1999) also indicates that impaired inhibitory regulation is a cardinal component of SUD risk. Of significance is that genetic factors contribute strongly to the variation of behavior disinhibition (Young, Stallings, Corley, Krauter, & Hewitt, 2000).

Ongoing prospective investigations conducted at the Center for Education and Drug Abuse Research (CEDAR; Tarter & Vanyukov, 2001) have confirmed and extended findings documenting the importance of inhibitory processes to the risk for SUD. Children of parents with SUD obtain higher scores on a construct measuring neurobehavioral disinhibition compared with children of control parents (Tarter et al., 2003). The indicators of this trait are executive cognitive capacity, affect modulation, and behavior control. Of note is that the neurobehavioral disinhibition score at age 10–12 is a significant predictor of SUD by age 19 (Tarter et al., 2003). Indeed, at age 16, neurobehavioral disinhibition is a stronger predictor of SUD than substance use frequency. Subsequent research has shown that cognitive distortions mediate the association between childhood neurobehavioral disinhibition and marijuana use during adolescence, which in turn predisposes the youth to SUD by young adulthood (Kirisci, Tarter, Vanyukov, Reynolds, & Habeych, 2004). In addition, childhood neurobehavioral disinhibition promotes social maladjustment in adolescence, leading to SUD (Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004).

A recently completed investigation at CEDAR has shown that family history of SUD, use of alcohol or tobacco at a young age, and neurobehavioral disinhibition comprise the dimensions of five empirically validated subtypes (Clark, Cornelius, Kirisci, & Tarter, 2005). The subtypes are discriminable with respect to acceleration toward daily tobacco use, emergence of alcohol-related problems, age of first use of cannabis, age of onset of diagnosis of cannabis use disorder according to the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV;* American Psychiatric Association, 1994), and age of first cocaine use.

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The research reported in this article was supported by Grants P50-DA005605, R01-DA011922, R01-DA019157, and K02-DA017822 from the National Institute on Drug Abuse.

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The present investigation, extending this line of research, determined the extent to which youth who develop SUD can be classified into distinct developmental trajectories based on family history, neurobehavioral disinhibition, and substance consumption during early adolescence.

## Method

### *Project Background*

This report describes results obtained from a longitudinal investigation directed at elucidating the biobehavioral and environmental components of SUD liability. The sample, consisting of almost 800 families accrued to date, has been recruited over 15 years. A long period of participant recruitment was necessitated by the time-consuming, comprehensive, and labor-intensive genetic, physiological, psychological, family, and social environment assessments. Interim results are reported herein inasmuch as the terminal outcome assessment will not be performed until 2020. Accordingly, this study is based on an ongoing project involving access to an expanding sample, including individuals who have been participants in previous research (Tarter et al., 2003; Tarter & Vanyukov, 2001).

### *Participants*

From a total sample of 485 boys enrolled in CEDAR between 1990 and 2004, a subset of 351 participated in this study. To qualify, the boys were required to have completed the baseline evaluation (age 10–12) and at least one of the follow-up assessments that were scheduled at ages 12–14, 16, 19, and 22.

The boys were ascertained on the basis of presence or absence of lifetime diagnosis of SUD (abuse or dependence) concomitant to consumption of an illicit compound in the biological father. The sample consisted of 167 (48%) boys whose fathers had SUD and 184 (52%) boys whose fathers had no adult Axis I and Axis II psychiatric disorder. The sample was 79% European American and 21% African American. Mean family socioeconomic status (SES; Hollingshead, 1975) was 40.6 ( $SD = 13.3$ ), indicating that the sample was primarily middle class. Females were not included in this study because their recruitment began several years after the boys. Hence, an insufficient number of females was available to conduct longitudinal analyses.

Fathers (proband) with lifetime SUD diagnosis (SUD+) were recruited from substance abuse treatment programs, social service agencies, newspaper and radio advertisements, public service announcements, and random digit telephone calls. Use of multiple recruitment sources was necessitated by the difficulty associated with identifying a hard-to-reach population that also has a low prevalence disorder, namely, SUD consequent to illicit drug use. In addition, the ascertainment criteria are stringent. The SUD+ fathers must have a biological son 10–12 years of age, have no history of psychosis, a Wechsler Adult Intelligence Scale (Wechsler, 1997) full scale IQ in the normal range, and good health status.

The SUD– men were recruited using the same method except that none were acquired from treatment facilities. Because a random sampling procedure was not used, we have previously compared (Tarter & Vanyukov, 2001) the men recruited in this study with adult men in the Epidemiological Catchment Area Study (Anthony & Helzer, 1991). Analyses revealed that this sample and comparable men in the Epidemiological Catchment Area Study are similar with respect to SES, severity of SUD, and pattern of comorbid psychiatric disorder. These findings suggest that the proband fathers in this study are not atypical.

### *Procedure*

Written assent and informed consent were respectively obtained from the children and their biological parents before the research protocols were

administered. The informed-consent procedure was carried out separately for each individual. Probes were conducted during the consenting procedure to ensure that the proband and his spouse had not coerced the child into participating. In addition, each family member was informed that privacy of the findings from this research was protected by a Certificate of Confidentiality issued to CEDAR by the National Institute on Drug Abuse.

### *Instrumentation*

*Diagnostic formulation.* The biological parents were administered an expanded version of the Structured Clinical Interview for *DSM-III-R* (SCID) to obtain Axis I and II psychiatric diagnoses (Spitzer, William, Gibbons, & First, 1990). The SCID was expanded to more comprehensively characterize antisocial behavior in childhood and adulthood. Diagnoses were formulated in a clinical conference chaired by a psychiatrist certified in addiction psychiatry and attended by another psychiatrist or a psychologist along with the clinical associates who conducted the interviews. The best-estimate procedure was used to formulate diagnoses (Leckman, Sholomaskas, Thompson, Belanger, & Weissman, 1982). The results of the interview, accompanied by a review of information obtained in the protocols evaluating medical, legal, social, and psychiatric history, were used by the committee members to consensually formulate lifetime and current SUD diagnoses. We used *DSM-III-R* criteria (American Psychiatric Association, 1987) for ascertainment of probands and diagnoses of the mothers because this project was initiated prior to the advent of *DSM-IV*. The rate of lifetime SUDs and the most frequent co-occurring SUD diagnoses, along with the most frequent co-occurring psychiatric disorders, are summarized in Table 1.

Baseline assessment of the boys was conducted when they were 10–12 years of age. A total of 15 follow-up evaluations are projected between baseline and the terminal evaluation, to be conducted when the boys attain 30 years of age. As of December 31, 2003, a total of 351 participants had been tracked to age 12–14, 293 participants had been tracked to age 16, 183 participants had been tracked to age 19, and 91 participants had been tracked to age 22. The decreasing sample size does not indicate attrition; instead, it describes the varying stages of follow-up reflecting the protracted period of family recruitment as noted above. At age 22, only 25 participants who were scheduled for reevaluation refused to participate or could not be located.

Psychiatric evaluation was conducted using the Kiddie—Schedule for Affective Disorders and Schizophrenia (K-SADS; Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982) when the boys were 10–12, 12–14, and 16 years of age. Both the child and the primary caretaker (typically the mother) were administered the K-SADS, yielding both self- and informant-based diagnostic information. Both sources of data were used in the clinical conference to consensually determine current and lifetime diagnoses. From age 19 onward, the SCID was used. We used *DSM-IV* criteria (American Psychiatric Association, 1994) to categorize the presence or absence of SUD outcome because this taxonomic system was published before this outcome was manifest by any of the participants. Hence, we were able to incorporate the *DSM-IV* criteria after this project was initiated. The best-estimate procedure was used to formulate psychiatric diagnoses. The SUD diagnoses of the boys are summarized in Table 2.

*Drug Use Chart.* This self-report questionnaire was administered to the parents to assess their lifetime exposure to 42 psychoactive substances, grouped into 10 categories consistent with the National Institute of Mental Health's Epidemiological Catchment Area Study (Anthony & Helzer, 1991). The drug categories were alcohol, cannabis, cocaine, opiates, amphetamines, tranquilizers, sedatives, tobacco, PCP, and inhalants.

A continuous substance use index (SUI) was derived using these drug categories as items by applying two-parameter logistic item response theory (IRT) modeling (Kirisici, Vanyukov, Dunn, & Tarter, 2002). It was found that the 10 drug categories are indicators of a unidimensional latent trait. The IRT-based reliability coefficients were, respectively, .88 and .82 for the fathers and mothers. The mean SUI scores of the fathers and

Table 1  
*Distribution of Lifetime Substance Use Disorder (SUD) and Psychiatric Disorder in Parents*

Disorder	Fathers (n = 351)		Mothers (n = 351)	
	Abuse	Dependence	Abuse	Dependence
<b>SUD diagnosis</b>				
Alcohol	24.2	28.5	10.0	10.8
Amphetamines	2.6	2.9	1.1	3.1
Cannabis	18.5	15.7	5.1	5.1
Hallucinogens	0.9	2.3	0.9	0
Cocaine	6.8	17.4	1.4	4.8
Inhalants	0.9	0	0	0.6
Opiates	2.8	9.1	1.1	4.6
PCP	0.9	2.3	0	0
Sedatives	1.4	3.4	1.1	2.0
<b>Most frequent patterns of SUD comorbidity<sup>a</sup></b>				
Alcohol + cannabis	25.6		6.0	
Alcohol + cocaine	17.4		3.7	
Alcohol + opiates	7.7		3.7	
Cannabis + cocaine	10.2		2.0	
Cannabis + opiates	4.0		.3	
Cocaine + opiates	7.1		6.0	
Alcohol + cannabis + cocaine	8.8		1.4	
Alcohol + cannabis + opiates	3.7		.3	
Alcohol + cocaine + opiates	5.7		1.1	
Cannabis + cocaine + opiates	2.8		0.3	
<b>Psychiatric disorder</b>				
Anxiety	6.0		19.9	
Depression	14.0		31.1	
Antisocial personality	9.1		2.0	

Note. All table values are percentages.  
<sup>a</sup> Abuse and dependence combined.

mothers were  $-0.027$  ( $SD = 1.03$ ) and  $-0.001$  ( $SD = 1.02$ ). Compared with the mothers, the fathers had a higher probability of endorsing use of a substance at any given level of severity.

*Drug Use Screening Inventory—Revised (DUSI-R; Tarter, 1990).* This self-report questionnaire, consisting of 149 true-false items, was administered to the boys to document the severity of the problems that are commonly associated with the risk for SUD at age 12–14 and was repeated at ages 16, 19, and 22. Psychometric properties of the DUSI-R have been established using both classical test theory and IRT methods (Kirisci, Hsu, & Tarter, 1994; Kirisci, Mezzich, & Tarter, 1995; Tarter & Kirisci, 2001).

The DUSI-R evaluates severity of problems in the following domains: substance use, psychiatric disorder, health status, behavior patterns, school performance, family system, peer relationships, social competence, work adjustment, and leisure/recreation. The overall problem density score, which can range from 0% to 100%, quantified severity of psychosocial problems encompassing the 10 measurement domains. This score is derived by dividing the number of endorsed problems by the total number of questions and then multiplying the resulting ratio by 100.

*Neurobehavioral disinhibition (Tarter et al., 2003).* The neurobehavioral disinhibition latent trait was derived using indicators of behavior

Table 2  
*Substance Use Disorder (SUD) and Psychiatric Diagnoses of the Boys*

Diagnosis	Current				Lifetime
	Ages 12–14 (n = 351)	Age 16 (n = 293)	Age 19 (n = 183)	Age 22 (n = 91)	Ages 12–22 (n = 351)
Any SUD	1.4	7.2	14.8	37.4	18.5
Alcohol	0.9	3.4	21.3	28.6	13.7
Cannabis	0.9	5.8	16.9	29.7	16.0
CD	5.7	10.6	9.8	1.1	13.4
ODD	5.4	2.0	1.1	1.1	6.6
ADHD	6.8	4.1	3.8	2.2	7.7
Anxiety, child	4.0	2.0	1.1	1.1	5.4
Depression	2.0	3.8	9.3	6.6	10.0

Note. All table values are percentages. CD = conduct disorder; ODD = oppositional defiant disorder; ADHD = attention-deficit/hyperactivity disorder.

undercontrol, affect dysregulation, and executive cognitive functioning. Behavior undercontrol was determined by tabulating the number of symptoms of attention-deficit/hyperactivity disorder, conduct disorder, and oppositional defiant disorder endorsed in the K-SADS interview. Emotion dysregulation was evaluated with a difficult-temperament index derived from the revised Dimensions of Temperament Survey (Windle, 1992) as described previously (Blackson, Tarter, Loeber, Ammerman, & Windle, 1996; Blackson, Tarter, Martin, & Moss, 1994). Executive cognitive capacity was assessed using the Stroop Color-Word Test (Laplante, Everett, & Thomas, 1992; Stroop, 1935), Porteus Maze Test (Porteus, 1965), Vigilance Test (Schneider & Detweiler, 1987), Motor Restraint Test (Parsons, Tarter, & Edelberg, 1972), Forbidden Toys Test (Milich, Loney, & Landau, 1982), and Block Design subtest of the Wechsler Adult Intelligence Scale (Wechsler, 1997). The scores on these tests of executive cognitive functioning are indicators of a unidimensional latent trait (Aytaclar, Tarter, Kirisci, & Lu, 1999) and are associated with SUD risk (Tarter et al., 2003).

*Statistical Analysis*

Growth mixture modeling with binary outcome (SUD presence or absence; Muthén, 2001a, 2001b) was used to delineate distinct ontogenetic trajectories to SUD. A growth mixture model is a combination of a latent growth curve model and a latent class model, as illustrated in Figure 1. Growth mixture modeling simultaneously determines the number of distinct latent classes (i.e., cluster of developmental trajectories); estimates the growth model for each latent class; assigns individuals into distinct latent classes; determines the association between latent classes and covariates;

and determines the association between classes and outcome, which in this project is SUD diagnosis. The trajectory latent class is the latent class variable that may have different latent growth curve parameters (e.g., intercept and slope). This is indicated by arrows from the latent trajectory classes to the growth factors. Membership in the latent class is unobserved; however, the posterior probabilities of class membership (i.e., the conditional probabilities of the binary outcome) and marginal probability of belonging to the latent class can be obtained. Hence, posterior probabilities are used to assign individuals to their respective classes. Multinomial logistic regression predicts the latent class variable using covariates, which in this study is neurobehavioral disinhibition. Furthermore, logistic regression predicts SUD outcome using the latent class variable as a predictor. Mplus software (Muthén & Muthén, 2001) was used to conduct the data analysis. Maximum likelihood method with robust standard errors was used to estimate parameters. The analysis was conducted with the “missing” option; that is, all observations in the data set were used to estimate parameters.

The model tested in this study is depicted in Figure 1. A two-class growth mixture model was based on measurements conducted at four time points, along with four time-invariant covariates (ethnicity, SES, neurobehavioral disinhibition, and parental substance use severity index or SUD diagnosis evaluated at baseline) and one binary latent class outcome indicator variable (son’s SUD diagnosis at age 22). The DUSI-R overall problem density score at ages 12–14, 16, 19, and 22, along with latent growth factors (intercept and slope), comprised the linear latent growth curve model. The path coefficients for intercept were set to 1, whereas the path coefficients for slope were set to 0, 1, 2.5, and 4 in consideration of the time of the assessments.

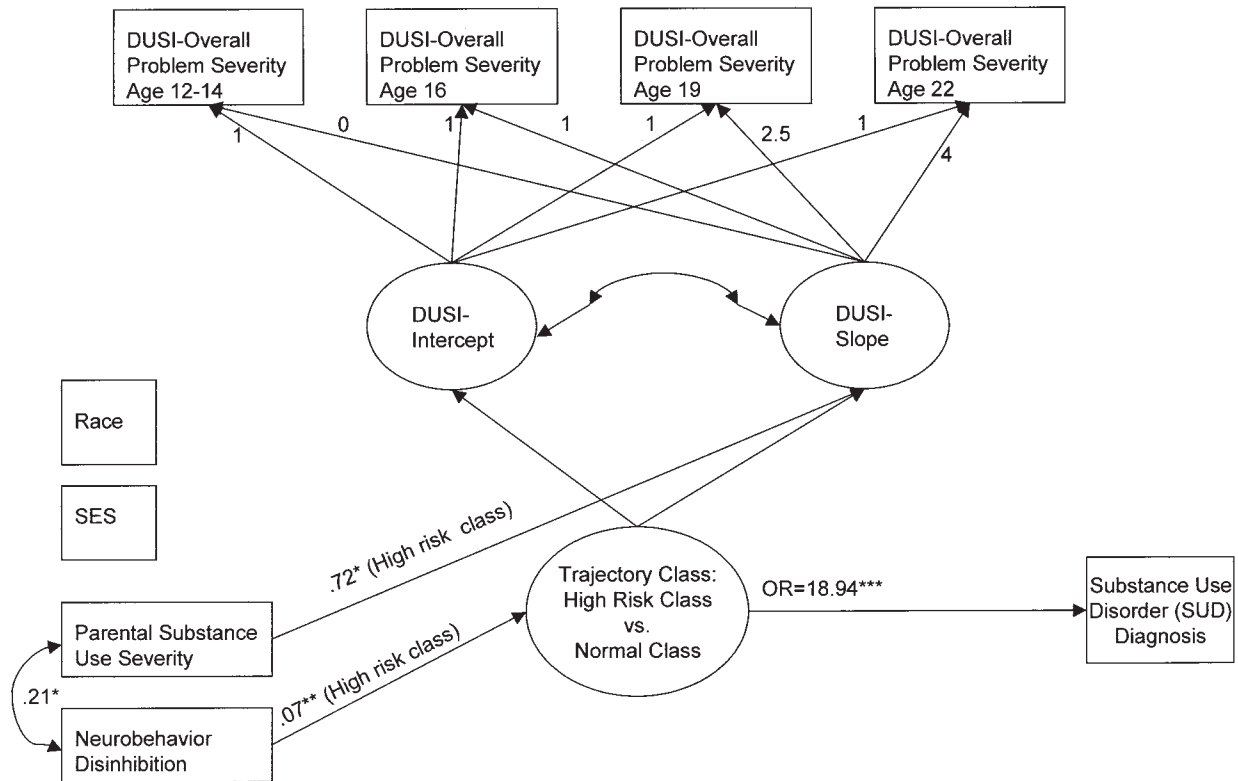


Figure 1. Growth mixture model of Drug Use Screening Inventory—Revised (DUSI-R) overall problem density severity scores with covariates of neurobehavioral disinhibition and parental substance use severity and son’s substance use disorder (SUD) as an outcome. SES = socioeconomic status; OR = odds ratio. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

Results

Correlations Between Predictors and Child's SUD

Diagnosis

Correlations between the child's DUSI-R overall problem density score, neurobehavioral disinhibition score, parental SUI, parental SUD diagnosis, and SUD diagnosis in the boys at age 22 are presented in Table 3. As can be seen, the correlations between the DUSI-R overall problem density scores across the four assessments are highly significant, as are point-biserial correlations between the overall problem density score and parental SUD and child's SUD. Severity of neurobehavioral disinhibition also strongly correlated with the overall problem density score at each time point.

Of note is that the mean score and variance of the overall problem density score on the DUSI-R increased across the four assessments:  $M \pm SD = 16.9 \pm 12.3$  at age 12-14;  $18.0 \pm 12.9$  at age 16;  $19.7 \pm 13.8$  at age 19; and  $20.4 \pm 17.0$  at age 22. Between ages 12-14 and 22, severity of substance use, as measured by the DUSI-R overall problem density score, increased approximately by 25% in the sample.

Model Fitting: Unconditional Models

Unconditional latent growth mixture models (models with no covariates) with binary outcomes specifying two or three latent classes and linear or quadratic growth were first tested to determine the best fitting model. No significant differences were found between models with linear and quadratic growth curves regardless of the number of latent classes in the model, two-class linear versus two-class quadratic model: change in  $\chi^2(5) = 9.6, p = .09$ ; three-class linear versus three-class quadratic model: change in  $\chi^2(7) = 4.4, p = .73$ . To compare the model fit between two-class and three-class mixture linear growth models, the Bayesian information criterion has been recommended (Muthén, 2001b). The Bayesian information criterion difference between the models was trivial (7,492.05 vs. 7,476.20). Thus, the more parsimonious two-class linear growth mixture model was accepted.

In the unconditional linear growth model, the estimated mean initial status (standardized) of the DUSI-R overall problem density score and linear growth rate mean (standardized) were 2.04 ( $z =$

1.96,  $p = .05$ ) and 0.82 ( $z = .93, p = .35$ ), respectively. The residual variances of initial status and rate of change were significantly different from zero (variance of initial status estimate = 46.63,  $z = 2.76, p < .01$ ; variance of rate of change estimate = 6.37,  $z = 2.04, p < .05$ ). The results indicated that participants differed in overall problem severity due to substance use at age 12-14 with a variable rate of change over time. Furthermore, a negative correlation was observed between initial status in overall problem severity and rate of change ( $r = -.47, z = -1.93, p = .054$ ).

Model Fitting: Conditional Models

Fitting a model with maternal and paternal SUI scores as separate variables failed to provide an acceptable data-model fit (data not shown). A likely reason is collinearity due to homogamy (spousal similarity) for SUD liability (Vanyukov, Neale, Moss, & Tarter, 1996). Thus, the scores were summed within parental couples, and the summed parental SUI was used in subsequent analyses. The results of model testing are summarized in Figure 1.

Twenty percent of the participants were classified into the first class, and 80% were classified into the second class. As shown in Table 4, the two classes did not differ in SES or ethnicity. However, as can be seen, severity of the DUSI-R overall problem density score was four to five times greater in Class 1 participants than in Class 2 participants. Referring to Figure 2, it can be seen that participants in Class 2 did not exhibit a significant change in the DUSI-R overall problem density score with increasing age. In contrast, Class 1 participants exhibited a significant increase in severity from age 12-14 to age 22, with the increment occurring between early (age 12-14) and mid-adolescence (age 16), after which problem severity stabilizes. The effect size ( $\eta^2$ ) of the difference in the DUSI-R overall problem density score between Class 1 and Class 2 was large at each assessment: 0.34 at age 12-14, 0.54 at age 16, 0.51 at age 19, and 0.43 at age 22.

Class 1 participants had parents with significantly higher SUI scores than the parents of Class 2 participants (0.46 vs.  $-0.15, p < .01$ ). They also scored higher on neurobehavioral disinhibition (58.24 vs. 49.19,  $p < .001$ ). A ninefold higher rate of lifetime abuse or dependence SUD diagnoses (63.4% vs. 7.1%,  $p < .001$ ) was observed in Class 1 participants. Class 1 and Class 2 were

Table 3

Correlations Between Drug Use Screening Inventory—Revised (DUSI-R), Overall Problem Density (OPD) Score, Neurobehavioral Disinhibition (ND), Parental Substance Use Index (SUI) and Substance Use Disorder (SUD) Diagnosis, and Son's SUD Diagnosis Across the Four Assessments

Variable	1	2	3	4	5	6	7	8	9
1. DUSI-R OPD, ages 12-14	—	.52***	.46***	.53***	.28***	.13*	.12*	.15**	.24***
2. DUSI-R OPD, age 16			.74***	.64***	.24***	.29***	.21***	.23***	.38***
3. DUSI-R OPD, age 19				.81***	.27***	.33***	.27***	.33***	.51***
4. DUSI-R OPD, age 22					.42***	.43***	.40***	.36***	.52***
5. ND						.21***	.13*	.18**	.17**
6. SUI, parents							.71***	.54***	.12*
7. SUD diagnosis, father								.39***	.08*
8. SUD diagnosis, mother									.22***
9. SUD diagnosis, son									—

\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

Table 4  
Distribution of Demographic and Clinical Variables According to the Class 1–Class 2 Dichotomy

Variable	Class 1		Class 2		$\chi^2$	<i>p</i>
	<i>n</i>	%	<i>n</i>	%		
Race, White ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	55	77.5	223	79.6	0.16	.69
Lifetime (SUD) diagnosis, sons ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	45	63.4	20	7.1	118.71	<.001
SUD diagnosis, sons, age 22 ( $n_{Class 1} = 28, n_{Class 2} = 63$ )	23	82.1	11	17.5	34.65	<.001
Alcohol use disorder, sons, age 22	18	64.3	8	12.7	25.28	<.001
Cannabis use disorder, sons, age 22	18	64.3	9	14.3	23.22	<.001
Cocaine use disorder, sons, age 22	4	14.3	1	1.6	3.82	.051
Lifetime SUD diagnosis, fathers ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	39	54.9	124	44.3	2.58	.11
Lifetime SUD diagnosis, mothers ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	31	43.7	56	20.0	17.01	<.001
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>F</i>	<i>p</i>
DUSI–R, ages 12–14 ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	31.16	14.04	13.28	8.73	180.44	<.001
DUSI–R, age 16 ( $n_{Class 1} = 61, n_{Class 2} = 232$ )	35.89	11.35	12.98	7.99	309.87	<.001
DUSI–R, age 19 ( $n_{Class 1} = 47, n_{Class 2} = 136$ )	35.72	12.68	13.65	8.38	172.16	<.001
DUSI–R, age 22 ( $n_{Class 1} = 28, n_{Class 2} = 63$ )	36.32	19.85	12.66	7.61	56.21	<.001
No. SUD symptoms, age 22	6.79	6.02	1.79	3.07	35.28	<.001
Frequency of drug use in last month, age 22	9.67	5.51	4.12	4.04	31.17	<.001
No. of drugs ever tried, age 22	9.43	4.59	5.39	3.52	25.69	<.001
Substance use severity, parent ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	0.46	1.86	–0.15	1.75	6.72	.01
Neurobehavioral disinhibition ( $n_{Class 1} = 71, n_{Class 2} = 280$ )	58.24	12.50	49.19	8.88	49.16	<.001

Note. SUD = substance use disorder; DUSI–R = Drug Use Screening Inventory—Revised.

compared on number of SUD symptoms endorsed on the SCID, frequency of drug use in past month, number of different drugs ever tried, and rate of SUD diagnosis at age 22. As shown in Table 5, parental SUI was the only covariate that was significantly associated with the rate of change of the DUSI–R overall problem density score in the Class 1 participants ( $\beta = 0.72, z = 2.37, p < .05$ ).

**Predicting trajectory class membership.** Neurobehavioral disinhibition score was the only covariate that predicted trajectory class membership ( $\beta = 0.07, z = 2.82, p < .01$ ); that is, the likelihood of belonging to Class 1 compared to Class 2 is significantly increased among youth who have a high neurobehavioral disinhibition score. None of the other covariates were significant

(SUI:  $\beta = 0.13, z = 1.15, p > .05$ ; ethnicity:  $\beta = 0.19, z = 0.38, p > .05$ ; SES:  $\beta = 0.01, z = 0.58, p > .05$ ).

**Predicting SUD diagnosis.** Next, we used growth mixture modeling to estimate logits of qualifying for an SUD diagnosis in Class 1 and Class 2. The logits were 2.49 ( $z = 8.75, p < .01$ ) for Class 1 and –0.44 ( $z = -1.13, p = .26$ ) for Class 2. As a result, the probability of qualifying for SUD diagnosis in the Class 1 participants was .924 (probability =  $1/[1 + e^{-(\text{logit} = 2.498)}]$ ) compared to .391 (probability =  $1/[1 + e^{-(\text{logit} = -0.442)}]$ ) for the Class 2 participants. Accordingly, the odds of developing an SUD diagnosis in Class 1 are 12.16 (odds =  $0.924/0.076; p < .001$ ; 95% confidence interval: 6.95, 21.25), and in Class 2 the odds are 0.64 (odds =  $0.391/0.609; p = .13$ ; 95% confidence interval: 0.30, 1.38). The corresponding odds ratio when comparing Class 1 and Class 2 is 18.94 (odds ratio =  $12.16/0.64$ ).

**Neurobehavioral disinhibition and parental SUD diagnoses as covariates.** An alternative growth mixture model analysis was conducted, replacing the parental SUI with paternal and maternal SUD diagnosis. The results were similar to those obtained in the first analysis: Twenty percent of the participants were classified into Class 1, and 80% were classified into Class 2. The agreement between the two analyses in terms of classification of participants into two classes was 99%. In effect, clustering participants into the two classes using either parental substance use behavior or *DSM–III–R* diagnosis yielded the same results.

Both paternal and maternal SUD diagnoses predicted the slope of the developmental trajectory of the DUSI–R overall problem density score in Class 1 boys, but not in Class 2 boys ( $\beta = 0.41, z = 2.06, p < .05$ , and  $\beta = 0.50, z = 2.20, p < .05$ , respectively). Maternal SUD diagnosis also predicted intercept of the developmental trajectory ( $\beta = -0.66, z = 2.18, p < .05$ ) in Class 1 participants. Furthermore, both neurobehavioral disinhibition score and SUD in the mother predicted trajectory class member-

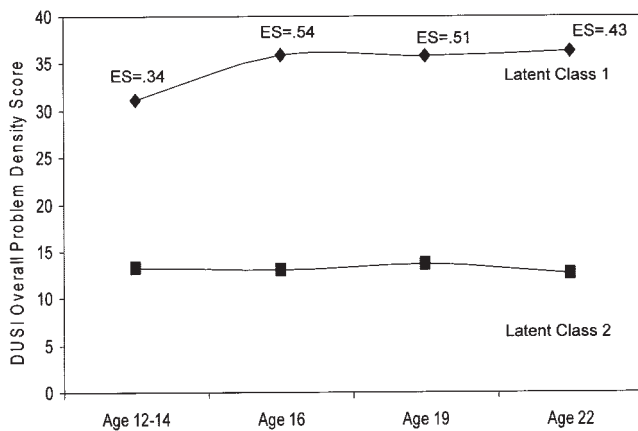


Figure 2. Comparison of high-risk and low-risk groups on the Drug Use Screening Inventory—Revised (DUSI–R) overall problem density severity score. ES = effect size (partial  $\eta^2$ ).

Table 5  
Results of Latent Growth Curve Model

Covariate	Class 1				Class 2			
	Intercept		Slope		Intercept		Slope	
	$\beta$	$z$	$\beta$	$z$	$\beta$	$z$	$\beta$	$z$
Substance use index, parent	-0.03	-0.09	0.72	2.37*	0.17	1.79	-0.01	-0.06
Neurobehavioral disinhibition	-0.12	-0.47	0.14	0.57	0.19	1.38	-0.09	-0.60
Ethnicity	-0.03	-0.11	-0.07	-0.28	-0.08	-0.72	-0.05	-0.48
Socioeconomic status	-0.51	-1.72	0.31	0.89	-0.11	-0.87	-0.01	-0.13

\*  $p < .05$ .

ship (latent class variable;  $\beta = 0.07$ ,  $z = 2.65$ ,  $p < .05$ , and  $\beta = 1.61$ ,  $z = 3.27$ ,  $p < .01$ , respectively); that is, the likelihood of belonging to Class 1 compared to Class 2 is significantly increased among youth having SUD mothers and a high neurobehavioral disinhibition score. Finally, the probability of qualifying for SUD diagnosis was .92 in Class 1 participants and .39 in Class 2 participants.

*Excluding substance use domain from DUSI-R overall problem density score.* The analyses were conducted again after removing the substance use domain responses from the DUSI-R overall problem density score. The results were compared with the results of the first analysis in which all 10 DUSI-R domains were used in the analysis. The results were similar: Twenty-two percent of the participants were classified into the high-risk class, and 78% were classified into the low-risk class. Agreement between the first analysis and this analysis was 99%. Thus, excluding the DUSI-R substance use domain from the overall problem density score did not change the assignment of participants into the two classes. Similar to the results obtained in the first analysis, the parental SUI predicted the slope of the developmental trajectory of the DUSI-R overall problem density score in the high-risk class ( $\beta = 0.69$ ,  $z = 2.33$ ,  $p < .05$ ), but not in the low-risk class. Furthermore, neurobehavioral disinhibition predicted trajectory class membership ( $\beta = 0.09$ ,  $z = 3.10$ ,  $p < .01$ ). Finally, the probability of qualifying for an SUD diagnosis was .92 in the high-risk class and .43 in the low-risk class.

*Predicting SUD at age 22 using individual DUSI-R domains assessed at ages 12–14, 16, 19, and 22.* Four separate logistic regression analyses were conducted to predict SUD diagnosis at age 22 using the 10 DUSI-R domains measured at ages 12–14, 16, 19, and 22. At age 12–14, peer relationships significantly predicted SUD at age 22 ( $\beta = 0.05$ ,  $p < .01$ ). The overall  $R^2$  was .17 ( $p < .01$ ). At age 16, social competence ( $\beta = -0.08$ ,  $p = .03$ ) and work adjustment ( $\beta = 0.10$ ,  $p = .04$ ) predicted SUD at age 22. The overall  $R^2$  was .34 ( $p < .01$ ). At age 19, three domains significantly predicted SUD at age 22: psychiatric disorder ( $\beta = 0.06$ ,  $p = .03$ ), social competence ( $\beta = -0.06$ ,  $p = .04$ ), and school performance ( $\beta = 0.08$ ,  $p < .01$ ). The overall  $R^2$  was .40 ( $p < .01$ ). Finally, at age 22, substance use ( $\beta = 0.08$ ,  $p < .01$ ), social competence ( $\beta = -0.11$ ,  $p = .02$ ), family system ( $\beta = 0.08$ ,  $p < .01$ ), and work adjustment ( $\beta = 0.09$ ,  $p = .02$ ) were significant predictors. The overall  $R^2$  was .46 ( $p < .01$ ). In effect, the capacity of the DUSI-R to predict SUD at age 22 increases along with the number of salient domains as the participants become older.

## Discussion

Individual risk for SUD—that is, the phenotype for liability to SUD—undergoes a complex process of ontogenesis involving the interplay of organismic characteristics (genetic, biochemical, physiological, psychological) and environmental factors (Vanyukov et al., 2003). This study attempted to model certain important aspects of this process, specifically the contribution of parental SUD liability and offspring’s psychological characteristics pertaining to self-regulation.

The results of latent growth mixture analysis revealed that neurobehavioral disinhibition is significantly associated with a latent class variable that aggregates male participants into two classes. Class 1 participants are at extremely high risk (.92) for succumbing to SUD, wherein neurobehavioral disinhibition and parental SUD are conjointly operating in the context of increasing psychosocial problems between ages 12–14 and 22. The Class 2 participants are also at considerable risk (.39) for developing SUD based on neurobehavioral disinhibition and parental SUD; however, a lower and more stable level of psychosocial problems characterizes the ontogenetic trajectory to SUD outcome at age 22. These findings indicate that addressing psychosocial problems during the critical transition through adolescence among youth featured by high neurobehavioral disinhibition and parental SUD may lower the risk of succumbing to SUD by young adulthood.

Consistent with transmissibility of SUD liability (Kendler, Jacobson, Prescott, & Neale, 2003), it was found that parental SUD diagnosis influenced child’s increment in severity of psychosocial problems. Both paternal and maternal diagnosis influenced the rate of growth of substance use related and other behavioral disturbances as measured by the DUSI-R overall problem density score in Class 1 participants. Only maternal SUD predicted the initial level (intercept) of these problems. In addition, maternal but not paternal SUD diagnosis, along with neurobehavioral disinhibition, predicted the trajectory of Class 1 and Class 2 participants. It should be noted, however, that these relationships may be somewhat confounded by the fact that the proband in this study is the father, who is either affected (SUD) or is psychiatrically normal. Thus, the mother’s SUD may be secondary to her husband’s because of assortative mating, contagion, or both (Vanyukov et al., 1996) and reflect, in addition to her own influence, paternal contribution. Contribution of contagion (direct influence of a spouse’s behavior on the behavior of the other spouse) is likely asymmetric, being mainly the direct influence of the husband’s

drug use on his mate's drug use, thus possibly amplifying the maternal effect. The same explanation may be valid for the results of testing separately the influences of paternal and maternal substance use indices. Whereas the sum of the paternal and maternal scores reflects sources of mate similarity, and is associated with an increment in the child's behavioral problems, the models accounting for the paternal and maternal indices separately fail to yield a reasonable data-model fit.

Latent growth mixture modeling, used to identify a latent class variable aggregating male offspring into high- and low-risk groups, also revealed that the odds of qualifying for SUD diagnosis in the Class 1 group, consisting of 20% of the sample, are 12.16. The observed parental contribution to the variation in the increment of substance use problems is consistent with significant transmissibility of SUD liability, including its heritability (see Vanyukov & Tarter, 2000, for a review).

The main finding of this study is that two latent classes can be delineated, both of which are associated with amplified risk for SUD. The two classes have, respectively, a .92 and .39 probability of developing SUD. These findings concur with an accumulating body of evidence demonstrating that early age onset SUD is a developmental outcome (Clark & Winters, 2002; Vanyukov et al., 2003) featured by failure to acquire self-regulatory control. This disturbance likely has a neurobiological basis. Many studies have documented amplitude attenuation of the P300 component of the event-related potential in high-risk youth (see Begleiter & Porjesz, 1999, for a review). Recent research conducted on a large proportion of the present sample demonstrated that neurobehavioral disinhibition mediates the association between P300 amplitude attenuation in childhood (age 10–12) and SUD manifest by age 19 (Habeych, Charles, ScLabassi, Kirisci, & Tarter, 2005). Moreover, source density analyses have revealed that the amplitude reduction is most pronounced over the frontal convexity (Bauer & Hesselbrock, 1999, 2001). Significantly, the P300 wave is widely recognized as comprising a neurophysiological indicator of attention, working memory, and information-processing efficiency (Berman & Friedman, 1995; Cykowicz, 2000). This latter finding provides confirmation of the hypothesis that prefrontal cortex dysfunction is an integral component of SUD liability (Tarter et al., 1999) and substantiates the conclusion that the neurobehavioral disinhibition construct derived in this study is an accurate indicator of disrupted neurologic functioning.

Several limitations of this investigation potentially limit generalizability of the findings. First, it should be recognized that ascertainment of the children was through affected and nonaffected proband fathers. Although this factor may have biased the results, it should be noted that comparison of the children in the CEDAR sample with youth surveyed in epidemiological studies has not revealed systematic differences (Tarter & Vanyukov, 2001). Second, the sample, as noted above, was confined to males. Gender differences have been frequently documented in research on SUD etiology; thus, it is important to caution against assuming that the two latent classes observed in males also apply to females. As the sample of females increases, this issue will be directly addressed in future research. Third, although neurobehavioral disinhibition significantly correlates with substance use frequency, other facets of substance consumption, including behavioral pharmacological effects, motive state, social context, and beliefs, remain to be elucidated. Fourth, it should be noted that growth

mixture modeling does not afford the opportunity to determine whether neurobehavioral disinhibition mediates the effects of other nondiagnostic variables. Last, the model tested in this investigation should be viewed as provisional; as additional variables are identified that are salient to SUD liability, the theoretical framework will undoubtedly be modified to reflect our increasingly comprehensive understanding of etiology.

The importance of this investigation resides in the demonstration that individuals with early age onset SUD can be subdivided into two groups differing in their phenotypic developmental trajectories. From the perspective of prevention, it would appear to be important to monitor youth during adolescent development so as to identify those who are at high risk and, accordingly, apply interventions that are tailored to the pattern and severity of psychosocial problems manifest during this critical period. Augmentation of psychosocial problems during adolescence in the context of parental SUD and childhood neurobehavioral disinhibition, as shown herein, is associated with exceptionally high probability (.93) of succumbing to SUD by age 22.

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Received April 22, 2004

Revision received September 9, 2004

Accepted September 13, 2004 ■

### New Editors Appointed, 2007–2012

The Publications and Communications (P&C) Board of the American Psychological Association announces the appointment of three new editors for 6-year terms beginning in 2007. As of January 1, 2006, manuscripts should be directed as follows:

- *Journal of Experimental Psychology: Learning, Memory, and Cognition* ([www.apa.org/journals/xlm.html](http://www.apa.org/journals/xlm.html)), **Randi C. Martin, PhD**, Department of Psychology, MS-25, Rice University, P.O. Box 1892, Houston, TX 77251.
- *Professional Psychology: Research and Practice* ([www.apa.org/journals/pro.html](http://www.apa.org/journals/pro.html)), **Michael C. Roberts, PhD**, 2009 Dole Human Development Center, Clinical Child Psychology Program, Department of Applied Behavioral Science, Department of Psychology, 1000 Sunnyside Avenue, The University of Kansas, Lawrence, KS 66045.
- *Psychology, Public Policy, and Law* ([www.apa.org/journals/law.html](http://www.apa.org/journals/law.html)), **Steven Penrod, PhD**, John Jay College of Criminal Justice, 445 West 59th Street N2131, New York, NY 10019-1199.

**Electronic manuscript submission.** As of January 1, 2006, manuscripts should be submitted electronically through the journal's Manuscript Submission Portal (see the Web site listed above with each journal title).

Manuscript submission patterns make the precise date of completion of the 2006 volumes uncertain. Current editors, Michael E. J. Masson, PhD, Mary Beth Kenkel, PhD, and Jane Goodman-Delahunty, PhD, JD, respectively, will receive and consider manuscripts through December 31, 2005. Should 2006 volumes be completed before that date, manuscripts will be redirected to the new editors for consideration in 2007 volumes.

In addition, the P&C Board announces the appointment of **Thomas E. Joiner, PhD** (Department of Psychology, Florida State University, One University Way, Tallahassee, FL 32306-1270), as editor of the *Clinician's Research Digest* newsletter for 2007–2012.