Adolescent Stressors, Psychopathology, and Young Adult Substance Dependence: A Prospective Study*

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ABSTRACT. Objective: There is much theory, but sparse empirical evidence, supporting the notion that internalizing symptoms and negative affect are the mechanism by which exposure to stressful life events influence the development of substance-use disorders in adolescence and young adulthood. However, many empirical studies have shown that, in addition to elevations in internalizing symptoms, exposure to stressful life events also produces elevations in externalizing behaviors and conduct problems, which are important risk factors for substance-use disorders. The current study tested adolescent externalizing and internalizing symptoms as competitive mediators of the effects of stressors on young adult drug dependence. **Method:** Data from an ongoing study of children of alcoholics ($n = 223$) and matched controls ($n = 204$) were collected in

two annual interviews in adolescence and two follow-ups in young adulthood. **Results:** Experiencing stressful life events during adolescence led to increases in both externalizing and internalizing symptoms, but only externalizing symptoms mediated the later effects of adolescent stressors on young adult drug dependence. **Conclusions:** These findings suggest that understanding how stressors produce elevations in behavioral problems may provide important insights into understanding how broad environmental risk factors lead to substance dependence and suggests that processes other than affect regulation may operate in the pathway from the experiences of stressors to substance use and disorder. (*J. Stud. Alcohol Drugs* 69: 629-638, 2008)

THE PREVALENCE OF SUBSTANCE DEPENDENCE peaks during young adulthood (Substance Abuse and Mental Health Services Administration, 2002), yet theories of the etiology of substance dependence consistently point to behavioral, environmental, and physiological precursors in childhood and adolescence (Sher, 1991). Stressful life events have commonly been identified as a risk factor for psychopathology in general (McMahon et al., 2003) and substance use and dependence specifically (Cerbone and Larison, 2000; Sinha, 2001; Wills and Filer, 1996). However, there have been no prospective studies linking the occurrence of stressors during adolescence with the development of new substance-use disorders by young adulthood. Moreover, the most commonly hypothesized mechanism (affect regulation or self-medication) by which stressors are thought to lead to substance use has mixed empirical support among adolescents (Chassin et al., 2003). The current study explores the prospective effects of stressors on adolescent internalizing and externalizing symptomatology and young adult substance dependence, and tests a novel mediator of the effects of stressors.

Stressful life events, substance use, and disorder

Cross-sectional and longitudinal evidence has demonstrated that both adults and adolescents who experience stressful life events exhibit increased substance use (see Cerbone and Larison, 2000; Sinha, 2001). For example, Havey and Dodd (1995) showed that the most important predictor of early alcohol and drug use among a sixth-grade community sample was negative life events. Stressful life events have also been linked to increased drug use among adolescents over time (e.g., Hoffmann et al., 2000; Wills et al., 2001).

However, substance use does not necessarily result in clinical substance-use disorders, and the predictors of substance use and substance-use disorders are not necessarily identical. There has been less research on the role of stressful life events in the development of substance-use disorders, and most existing data come from clinical samples of adult alcoholics (e.g., Clark et al., 1997) or cross-sectional samples of adolescents (Steinhausen and Metzke, 2003) or adults (McCreary and Sadava, 1999). Sher and colleagues (e.g., Jackson and Sher, 2003; Sher et al., 1997) demonstrated that retrospectively reported childhood stressors differentiated participants with adult alcohol-use disorder (and alcohol-use disorder comorbid with tobacco dependence) from those with no diagnoses. Moreover, Wills and colleagues (2002) demonstrated that the relation between substance use and problems was stronger among adolescents who experienced higher life stressors. Some prospective studies have demonstrated that children and adolescents who experience physical or sexual abuse are more likely to develop substance-use disorders in adolescence and young adulthood (e.g., Clark et

Received: January 2, 2008. Revision: April 2, 2008.

*This research was supported by National Institute on Alcohol Abuse and Alcoholism grant AA016213 and a National Research Service Award from National Institute on Drug Abuse grant DA019753 to Kevin M. King. A version of these analyses was presented at the 2006 meeting of the Society for Research on Adolescence conference in San Francisco, CA.

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al., 2003; Widom et al., 1995), but little research has examined the effects of exposure to other kinds of stressful life events, leaving it unclear whether it is the stressful nature of abuse or some other related characteristic that drives the observed relation. Thus the first goal of the current study was to test the effects of adolescent life events on the development of drug dependence by young adulthood. We focused on the development of drug dependence, rather than alcohol dependence, because recent research has suggested that drug dependence, which very commonly co-occurs with alcohol dependence in young adults, represents a more severe and pathological form of substance dependence than alcohol dependence that occurs in the absence of a drug-dependence disorder (Dick et al., 2007).

Theoretical mediators

Negative affect. Multiple theories have proposed that stress leads to increases in substance use as a means of reducing stress-related negative affect and increasing positive affect, which itself becomes reinforcing as a coping mechanism (Sinha, 2001), leading to escalations in substance use and potentiating substance-use disorders, for example, Cloninger's (1987) Type I alcoholism, Wills and Filer's (1996) stress-coping model, Sher's (1991) stress-negative affect model, and general strain theory (Agnew, 1992). Broadly, each theory hypothesizes that exposure to stressors produces elevations in negative emotions (or internalizing symptoms), and these increases in negative emotions in turn lead to increased substance use and risk for substance-use disorders. However, the few prospective studies of the effects of adolescent internalizing symptoms on substance-use disorders have shown no effects (e.g., Chassin et al., 1999) or have suggested that only certain aspects, such as anger, are the specific mechanism of effect (McCreary and Sadava, 1999; Tarter et al., 1995). For example, anger has been theorized to lead to substance use either through poor adaptive coping skills (Wills et al., 1999) or through affiliation with deviant peers (Swaim et al., 1989).

Some of the reason for the lack of findings could be that adolescents may increase their substance use immediately following the experience of stress-related negative affect, but it is possible that these temporary escalations in use may not be detectable in the long term or that this stress-induced substance use may not substantially contribute to escalations in substance use that lead to disorder. Alternately, given that adolescent substance use differs significantly in prevalence, effects, and etiology from adult substance use (Hawkins et al., 1992; Johnston et al., 2005), processes that may strongly influence adult substance use (such as tension-reduction motives; Conger, 1956) may be overshadowed during adolescence by stronger influences, such as peer influence or family effects. For example, alcoholism subtypes such as Cloninger's (1987) Type I alcoholism and Del Boca and

Hesselbrock's (1996) internalizing type, which prominently feature negative affect, are proposed to onset in adulthood rather than adolescence. In general, the weak empirical support for the effects of negative affect on the development of substance use and substance-use disorders in adolescence and young adulthood suggests either that the observed effects of stressful life events on substance dependence are spurious or that they are mediated via an alternative pathway.

Externalizing behaviors. Reviews of the empirical literature indicate that the effects of exposure to stressors on child and adolescent psychopathology are broad and nonspecific (McMahon et al., 2003). Exposure to stressful life events has been shown to produce elevations in not only negative affect but also behavior problems in children and adolescents (Aseltine et al., 2000; Grant et al., 2004). In turn, adolescents who exhibit high levels of externalizing behaviors have been shown to initiate substance use earlier (King and Chassin, 2007; McGue et al., 2001), use higher amounts and more types of substances during adolescence (see reviews by Chassin et al., 2003; Hawkins et al., 1992), and be more likely to develop substance abuse and dependence (Cloninger, 1987; Iacono et al., 1999; Kreuger et al., 2002). Moreover, Wills and colleagues have repeatedly demonstrated that the experience of negative life events is related to affiliation with deviant peers and peers' substance use (e.g., Wills et al., 1999), both of which are influenced by behavioral problems and proximal predictors of substance use (Hawkins et al., 1992). Thus it could be that stressful life events influence the development of substance-use disorders because they increase behavioral problems rather than because they increase negative emotions. However, there have been no empirical studies demonstrating a pathway to substance dependence via externalizing symptoms. Thus the second goal of the current study was to test whether stressful life events might lead to later drug dependence via their influence on adolescent externalizing symptoms rather than internalizing symptoms.

In short, the current study aimed to provide a prospective test of the relations among adolescent stressors, internalizing and externalizing symptoms, and young adult drug dependence in a high-risk sample. The current study asked two important questions:

1. Does exposure to stressful life events in adolescence increase the risk for drug dependence during young adulthood?
2. Is the effect of adolescent life events on young adult drug dependence mediated via increases in externalizing or internalizing symptomatology during adolescence?

Method

Participants

Participants were from an ongoing longitudinal study of familial alcoholism (Chassin et al., 1991, 1996, 1999, 2004).

At Time 1, there were 454 adolescents ($\text{mean}_{\text{age}} [\text{SD}] = 13.18 [1.44]$ years, range: 10.5-15.5); 246 of these had at least one biological, custodial alcoholic parent (these adolescents were called *children of alcoholics* [COAs]), and the remaining 208 were demographically matched adolescents with no biological or custodial alcoholic parents (controls).

COA families were recruited using court records of driving under the influence arrests, health maintenance organization wellness questionnaires, and community telephone screening. Direct interview data from the computerized Diagnostic Interview Schedule (DIS, Version III; Robins et al., 1981) confirmed that a biological and custodial parent met diagnostic criteria for lifetime alcohol abuse or dependence per criteria listed in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III, American Psychological Association, 1980). Demographically matched control families were recruited using telephone interviews. When a COA participant was recruited, reverse directories were used to locate families living in the same neighborhood. Families were screened to match the COA participant in ethnicity, family structure, target child's age (within 1 year), and socioeconomic status (using the property value code from the reverse directory). Structured interviews were used to confirm that neither parent met lifetime DSM-III criteria for alcohol abuse or dependence.

A complete description of sample recruitment and representativeness is reported elsewhere (Chassin et al., 1991, 1992). The sample was unbiased with respect to alcoholism indicators available in archival records (e.g., blood alcohol concentrations recorded at the time of the arrest; see Chassin et al., 1992, for details). Moreover, the alcoholic sample had rates of other psychopathology similar to those reported for a community-dwelling alcoholic sample (Helzer and Pryzbeck, 1988). However, those who refused participation were more likely to be Hispanic, suggesting some caution in generalization.

There were three annual assessments (Time 1, Time 2, and Time 3: $\text{median}_{\text{age}} = 13, 14, 15$ years, respectively) of the adolescent participants and their parents and two long-term follow-ups (Time 4 and Time 5). The follow-ups were conducted when participants were in emerging adulthood (Time 4: $\text{median}_{\text{age}} = 20$ years) and in young adulthood (Time 5: $\text{median}_{\text{age}} = 25$ years). Sample retention was excellent at both follow-ups (Time 4: $n = 407$, 90% of the total sample; Time 5: $n = 411$, 91% of the total sample). Retention was unbiased by gender and ethnicity, but slightly more COAs than controls were lost at Time 4 ($\chi^2 = 5.45$, 1 df, $p < .05$, $n = 454$) and at Time 5 ($\chi^2 = 4.12$, 1 df, $p < .05$, $n = 454$).

Procedure

Interviews were conducted at the family's residence or on campus. At all waves, trained project personnel used laptop computers to enter data. Interviewers read items aloud, and

participants could either enter responses themselves or respond verbally to questions. In most cases, family members were interviewed simultaneously but in different rooms to avoid threats of contamination and to increase privacy. Interviewers were unaware of the family's group membership. To further encourage honest responding, confidentiality was reinforced with a Department of Health and Human Services Certificate of Confidentiality. Interviews lasted approximately 1-3 hours, and participants were paid up to \$70 over the waves.

Selection of the current sample

Data from the Time 1, 2, 3, 4, and 5 assessment periods were used in the current analyses. Table 1 provides a summary of the variables used by assessment period, and Table 2 provides a summary of the participants' demographic information. Participants were included in analyses if they did not already meet criteria for drug-use disorder in adolescence ($n = 13$) and were not missing substance dependence data from the Time 4 or Time 5 follow-up ($n = 14$, final $n = 427$, 94% of the total sample). We compared included participants ($n = 427$) with excluded participants ($n = 27$) on all covariates and predictors using chi-square tests and t tests. Excluded participants were more likely to be COAs (85% of those excluded vs 52% of those included) ($\chi^2 = 11.11$, 1 df, $p < .01$, $n = 454$), have an antisocial parent (30% of those excluded vs 7% of those included) ($\chi^2 = 16.18$, 1 df, $p < .001$, $n = 454$), be older at the initial assessment ($\text{mean}_{\text{excluded}} = 13.89 [1.18]$ vs $\text{mean}_{\text{included}} = 13.18 [1.44]$) ($t = 2.50$, 452 df, $p < .01$), have experienced more stressful life events at either time point ($\text{mean}_{\text{excluded}} = 3.00\text{-}3.20 [2.01\text{-}2.50]$ vs $\text{mean}_{\text{included}} = 1.81\text{-}1.96 [1.67\text{-}1.70]$) ($t \geq 3.12$, 452 df, $p < .01$), and have somewhat more externalizing symptoms ($\text{mean}_{\text{excluded}} = 1.28\text{-}1.36 [0.52\text{-}0.53]$ vs $\text{mean}_{\text{included}} = 1.00\text{-}1.02 [0.35\text{-}0.36]$) ($t \geq 3.35$, 452 df, $p < .01$) and internalizing symptoms ($\text{mean}_{\text{excluded}} = 2.41\text{-}2.53 [0.59\text{-}0.68]$ vs $\text{mean}_{\text{included}} = 2.13\text{-}2.14 [0.72\text{-}0.74]$) ($t \geq 1.98$, 452 df, $p < .05$) during adolescence. This suggests that we excluded the most extreme adolescent participants, consistent with a subsample of participants that included individuals who already met criteria for a substance-use disorder in adolescence.

Measures

Parental psychopathology. At Time 1, lifetime DSM-III diagnoses of parental alcoholism (abuse or dependence), depression, and antisociality were assessed with the DIS (Robins et al., 1981). For noninterviewed parents (for 19.3% of fathers, 7.7% of mothers), lifetime alcoholism diagnoses were established using the Family-History Research Diagnostic Criteria (Version 3; Endicott et al., 1975) based on spousal reports. For the present analyses, diagnoses were dichotomous: either present (at least one biological and

TABLE 1. Variables by time of assessment

Variable	Time 1	Time 2	Time 3	Time 4	Time 5
Mean age	13.17	14.17	15.1	20.37	25.70
(SD)	(1.44)	(1.44)	(1.44)	(1.36)	(1.61)
Life events		X	X		
Externalizing symptoms		X	X		
Internalizing symptoms		X	X		
Drug-dependence diagnosis			X	X	X
Parental alcoholism	X				
Parental depression	X				
Parental antisociality	X				

Notes: Drug-dependence diagnoses were used at Time 3 to screen out already diagnosed participants. Young adult dependence was defined as a positive lifetime diagnosis at Time 4 or Time 5.

custodial parent met lifetime criteria) or absent (neither biological parent met lifetime criteria).

Stressful life events. At Times 2 and 3, the adolescent's experience of stressful life events in the past year was assessed with 18 self-report items adapted from the General Life Events Schedule for Children (GLESC; Sandler et al., 1986). Because Pillow et al. (1998) showed that items from the GLESC relating to illness and bereavement had unique effects distinct from the other GLESC items, we dropped those 5 items for the current analyses to facilitate interpretation of the latent factors, leaving 13 life event items assessed at each time point. These items were judged by expert raters to be both negative and uncontrollable events from the perspective of the adolescent (Sandler et al., 1986); all items are listed in Table 3. Broadly, these events reflect life events that happen to the parents and the family that are beyond the immediate control of the adolescent (such as parental financial difficulty or divorce). This measure has been well established in the literature (Grant et al., 2004) and has been shown to predict increases in adolescent substance use in the current sample (Chassin et al., 1996; Pillow et al., 1998) and

to be related to adolescent negative affect and parent alcoholism (Pillow et al., 1998). Adolescents reported at each wave whether each of the 13 events had occurred within the past year.

Child psychopathology and externalizing and internalizing symptomatology. At Times 2 and 3, participants and their mothers reported on the participants' past-year externalizing symptomatology (22 items, excluding drug and alcohol items; e.g., rebellious, stole things, mean or cruel to others, and destroyed property) and internalizing symptomatology (7 items; e.g., felt lonely, cried a lot, felt worthless, felt worried, and felt fearful/anxious) using the Achenbach Childhood Behavior Checklist (Achenbach and Edelbrock, 1978). Coefficient α 's were .89 for participant self-report of externalizing, .78 for internalizing, .81 for mother report of externalizing, and .75 for mother report of internalizing. Based on a moderate correlation between participant and maternal report of externalizing symptoms ($r = .42$), we used the mean of these scale scores to reduce shared source variance. However, because participants' self-report and mothers' self-report of internalizing were not significantly

TABLE 2. Demographic characteristics of the current sample ($n = 427$)

Variable	Parental alcoholism status		
	Total ($N = 427$)	Non-COA ($n = 204$)	COA ($n = 223$)
Females, %	46.80	45.60	48.00
Parental antisociality, %	7.30	0.50 [‡]	13.50 [‡]
Parental depression, %	5.60	2.00 [†]	9.00 [†]
Lifetime drug dependence, %	19.20	11.80 [‡]	26.00 [‡]
	Mean (SD)	Mean (SD)	Mean (SD)
Age at Time 1	13.18 (1.44)	13.26 (1.45)	13.10 (1.43)
Externalizing symptoms, Time 2	1.00 (0.35)	0.89 (0.27) [‡]	1.09 (0.39) [‡]
Externalizing symptoms, Time 3	1.02 (0.36)	0.94 (0.31) [‡]	1.10 (0.38) [‡]
Internalizing symptoms, Time 2	2.14 (0.74)	2.04 (0.74) [†]	2.24 (0.73) [†]
Internalizing symptoms, Time 3	2.13 (0.72)	2.04 (0.67) [*]	2.22 (0.77) [*]
General life events, Time 2	1.97 (1.70)	1.61 (1.50) [‡]	2.31 (1.81) [‡]
General life events, Time 3	1.81 (1.67)	1.50 (1.51) [‡]	2.10 (1.75) [‡]

Notes: COA = child of alcoholic.

*Difference is significant, $p < .05$; [†] $p < .01$; [‡] $p < .001$.

TABLE 3. Stressful life events assessed in the current study

Your brother or sister had serious trouble (with the law, school, drugs, etc.).
Your close friend had serious troubles, problems, illness, or injury.
Your mom or dad talked about having serious money troubles.
Your relatives said bad things about your mom or dad.
Your mom or dad fought or argued with your relatives.
People in your neighborhood said bad things about your mom or dad.
Your mom or dad acted badly in front of your friends.
Your mom or dad was arrested or sent to jail.
Your mom or dad lost their job.
You changed schools because of a family move.
A close friend of yours moved away.
Your mom or dad got divorced or separated.
You were the victim of a crime.

correlated, participants' self-reports were chosen as the more reliable indicator of their own internal state (Achenbach et al., 1987).

Adolescents' and young adults' alcohol- and drug-dependence diagnoses. Adolescents' lifetime drug-abuse and -dependence diagnoses were assessed at Time 3 using parents' reports from the Diagnostic Interview for Children and Adolescents (DICA) parent interview (Herjanic and Reich, 1982). These diagnoses were used to exclude participants who met lifetime criteria for drug-use disorder during adolescence. At Times 4 and 5, participants' lifetime DSM-III-R drug-dependence diagnoses (of eight different classes of drugs, such as marijuana, cocaine, or opiates) were obtained with a computerized version of the DIS (CDIS-III-R; Robins et al., 1981). Because of concerns about the validity of DSM-III-R substance-abuse diagnoses (Pollock et al., 2000), participants ($n = 12$) who met criteria for drug abuse only (but not dependence) at either wave were considered to be undiagnosed (which may produce conservative estimates of the effects in the current models). The rate of lifetime drug dependence, diagnosed at either wave, was 21.6%, with the majority of participants being diagnosed with cannabis dependence (68% of those meeting criteria), amphetamine dependence (48%), cocaine dependence (21%), or hallucinogen dependence (17%). The majority of participants (56%) met criteria for dependence on a single drug, whereas 31% met dependence criteria for two drugs, and 13.4% met criteria for three or more drugs. As expected in a study that oversamples individuals at high risk, these prevalences were higher than national data. For example, 9% of National Comorbidity Survey participants ages 18-25 met criteria for drug dependence (Kessler, 2002). Finally, drug-dependent participants were much more likely than nondependent participants to meet criteria for other psychiatric disorders. Those meeting drug-dependence criteria were significantly more likely to have a lifetime diagnosis of an anxiety (48% of those with drug dependence vs 22% of those without drug dependence [$\chi^2 = 21.34$, 1 df, $p < .001$, $n = 427$]), depressive (42% vs 14% [$\chi^2 = 31.44$, 1 df, $p < .001$, $n = 454$]), or alco-

hol-dependence (66% vs 22% [$\chi^2 = 60.09$, 1 df, $p < .001$, $n = 454$]) disorder.

Results

Analytic strategy

Descriptive data analyses were performed using SPSS 13.0 (SPSS Inc., Chicago, IL), and the hypotheses were tested using SPSS 13.0 and Mplus 3.1 with the maximum likelihood estimator with robust standard errors (MLR) estimator for continuous outcomes and weighted least squares means and variances (WLSMV) adjusted for categorical outcomes (Muthén and Muthén, 2004). These estimators are well suited for the natural skewness observed in our symptom and dependence data. Because a few ($n = 6$) participants were missing life events data from Time 3, we accounted for missing data by using full information maximum likelihood estimation, using the expectation maximization (EM) algorithm, assuming ignorable missingness at random (Little and Rubin, 1987; Muthén and Muthén, 1998). Chi-square, Tucker-Lewis index (TLI), comparative fit index (CFI), and root mean square error of approximation (RMSEA) were used to assess model fit based on the guidelines provided by Hu and Bentler (1999) and the cautions of Marsh et al. (2004).

For all analyses, we controlled for the effects of age, parental psychopathology, and gender and tested for interactions between the covariates and the predictors and hypothesized mediators (stressors and externalizing and internalizing symptoms). We tested the indirect effects of life events on substance dependence via adolescent symptoms using the product of coefficients method and the Sobel standard error (MacKinnon and Dwyer, 1993; MacKinnon et al., 2004). Although recent research has suggested that the bias-corrected bootstrap method is the most powerful test of mediation for continuous outcomes, this method has not been well established for use with categorical outcomes (see MacKinnon et al., 2002; Preacher and Hayes, 2004).

Prospective effects of adolescent stressors on young adult substance-use disorders

We first examined the effects of stressful life events on drug dependence using multiple logistic regression in SPSS 15.0 (SPSS Inc., Chicago, IL), controlling for age, gender, parental alcoholism, antisociality, and depression. As summarized in Table 4 and demonstrated by previous studies using data from the same larger sample (e.g., King and Chassin, 2004, 2007; Zhou et al., 2006), having alcoholic parents (odds ratio [OR] = 2.32, $p < .01$) was related to higher odds of developing drug dependence, and being male (OR = 1.57, $p < .10$) was related to somewhat higher odds of drug dependence. Experiencing an increase of one stressful

TABLE 4. The prospective effects of adolescent life events on young adult drug dependence

Adolescent life events	Drug dependence		
	B (SE)	Wald χ^2	OR
Intercept	-0.73 (1.20)	0.37	0.48
Time 1 age	-0.15 (0.09)	2.71	0.86
Gender	0.45 [§] (0.26)	2.98	1.57
Parental alcoholism	0.84 [†] (0.28)	8.95	2.32
Parental antisociality	0.13 (0.43)	0.08	1.13
Parental depression	-0.10 (0.51)	0.04	0.91
Stressful life events, Time 2	0.23 [‡] (0.07)	9.40	1.25

Notes: OR = odds ratio.

[§]Coefficient approaches significance, $p < .05$; [†]coefficient is significant, $p < .01$; [‡] $p < .001$.

life event in adolescence increased the odds of developing a drug-dependence disorder by young adulthood by 25% (OR = 1.25, $p < .001$), over and above the covariates.

Mediational role of internalizing and externalizing symptoms

We next tested whether the effects of adolescent stressors on young adult drug dependence were mediated by changes in adolescent symptoms. Using path analyses with the MLR estimator in Mplus 3.13 (Muthén and Muthén, 2004), we first tested the prospective effects of Time 2 stressors on Time 3 symptoms, and we then extended that model to predict the development of drug dependence by young adulthood using the WLSMV estimator. We included Time

3 stressors in this final model, predicted by Time 2 stressors and symptoms. Although effects of symptoms on stressors would not be expected, as our measure was designed to assess life events that were uncontrollable from the perspective of the adolescence, we decided to explicitly test this hypothesis. This allowed us to rule out the possibility of reciprocal relations between stressors and symptoms and to assess the independent effects of Time 3 stressors on drug dependence. Although using such life events should also preclude third-variable explanations for the effects of stressful life events on externalizing symptoms and drug dependence, we explicitly tested this assumption by re-analyzing the current models to include a census-based measure of socioeconomic status, which has been connected to the experience of stressors and to psychopathology. Including socioeconomic status in the current models did not substantively change the main effect or mediational findings; thus we present the more parsimonious models here. Figure 1 provides an illustration of the final model.

Prediction of stressors and symptoms at Time 3

We first regressed Time 3 symptoms on Time 2 stressors and symptoms, and to rule out reciprocal effects, we regressed stressors at Time 3 on symptoms at Time 2. Stressors and symptoms within time points were allowed to correlate freely. Finally, because our covariates (age, gender, parental antisociality, and alcoholism) were all measured at Time 1, we regressed the Time 2 predictors on the covariates. Results suggested that this path model fit the data well ($\chi^2 = 16.24$,

FIGURE 1. A graphical model of the interrelations among stressors and symptoms across two time points (Time 2 and Time 3) during adolescence. All solid lines shown are significant; dotted lines were tested but found to be nonsignificant. Residual variances are indicated by e_x . Path coefficients and standard errors may be found in Table 5. Model fit: $\chi^2 = 16.24$, 15 df, $p = 0.18$, $n = 427$; root mean square error of approximation = 0.02; comparative fit index = 0.99; Tucker-Lewis index = 0.99. T = Time.

TABLE 5. Autoregressive path model with covariates of stressors and symptoms during adolescence

	Time 3 externalizing symptoms			Time 3 internalizing symptoms			Time 3 stressful life events		
	<i>b</i> (SE)	<i>z</i>	<i>B</i>	<i>b</i> (SE)	<i>z</i>	<i>B</i>	<i>b</i> (SE)	<i>z</i>	<i>B</i>
Time 2 externalizing symptoms	0.77 (0.04) [‡]	19.04	0.75	0.00 (0.10)	0.03	0.00	0.25 (0.23)	1.08	0.05
Time 2 internalizing symptoms	-0.06 (0.02) [‡]	-3.33	-0.13	0.52 (0.05) [‡]	11.56	0.53	0.18 (0.11)	1.66	0.08
Time 2 life events	0.02 (0.01) [†]	2.79	0.10	0.05 (0.02) [†]	2.67	0.12	0.47 (0.05) [‡]	10.55	0.48
Gender	0.04 (0.02)	1.82	0.06	-0.19 (0.06) [‡]	-3.37	-0.13	-0.35 (0.14) [*]	-2.55	-0.11
	Time 2 externalizing symptoms			Time 2 internalizing symptoms			Time 2 stressful life events		
	<i>b</i> (SE)	<i>z</i>	<i>B</i>	<i>b</i> (SE)	<i>z</i>	<i>B</i>	<i>b</i> (SE)	<i>z</i>	<i>B</i>
Time 1 age	0.04 (0.01) [‡]	3.43	0.16	0.09 (0.02) [‡]	3.71	0.17	0.12 (0.06) [*]	2.10	0.10
Gender	0.04 (0.03)	1.28	0.06	-0.23 (0.07) [‡]	-3.34	-0.16	-0.34 (0.16) [*]	-2.14	-0.10
Parental alcoholism	0.19 (0.03) [‡]	5.52	0.26	0.19 (0.07) [†]	2.58	0.13	0.55 (0.17) [‡]	3.35	0.16
Parental depression	-0.01 (0.07)	-0.07	0.00	0.19 (0.15)	1.25	0.06	0.49 (0.35)	1.40	0.07
Parental antisociality	0.17 (0.06) [†]	2.61	0.12	0.09 (0.14)	0.66	0.03	0.92 (0.32) [†]	2.92	0.14

*Coefficient is significant, $p < .05$; [†] $p < .01$; [‡] $p < .001$.

15 df, $p = .18$, $n = 427$; RMSEA = 0.02; CFI = 0.99; TLI = 0.99). Table 5 summarizes these results.

Time 2 internalizing symptoms predicted decreases in Time 3 externalizing symptoms from Time 2 levels ($B = -.13$, $p < .001$), but Time 2 externalizing symptoms were unrelated to Time 3 internalizing symptoms. Consistent with previous research, stressful life events at Time 2 predicted increases in both Time 3 externalizing ($B = .10$, $p < .01$) and Time 3 internalizing ($B = .12$, $p < .01$) symptoms, over and above Time 2 symptoms and the covariates. Being male predicted decreases in Time 3 internalizing symptoms and life events from Time 2 but was unrelated to externalizing symptoms at Time 3. Finally, Time 2 symptoms were unrelated to Time 3 life events, suggesting that the effects of stressors on symptoms are unidirectional.

Test of mediation

Next, we tested whether the effects of adolescent life events on the development of young adult drug dependence were mediated by externalizing and internalizing symptoms in a path modeling framework using WLSMV for categorical outcomes in Mplus 4.0. Model coefficients and standard errors can be found in Table 6. Because Mplus uses the probit function for categorical outcomes (rather than the logit

function), ORs are not readily computable from the model coefficients. Thus, to aid interpretation of the effects in the current models, we estimated a version of the final model in SPSS 15.0 to obtain OR estimates, predicting drug dependence from Time 3 stress and symptoms and the covariates.

Fit indices suggested that the model adequately reproduced the observed covariance matrix ($\chi^2 = 14.96$, 10 df, $p = 0.13$, $n = 427$; RMSEA = 0.03; CFI = 0.99; TLI = 0.98). Along with parental alcoholism (OR = 2.07), the direct effects of Time 3 externalizing symptoms on drug dependence were significant and positive ($B = 0.88$, $p < .001$; OR = 3.55). Moreover, in this final model, stressful life events at Time 2 or Time 3 were unrelated to drug dependence. However, the indirect effect of Time 2 stressful life events on drug dependence via Time 3 externalizing symptoms was significant ($a \times b = .019$, SE = .008, $p < .05$), accounting for roughly 20% of the total effect of life events on drug dependence. On the other hand, internalizing symptoms did not mediate the life events–drug dependence relation. Thus in this final model, the effects of stressful life events on drug dependence were not significant but were mediated via externalizing symptoms. Experiencing more stressful life events led to increases in externalizing symptoms by Time 3, which in turn raised the likelihood of drug dependence by young adulthood.

Because of the high rates of comorbidity with alcohol dependence (66%), it is possible that the effects of stressors and symptoms observed in the current models may not reflect the development of drug dependence but rather the development of co-occurring alcohol dependence. To test this possibility, we analyzed the current models considering alcohol dependence that did and did not co-occur with drug dependence. Both the main effects and the mediational findings were unchanged when predicting co-occurring drug and alcohol dependence. However, the effects of stressors and symptoms on alcohol dependence that occurred in the absence of drug dependence were weak, and the mediational effect was not found. This suggests that the stress-symptom pathway observed in the current model is not being driven

TABLE 6. Prediction of drug dependence from symptoms and life events

Symptom/life event	Drug dependence		
	<i>b</i> (SE)	<i>z</i>	OR
Parental alcoholism	0.36 (0.15) [*]	2.34	2.07
Parental depression	0.00 (0.29)	0.00	0.78
Parental antisociality	0.07 (0.25)	0.27	1.30
Time 1 age	-0.09 (0.05)	-1.89	0.82
Gender	0.19 (0.15)	1.30	1.59
Externalizing symptoms, Time 3	0.88 (0.20) [‡]	4.32	3.55
Internalizing symptoms, Time 3	0.18 (0.12)	1.43	1.46
Stressful life events, Time 3	0.01 (0.05)	0.23	1.01

Notes: OR = odds ratio.

*Coefficient is significant, $p < .05$; [‡] $p < .001$.

by co-occurring alcohol dependence but rather reflects the development of drug dependence.

Discussion

The goal of the current study was to test whether stressful life events experienced during adolescence increased internalizing and externalizing symptoms and whether these increases in symptoms mediated the effects of life events on the development of young adult drug dependence. As in the current study, previous research using data from the same prospective study demonstrated that young adult drug dependence was related to parental alcoholism, externalizing symptoms, and gender (e.g., Chassin et al., 1999, 2004; King and Chassin, 2004; Zhou et al., 2006). Moreover, our results support previous research that demonstrated that exposure to stressors during adolescence leads to increases in both emotional and behavioral problems (Kim et al., 2003; McMahon et al., 2003; Windle and Davies, 1999) and extended that research to show that those stress-related increases in behavior problems can exacerbate adolescents' risk for developing drug dependence. It is notable, too, that the life events considered in the current study were chosen to reflect events that were uncontrollable and external from the adolescent's perspective, making third-variable explanations less likely.

Previous research had suggested that exposure to stressors might elevate the risk for substance-use disorders (Clark et al., 1997, 2003; Jackson and Sher, 2003; Sher et al., 1997; Steinhausen and Metzke, 2003; Widom et al., 1995), but no studies that followed adolescents into young adulthood had explicitly tested this pathway. In the current study, exposure to life events during adolescence predicted risk for drug dependence 5-10 years later, controlling for age, gender, socioeconomic status, and psychopathology. Specifically, the experience of one additional stressor at a median age of 14 increased the odds of developing a drug-dependence disorder in young adulthood by 25%. This adds to previous findings from the same larger sample that indicate that characteristics of the adolescent family environment such as parenting and familial conflict influence the development of drug dependence (e.g., King and Chassin, 2004; Zhou et al., 2006) and suggests that risk for drug dependence is shaped in part through the adolescent's exposure to chaotic, conflictual, and ultimately stressful environments (i.e., Sher et al., 1997).

Many theories of the role of stressors in adolescent substance use hypothesize that adolescent substance use and disorder stem from attempts to cope with negative affect (Agnew, 1992; Sher, 1991; Wills and Filer, 1996); yet the current study suggests a different pathway. Previous research had shown that stressful life events lead to both internalizing and externalizing psychopathology (see review by McMahon et al., 2003), and multiple empirical studies have connected stressful life events to elevated behavioral problems in ado-

lescence (e.g., Kim et al., 2003; Windle and Davies, 1999). The current study confirms this prior research, demonstrating that the experience of stressful life events was related to higher levels of both internalizing and externalizing psychopathology in adolescence. Moreover, the current study is unique in that we extended those effects to show that it is stressors' effects on *externalizing* symptoms (not internalizing symptoms) that seems to connect it to the development of substance-use disorders. Although our analyses do not conclusively rule out specific aspects of internalizing symptoms, such as depression or anger (McCreary and Sadava, 1999; Tarter et al., 1995), they do rule *in* a novel pathway from stressful life events to substance use.

There are several ways that stressful life events may elevate levels of behavior problems and in turn lead to substance-use disorders. First, there are multiple theories that propose links from stressors to problem behaviors via stress' impact on negative affect. For example, Wills et al. (2001) propose that the experience of stressful life events leads to externalizing problems as adolescents attempt to cope using social strategies (e.g., "hangout coping"), which further embeds them in a deviant peer context that encourages externalizing behaviors and drug use. Alternately, the negative affect produced by exposure to stressful life events may be particularly connected to risky, antisocial behavior for adolescents, whose executive functioning or "cognitive control networks" are more strongly suppressed in the presence of peers or during periods of high emotional arousal (Steinberg, 2007). Thus the emotional arousal caused by exposure to stressors may lead more adolescents to engage in more risky behaviors as they choose highly rewarding short-term gains despite the potential long-term consequences. Finally, exposure to stressors may also impair self-regulation, reactivity, and self-control (Boyce and Ellis, 2005; Muraven and Baumeister, 2000), which are key precursors of antisocial behaviors and substance-use disorder (Sher and Trull, 1994). It could be that when adolescents are exposed to stressful life events, their ability to regulate their behavior, consciously or unconsciously, is diminished across all areas of life. Future research should specifically explore the mechanisms by which the experience of stressors in adolescence influences conduct and behavior problems.

Although the current study provides the first prospective test of externalizing symptoms as a mediator of the effects of life events on substance dependence using a high-risk sample, it also has limitations that should be acknowledged. First, the lag of effect between stressors and symptoms (e.g., 1 year) may be too long to observe more time-dependent relations between stressors and symptoms or, as noted above, may bias the findings in the direction of broader effects on self-regulation and externalizing problems and away from the potentially short-term impact that stressors may have on internalizing symptoms and negative affect. Moreover, although we used a well-established measure of stressful life

events, the inventory of stressful life events captured only 13 unique stressors. Future studies should examine a broader range of stressors to better understand the mechanisms of the relation between exposure to stressors and substance dependence. Moreover, although the current measure of stressors that were uncontrollable from the perspective of the adolescent should reduce the likelihood of third-variable explanations, it may be that unmeasured variables account for the covariation between stressors, symptoms, and drug dependence. Finally, the current study did not test the underlying processes by which life events could lead to externalizing psychopathology. Future studies should examine the role of stress-induced changes in self-regulation to determine the mechanisms of this effect.

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