Published in final edited form as:
J Abnorm Psychol. 2009 August ; 118(3): 610–622. doi:10.1037/a0016315.

Prospective relations between growth in drinking and familial stressors across adolescence

Kevin M. King,
Department of Psychology University of Washington

Brooke S.G. Molina, and
Departments of Psychiatry and Psychology University of Pittsburgh

Laurie Chassin
Department of Psychology Arizona State University

Abstract

Although there is much empirical support for the relation between stress and alcohol consumption in adolescence, it is unclear whether exposure to stressors is associated with overall trajectories or temporary elevations in drinking. Moreover, little research has explored whether the stress-alcohol use association in adolescence may be explained by shared risk factors that produce both individual differences in stress exposure and elevated risk for alcohol use. The current study tested these hypotheses within the context of a state-trait model of family stressors in a prospectively studied sample of children at high risk for alcoholism: children of alcoholic parents and matched controls (n = 451). Levels and growth in alcohol use were modeled longitudinally from ages 13 to 17. Results indicated that shared risk factors accounted for 53% of the impact of trait family stressors on growth in adolescent drinking, but time-specific exposure to familial stressors still predicted short-term boosts in alcohol use in adolescence. These findings imply that trait familial stressors mark adolescents at risk for alcohol use, and also impact adolescent alcohol use within a short time frame (i.e. over a year versus over many years) when they occur above and beyond the adolescent's "usual load" of stressors.

The experience of stress is commonly theorized to be a risk factor for adolescent alcohol use (Hawkins, Catalano & Miller, 1992). Cross-sectional and longitudinal studies have shown that adolescents who experience family stressors exhibit increased alcohol use (see reviews by Cerbone & Larison, 2000 and Grant et al., 2003). Although the majority of studies have explored the impact of stressors on the level of alcohol use, advances in growth curve modeling have produced research on the impact of family stressors on change in alcohol use across adolescence. For example, Wills, Sandy, Yaeger, Cleary and Shinar (2001) found that stressors were positively related to both initial levels of alcohol use at grade 7 and greater growth in alcohol use from grade 7 to grade 9. Similarly, Aseltine & Gore (2000) used HLM to show that during adolescence and young adulthood (aged 11-25) stressors in the past year accounted for time-specific increases in alcohol use that went above and beyond the effects of age related increases in drinking. Taken together, these studies suggest that between-individual differences

Correspondence concerning this article should be addressed to Kevin M. King, Department of Psychology, University of Washington, Box 351525, Seattle WA 98195-1525, kingkm@u.washington.edu. .

in exposure to stressors may influence how drinking changes in general across adolescence, and within-individual, or time-specific, differences in stress exposure from one time to the next might also explain deviations from individuals' average trajectories. However, no studies have yet tested both hypotheses in the same model.

Hussong, Curran, Moffitt, Caspi & Carrig (2004) described two putative processes by which risk factors, measured repeatedly over time, might influence a developmental process such as changes in drinking across adolescence at *both* the between and within-individual levels. One is dubbed the *launch* hypothesis, in which a factor at the initial time point influences an individual's general trajectory of behavior change over time. For the relation between stress and drinking, a launch hypothesis would be supported if stressors predicted between-individual differences in the rates of increase in adolescent drinking over time. The second hypothesis tests whether time-specific factors predict within-individual deviations, or deflections, from an individual's expected rate of change over time. Dubbed the *snares* hypothesis (Hussong et al., 2004), for the current study we reverse the direction of effect and label it the *boost* hypothesis. This hypothesis suggests that during times that stress is elevated above an individual's average levels, an increase in his/her alcohol use will occur.

Testing the Developmental Associations between Stressors and Alcohol Use

Many studies, including much previous research on family stressors and alcohol use (e.g.; Wills et al., 2001), treat stressful life events as a measure that is expected to vary randomly across individuals. Thus, most studies that explore the effects of stressors at multiple time points have treated stressors as time-varying (i.e. random) covariates (Aseltine & Gore, 2000; Cole, Nolen-Hoeksema, Girgus & Paul, 2006; Kim et al., 2003). Indeed, checklist measures of stressors are often deliberately constructed to *avoid* including events that might be related to individual differences. The stressors included on such checklists are often referred to as being "external and uncontrollable" from the perspective of the individual, theoretically ensuring that exposure is random (Grant, Compas, Thurm & McMahon, 2004). However, children and adolescents are embedded within environments such as neighborhoods and families that could produce non-random, stable levels of stress exposure over time, even though those stressors are uncontrollable from the perspective of the adolescent. For example, an adolescent whose parents divorce may be exposed to high levels of stress over many years because of pre-divorce marital conflict, and the adolescent may continue to experience high levels of stress after the divorce because new post-divorce stressors emerge. Even within this long-term pattern of high stress, however, the levels of stress may peak around the time of the divorce. As illustrated in Figure 1, this adolescent may experience few significant events in the first year, a major stressor such as parental divorce in the second (with many associated stressors) and somewhat fewer stressors in the third year (e.g. post-divorce inter-parental conflict). In all, this adolescent may have experienced more stressors in general than do other adolescents because of his/her family environment, but the adolescent also had times that were better or worse than that average. Thus, it may be that *both* the average level of stress exposure *and* time points when stress exceeds that average level are important in understanding how adolescent stressors impact psychopathology.

Diary studies, which ask whether individuals drink in response to daily stressors, have suggested that time-specific variation in hassles and minor stressors at the day-to-day level may influence alcohol use among young adults, particularly for those with high positive expectancies for alcohol use (Armeli et al., 2000; Armeli, Todd & Mohr, 2005; Hussong, Hicks, Levy & Curran, 2001). However, research has shown that stress also impacts important developmental processes on a time-scale larger than the daily level. That is, there are cascading effects of stress exposure after acute responses to stress. Some of the downstream effects of stress may only appear after a certain period of stress exposure, and these effects may have

important consequences for development. For example, recent research suggests that exposure to stress during adolescence may affect the development of the prefrontal cortex, which is associated with emerging self control (Anderson & Teicher, 2008). Thus, it follows that stress may not only impact day-to-day behaviors, but it may also affect the broader processes of adolescent development, which are likely to be detectable only with a longer time frame such as the annual assessments used in the current study.

State-trait models (Schmitt & Steyer, 1993; Jackson, Sher & Wood, 2000; Jackson & Sher, 2003) can be used to model processes at multiple time scales. Applied to stressors, these methods model the number of family stressors experienced by an individual at a time point as resulting from both a latent "tendency" to experience family stressors (the trait factor, based on the average number of family stressors an individual experiences), and time-specific variation from that average (the state factors). Two studies (including one of our own) have previously supported this approach to modeling environmental and interpersonal stressors in adults (Foley, Neale & Kendler, 1996) and largely familial stressors in adolescence (King, Molina & Chassin, 2008). Indeed, our previous study showed that on average, a trait life events factor explained 46% of the variance in the occurrence of life events at any particular age (King, Molina & Chassin, 2008). This state-trait approach to stressors provides an opportunity to simultaneously test the launch and the boost hypotheses.

We drew on recent advances in the integration of latent growth and auto-regressive models (e.g. Bollen & Curran, 2004) to simultaneously model the putative effects of trait familial stressors on growth in drinking and of time-specific variation on short-term increases in drinking independent of growth. Figure 2 illustrates the parallel models of state-trait stressors and growth in drinking. Without considering these different types of variation in stressors, it is difficult to determine whether the effects observed in previous studies (e.g. Wills et al., 2001; Aseltine & Gore, 2000) resulted from stable individual differences in stress exposure (possibly reflecting the effects of chronic stress exposure) or from time-specific elevations in family stressors (reflecting the acute effects of adapting to stressors).

Although the vast majority of studies have utilized life events checklists that do not classify life events by type and which include a broad range of potential stressors (Grant et al., 2003), the state-trait hypothesis applies more readily to certain types of events (e.g. those resulting from environmental disruption) than to others (e.g., isolated events such as death due to illness or accident). Moreover, previous research has suggested that family-specific stressors may have particular importance in the development of substance use, whereas stressors associated with major illness or bereavement were less strongly related to adolescent substance use (Pillow, Barrera & Chassin, 1998). Thus, for the current study, we used a subset of items that largely captured stressors that occurred in the family, which provides a theoretically meaningful interpretation of the trait factor as a reflection of the average level of uncontrollable stressors produced by the adolescent's family.

Stress as a marker or a mediator of risk for adolescent drinking

In addition to differing conceptualizations of the temporal influences of stress on adolescent drinking, it is also possible that the observed effects of stressors on alcohol use (and other outcomes) are due, at least in part, to the shared risk factors that produce both stable exposure to stressors and that also launch adolescents onto higher trajectories of drinking. That is, experiencing many stressors during adolescence may simply be a marker of risk for drinking. Although multiple theories have proposed that stress leads to increases in alcohol use as a maladaptive coping strategy (e.g. Cloninger, 1987; Wills & Filer, 1996; Sher, 1991; Agnew, 1992; Sinha, 2001), this hypothesis has received mixed support for adolescents (see Colder, Chassin, Lee & Villalta, *in press*, for a review). Thus, it is reasonable to ask whether stressors are really a causal risk factor for alcohol use in this age range. Indeed, Jackson and colleagues

(Jackson, Sher & Wood, 2000; Jackson & Sher, 2003) writing about state-trait models suggest that trait-level associations between constructs often suggest third variable explanations.

On the other hand, it is also possible that exposure to higher stable levels of family stressors is one of the mechanisms by which these shared risk factors launch adolescents onto increasing trajectories of substance use. That is, perhaps high-risk environments lead to escalations in drinking in part because they consistently expose some adolescents to higher levels of stressors, and it is the chronic stress exposure that explains their influence on alcohol use. For example, a previous study of a subsample of participants from the current study showed that the effects of familial alcoholism on substance dependence were partially mediated by exposing the adolescent to higher levels of family conflict (Zhou, King & Chassin, 2006).

To test whether stress is a marker or a mediator of shared risk factors for adolescent drinking, we chose as shared risk factors key familial and personality variables that have been linked to both alcohol use and to the types of stressors assessed in the current study (King et al., 2008). Although it would be impossible to assess *all* potential confounders of the stress-alcohol use relation, we attempted to sample some of the most important candidate variables. For example, parental psychopathology, such as parental alcoholism and antisociality, can produce disruption in the family environment (Zhou, King, & Chassin, 2006) that increases exposure to negative life events (Chassin et al., 1996; Sher, Gershuny, Peterson & Raskin, 1997; Langrock, Compas, Keller, Merchant, & Copeland, 2002; Phillips, Erkanli, Keeler, Costello, & Angold, 2006), and has also been repeatedly linked to adolescent alcohol use (Sher, 1991; Chassin, Ritter, Trim & King, 2003). Moreover, parenting factors including poor emotional support have been linked to both the experience of negative life events (Bergeman et al. 1990; Champion, 1990) and adolescent alcohol use (Hawkins et al., 1992; Baumrind, 1991). Adolescent impulsivity has been implicated as an important factor in the development of alcohol use and alcohol use disorders (see Tarter et al., 2003; Wills & Dishion, 2004; see reviews by Chassin et al., 2003 and Hawkins et al., 1992). At the same time, individuals who report more stressors are also more impulsive (Fulker, Eysenck & Zuckerman, 1980; Wills, Sandy & Yaeger, 2000). Although many previous studies have shown a link between shared risk factors and both drinking and stress exposure, no studies have considered this marker/mediator distinction, nor have any previous studies that we are aware of considered a trait-like model of the occurrence of stressors and its implications for the downstream effects of stress exposure. Thus, the final goal of the current study was to test whether trait family stressors across adolescence are a marker of risk for escalations in alcohol use, or a mediator of the effects of shared risk factors.

In short, the current study contributes to the literature on stress and adolescent drinking by testing four important questions: a) whether between-individual differences in trait family stressors launched adolescents onto escalating trajectories of alcohol use, c) whether time-specific variation in exposure to family stressors produced boosts in adolescent alcohol use over and above trajectory-related increases c) whether the influence of exposure to family stressors on growth in alcohol use was accounted for by shared risk factors (that raise risk for both family stress and alcohol use), and d) whether stable exposure to family stressors mediated the effects of shared risk factors on growth in alcohol use.

Methods

Participants

Participants were from an ongoing longitudinal study of familial alcoholism (Chassin, Rogosch & Barrera, 1991; Chassin et al., 1996; 1999; 2004). At Time 1, there were 454 adolescents (*M* age = 13.22 years, range = 10.5-15.5), 246 of whom had at least one biological, custodial

alcoholic parent (COAs) and 208 demographically matched adolescents with no biological or custodial alcoholic parents (controls).

COA families were recruited using court records of DUI arrests, health maintenance organization wellness questionnaires, and community telephone screening. Direct interview data from the computerized Diagnostic Interview Schedule (DIS version III; Robins, Helzer, Croughan, & Ratcliff, 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-III (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 one year), and SES (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; Chassin, Barrera, Bech, Kossak-Fuller, 1992). There were no significant differences between individuals who did and did not participate with respect to alcoholism indicators available in archival records (e.g., blood alcohol levels recorded at the time of 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 & 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 Mdn_{age} = 13, range = 11-16) of the adolescent participants and their parents. Sample retention was excellent across all three waves (n = 444 (98%) had complete data) and unbiased by gender and ethnicity, but more COAs than controls were lost at Time 2 or 3, χ^2 (1, N = 454) = 8.65, $p < .01$.

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 one to three hours and participants were paid up to $65 over the waves.

Selection of the Current Sample

We modeled alcohol use and stressors by age rather than by wave of measurement in order to identify age related trends and to provide a more readily interpretable scale of time (Singer & Willet, 2003). Data from the three waves of measurement were reorganized into six age cohorts, with data from age 11 to age 17, based on the participant's age at first interview. Thus, each participant contributed data to three ages and was coded as missing for the remaining ages. This approach mirrors a cohort sequential design and allows age to be explicitly modeled in the analyses rather than included as a covariate. Sample sizes at each age and by cohort are listed in Table 1. Very few participants reported any drinking in the past year at ages 11 (3%) or 12 (10%); modeling alcohol use trajectories including those ages produced ill-fitting models, with particular problems estimating the variance of the intercept because the intercept was estimated to be close to zero with non-significant variance. At the same time, very few

participants were age 18 at the final interview (n = 22, 4.8%). Thus, we excluded data at ages 11, 12 and 18 from the current analyses, and participants were included if they had any data from age from 13 to 17 (n = 451, 99% of the total sample)[1].

Of the included participants, 47% were female, 54% were COAs, and 9% had a parent diagnosed with Antisocial Personality Disorder. The ethnic composition of the included sample was 72% non-Hispanic Caucasian and 26% Hispanic, with the remaining 2% representing other ethnicities. At the initial time point, participants were on average 13.2 years old (range 11-15), and 72% of their parents had achieved some post-high school education.

Measures

Stress and Drinking—Table 2 provides descriptive statistics for family stressors and alcohol use across each age.

Family Stressors: The adolescent's experience of family stressors in the past year was assessed with 18 self report items adapted from the General Life Events Schedule for Children (GLESC, Sandler, Ramirez & Reynolds, 1986). As noted earlier, we dropped five items relating to illness and bereavement[2] to facilitate interpretation of the latent factors, leaving 13 life event items assessed at each time point that predominantly assessed family stressors that reflect family functioning. Table 3 provides a list of included items. These items were judged by expert raters to be both negative and uncontrollable from the perspective of the adolescent (Sandler et al., 1986), although clearly many of these family stressors are not uncontrollable from the perspective of others, such as the adolescent's parents. This measure has been well established in the literature (Grant et al., 2004), and had been previously shown to predict increases in adolescent alcohol 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. Across each age, adolescents reported an average of two family stressors in the past year, although the range was 0 to nine. The number of family stressors reported at each time point was significantly inter-correlated (r = .43− .64, p<.001 across waves), suggesting chronicity of the average number of family stressors over time[3].

Alcohol Consumption: Adolescents self reported their frequency and quantity of consumption of alcohol in the past year with four items at each wave. Frequency was assessed with two items (one for beer/wine and one for hard liquor) with responses ranging from "Never" to "Every day" that were coded into two alcohol frequency variables representing "drinking occasions per month" in the past year for beer/wine and hard liquor. Responses ranged from 0 (no drinks in the past year) to 30 (Daily or near daily drinking). Quantity was assessed with two items (one for beer/wine, one for hard liquor) assessing how much the adolescent drank in the past year on a "typical" occasion, ranging from (1) one to (9) nine or more drinks per occasion. Because a simple multiplication of quantity*frequency produces a variable with a metric that is difficult to interpret, we transformed participants' report of past year drinking by

[1]The three excluded participants were not different from included participants in terms of any demographic variables or the covariates.
[2]It is possible that these bereavement/illness related life events also can be modeled with a state-trait approach, and perhaps should have been considered separately in the current analyses. We tested this hypothesis, but although model fit was excellent, χ^2 (5, n = 451) = 4.34, p = .50; CFI = 1.00; TLI = .99; RMSEA = .00, the latent life events factor had a variance that was not significantly different from zero (σ^2= .09, SE = .07, p = ns), suggesting that there were no between individual differences over time in exposure to bereavement/illness events; thus, a state-trait model fits better with some kinds of life events than others.
[3]This chronicity did not seem to stem from the same events being endorsed repeatedly over time. Only 3 events were endorsed by more than 5% of the sample at every wave of measurement (Friend moved, 6.8%, Friend had serious trouble, 9.8%, and Parent Money Problems, 18%), and other than those three family stressors, no event was endorsed by more than 10% of participants as having occurred twice over the three waves of measurement. This suggests that most of the observed covariation in family stressors over time is due to individual's rank order of stability in the level of event occurrence rather than the repeated appearance of the same family stressors over time.

multiplying the alcohol frequency variable by the alcohol quantity variables for beer/wine and for hard liquor, and summed the two products to obtain a measure that roughly represents the participants' estimate of drinks of alcohol per month in the past year. Because this variable could range from zero to 300, it was highly skewed and kurtotic across all ages, and we used a log transformation to reduce skewness and kurtosis. In general, as the adolescents aged they reported drinking with greater frequency (from around 2 drinks in the past year at age 13 to over 11 drinks per month at age 17); some adolescents (n = 209, 46.3% of the current sample) never reported drinking at any of the three interviews.[4]

Shared Risk Factors

Parental psychopathology: At Time 1, lifetime DSM-III diagnoses of parental alcoholism (abuse or dependence) and antisociality were assessed with the DIS (Robins et al., 1981). For the present analyses, diagnoses were dichotomous: either present (at least one biological and custodial parent met lifetime criteria) or absent (neither biological parent met lifetime criteria). For non-interviewed parents (for 19.3% of fathers, 7.7% of mothers), lifetime alcoholism diagnoses were established using the Family-History Research Diagnostic Criteria (FH-RDC, Version 3; Endicott, Andreason, & Spitzer, 1975) based on spousal report, and non-interviewed parents were considered to non-antisocial.

Parental support: Adolescents' perceptions of parental support were measured at Time 1 using items from the Network of Relationships Inventory (7 items each for mother's and father's support; Furman & Buhrmester, 1985). Sample items from this scale include "How much does (your mother/father) treat you like you are admired and respected?" and "How much can you count on (your mother/father) to be there when you need (him/her) no matter what?" Adolescent reports of maternal and paternal support were correlated at .61. The scale score was the mean of the adolescent's report of paternal and maternal support (14 items total). If an adolescent reported on only one parent, the mean of those seven items was used (Cronbach's $\alpha = 0.91$).

Adolescent impulsivity: Adolescents' impulsivity (12 items) was measured by Time 1 maternal and paternal report using items from the EASI (Buss & Plomin, 1984). The Impulsivity dimension of the EASI measures four distinct dimensions of impulsiveness (decision time, persistence, sensation seeking and inhibition). Recent research has highlighted the importance of considering the multidimensional nature of impulsive-like traits (see Smith et al., 2007 for a review). However, internal consistency across subscales was very poor ($\alpha = .32 - .71$; only persistence had $\alpha > .60$), and attempts to develop latent factor models of the sub-scales of impulsivity failed due to extremely poor model fit. Thus we decided to collapse across the four subscales and create a single measure of impulsivity. Internal consistency (Cronbach's α) was acceptable for both paternal ($\alpha = .73$) and maternal ($\alpha = .75$) reports. To allow inclusion of single parent families and because maternal and paternal reports of impulsivity were moderately correlated ($r = .48$), we computed impulsivity as the mean of the two parent reporters.

Results

The current study had two major goals. First, we tested how family stressors and alcohol use co-vary across adolescence. This model simultaneously tested the launch hypothesis, whether

[4]Including complete abstainers in models of alcohol use may cause problems in model estimation and interpretation. At the same time, abstainers are a theoretically meaningful group to include in analyses. To test whether including non-drinkers in the current models impacted our findings, we re-analyzed the data excluding any participants who reported no drinking at any time point during the adolescent waves. Our results were substantively identical to those obtained utilizing the total sample; thus, we present the models that used the entire sample above.

the average number of family stressors was related to growth in alcohol use, and the boost hypothesis, whether the number of family stressors at any one time point was associated with drinking at that time point. Second, we tested the marker/mediator hypotheses: testing whether the effects of family stressors on drinking were attenuated with the addition of shared risk factors (parental psychopathology, parental emotional support and adolescent impulsivity) and/or whether family stressors mediated the effects of these shared risk factors on adolescent drinking.

We accounted for missing data by using maximum-likelihood model estimation assuming ignorable missingness at random (Little & Rubin, 1987; L. K. Muthen & Muthen, 1998, pp. 363-364). To address skewness in the data, we used the Maximum Likelihood Estimator with Robust Standard Errors (MLR). Descriptive data analyses were performed using SPSS 15.0, and the hypotheses were tested using MPlus 5.0 (Muthen & Muthen, 2007). To test whether varying permutations of each model improved model fit, we used Satorra's chi-square difference tests for MLR chi-square (Satorra, 2000). Model fit was assessed using Chi-Square as an indicator of exact fit. Where exact fit was not achieved (as chi-square is sensitive to violations of normality and sample size, Hu & Bentler, 1999), we used several relative fit indices that are transformations of chi-square designed to avoid some of the problems with using chi-square. Specifically, we used the Tucker-Lewis Index (TLI), comparative fit index (CFI) and root-mean square error of approximation (RMSEA) to supplement Chi-Square based on the guidelines provided by Hu and Bentler (1999) and the cautions of Marsh, Hau & Wen (2004). Finally, to test for mediation, we utilized the product of coefficients method, which has been shown to be the most powerful test of mediation (MacKinnon, Lockwood, Hoffman, West & Sheets, 2004), and tested for significance of these indirect effects using asymmetric confidence intervals computed with the program PRODCLIN, described by MacKinnon, Fritz, Williams & Lockwood (2007). Finally, because of the skewness in the alcohol data, we examined casewise residuals and measures of influence, including Cook's D and Mahalanobis Distance to determine whether any cases exerted undue influence on the results.

Unconditional Models

Family Stressors—First we developed a state-trait model of family stressors across ages 13 to 17, as described in more detail in our previous study with the same sample (King, Molina & Chassin, 2008). In brief, a latent stress factor (the trait factor) was modeled by fixing the factor loading of family stressors at each age to one, with the variance of the latent factor freely estimated. This approach is similar to estimating an intercept-only latent growth curve model, except that the latent mean is not directly estimated. Results were nearly identical to our previous study that explored this model from ages 11 to 17 (King et al., 2008). Fit statistics suggested that the model adequately reproduced the observed covariance matrix, χ^2 (5, n = 451) = 5.34, p = .38; CFI = 0.99; TLI = 0.99; RMSEA = .01. The latent trait factor had statistically significant variability (σ^2= .95, SE = .30, p < .01), there was significant residual (time specific) variance in the occurrence of family stressors at each age, and the amount of variation differed across ages, $\Delta\chi^2$ (1.34) = 3.30, p < .01.

Finally, we estimated autoregressive effects between the residuals at adjacent ages, which tested whether the deviation score (or trait variation) of family stressors reported at one age predicted the deviation score of family stressors reported at the next, and reflected trends in family stressors at a smaller time scale then what is represented by the latent factor. The model-estimated autoregressive effects of family stressors on residual variance at adjacent ages were positive (β = .22, SE = .10, p< .05), indicating that experiencing more family stressors at one age predicted experiencing more family stressors at the next age, over and above the latent family stressors factor. Finally, these effects were equal across ages, $\Delta\chi^2$ (1.53) = 1.92, p = ns.

Latent growth curve model of alcohol use—We next tested the unconditional growth model of alcohol use (i.e., the latent growth curve model of alcohol use with no exogenous predictors). The latent status, or intercept, factor was set to age 13, reflecting inter-individual variation in mean levels of alcohol use at age 13. The latent slope factor was estimated to be linear, reflecting the average increase in alcohol use and inter-individual variation from that average from age 13 to 17[5].

The growth model of alcohol use fit the data well [χ^2 (7, n = 451) = 5.68, p = .58; CFI = 1.00, TLI =1.00, RMSEA = .00].The mean of the latent status (intercept) factor was significantly different from zero (μ = 0.07, σ = 0.02, p < .001), but it did not vary significantly across participants (ψ = 0.03, σ = 0.08, p = ns), reflecting the non-zero but very low level of alcohol use in the sample at age 13 (fewer than 20% of participants reported any past year alcohol use). On average, participants increased their alcohol across the five ages assessed (μ = 0.21, σ = 0.02, p < .001) and also varied significantly in how quickly they increased their alcohol use (ψ = 0.08, σ = 0.04, p < .05). The slope and status factors were uncorrelated (r = .03, p = ns), suggesting that rate at which participants' alcohol use increased from age 13 to 17 was unrelated to their level of drinking at age 13 (given the low variation in alcohol use at age 13 this is unsurprising). At age 13, participants were drinking on average 0.07 drinks per month (or 1 drink per year), and increased their drinking at a rate of 0.21 drinks per month (or 2.5 drinks per year), but this rate of increase significantly varied across participants.

The Relation Between Family Stressors and Alcohol Use—We next estimated within a single model state-trait variation in family stressors and latent growth in alcohol use across adolescence. This model fit the data well, χ^2 (29, n = 451) = 23.13, p = .78; CFI = 1.00; TLI = 1.00; RMSEA = .00. Time-specific variation in family stressors was treated as a time-varying covariate of alcohol use, and residual variance in alcohol use at each age was regressed on time-specific family stressors at the same age, representing how the number of family stressors that an individual experienced in a year (above and beyond their own average) affected how much they deviated from their own individual trajectory of drinking in that year. We tested these effects concurrently rather than with a one-year lag because the time-specific variation in stressors reflected deviation from the individual's "usual load" in a given year, and thus should have effects on alcohol use that are close in time to when that deviation is experienced[6].

The trait family stressors factor positively predicted the slope of drinking (b = .12, SE = .04, p < .01), suggesting that the tendency to experience more family stressors over time was associated with escalations in drinking across adolescence. Because the variance of the intercept was estimated to be not significantly different from zero, we did not estimate a covariance between the trait factor and the intercept of alcohol use. Chi-square tests indicated that freely estimating the time-specific effects of family stressors on drinking provided no significant improvement in model fit, $\Delta\chi^2$ (1.67) = 1.80, p = ns, and that fixing those time-specific effects to zero significantly degraded model fit, $\Delta\chi^2$ (.29) = 40.61, p < .001; thus, they

[5]We also tested whether other trajectory shapes might provide a better fit to the current data, including free-time scores and quadratic trajectory shapes. Chi-square difference testing suggested that a free-time score approach, freely estimating the slope loadings at ages 14, 15 and 16 and fixing the slope at age 17 to one, did not significantly improve model fit, $\chi^2\Delta$ (3, 451) = 0.63, p = ns. Moreover, modeling a quadratic latent slope factor produced no significant improvements in model fit, $\chi^2\Delta$ (5, 451) = 0.89, p = ns, and the estimated quadratic factor had non-significant variance and a mean of zero. This suggests that a latent linear slope factor provides the best estimate of changes in alcohol use over time in the current sample.

[6]Because the state variance in life events represents annual deviations from an individual's average, we hypothesize that its effects should be observed relatively close in time to that annual deviation, and that the state variance would have few prospective carryover effects. We tested this hypothesis by re-analyzing the current model, but including time varying effects of state-stressors predicting residual alcohol use one year later. Results indicated that whether we controlled for concurrent state stressors or not, residualized alcohol use in any specific year was unrelated to state-stressors in that previous year. This confirms the hypothesis that state stressors have contemporaneously rather than prospective effects on residualized alcohol use.

were estimated and fixed to be equal across all time points. The time-specific associations between family stressors and drinking were significant and positive (b = .053, SE = .016, p < .01). Experiencing more family stressors within the year (regardless of age), over and above one's tendency over time to experience family stressors, was associated with increases in alcohol use over and above the adolescent's trajectory of drinking.

Conditional Model: Considering shared risk factors for stress and alcohol use —To examine stress both as a marker and a mediator of risk for drinking, we estimated the same model and included gender, parental alcoholism and antisociality, social support, and adolescent impulsivity as shared risk factors predicting the slope and intercept of alcohol use and the latent trait factor for family stressors. We tested whether their effects on the slope of alcohol use significantly attenuated the effect of trait family stressors on slope, and whether trait stressors mediated the effects of those shared risk factors on growth in alcohol use. Examination of outliers in this model suggested that two heavy drinkers contributed strongly to the results; their deletion reduced the effect of the trait stress factor from b = .07, SE = .03, p = .02 to b = .05, SE = .03, p = .06. Removal of these two cases (or any other potentially influential cases) did not change the magnitude or significance of any other effects or parameter estimates in the model (or in the models presented above), including the effects of the shared risk factors on the trait and latent growth factors, or the time-specific association between stress and alcohol use. Thus, we report the results of this more conservative model below with the two outliers removed.

This model fit the data moderately well, χ^2 (63, n = 449) = 75.63, p = .13; CFI = .97; TLI = .97; RMSEA = .02. Importantly, the time-specific effects of family stressors on drinking remained effectively unchanged after including parental and adolescent risk factors (b = .059, SE = .018, p < .01). Figure 3 provides an illustration of this model, and Table 4 provides path estimates and standard errors.

Marker Hypothesis: When shared risk factors were accounted for, the relation between the trait family stress factor and slope of drinking was attenuated after inclusion of the shared risk factors (but still approached significance (b‘ = 0.05, SE = .03, p = .06). To test whether this attenuation was statistically significant, we drew on methods that are typically used for testing mediation (MacKinnon, Krull & Lockwood, 2000). Whereas mediational tests often refer to the degree to which the effect of the predictor on the outcome is attenuated (often notated as c and c‘), we were interested in the degree to which the effect of a putative mediator (trait family stressors) on alcohol use was attenuated with the addition of a set of shared risk factors. We tested the significance of the difference in coefficients (b-b‘) using the standard error for the difference in coefficients developed by Freedman, Graubard and Schatzkin (1992). This test confirmed that the reduction in magnitude was significant (b-b‘ = 0.06, SE = 0.008, p < .001), suggesting that a significant portion of the observed covariation between the tendency to experience family stressors and to accelerate in drinking over time was due to the influence of shared risk factors. Indeed, if we calculate the “proportion confounded” using a similar method to the commonly computed proportion mediated (i.e. MacKinnon, Yoon, Ryu & Fairchild, *under review*), we see that $\left(b - b'\right)/b = .55$, indicating that 55% of the effect of stressors on growth in alcohol use can be accounted for by shared risk factors.

Mediational Tests: However, the addition of shared factors did not completely attenuate the effects of stressors. As noted above, the effect of trait stressors on growth in alcohol use approached significance (p = .06) when shared risk factors were accounted for. This suggested that adolescents who tended to experience higher levels of family stressors across adolescence may have been launched onto increasing trajectories of drinking across adolescence over and above the effects of background risk variables. We thus tested whether this reflected a

mediational process by computing the indirect effect for the effect of each risk factor on the slope of alcohol use via trait stressors. We tested for the significance of that indirect effect by examining the asymmetric confidence intervals produced by the program PRODCLIN (MacKinnon et al., 2007). Results, presented in Table 5, did not provide robust support for a mediated effect. Trait family stress did not significantly mediate the effects of parental antisociality and adolescent impulsivity on growth in alcohol use. However, the mediated effects of parental alcoholism and social support on growth in alcohol use via trait family stress approached significance ($p<.10$).

Discussion

The current findings validate our previous study (King et al., 2008) by connecting stressful life events at both the trait and the state, or time-specific levels to an important outcome variable, namely, adolescent alcohol use. Moreover, the current study extends previous research by providing a more refined and conservative test of the association between the occurrence of stress and stress-alcohol use association across multiple levels of time. We modeled the associations between growth in alcohol use over time and family stressors separately for the latent tendency to experience negative events over time as well as for time- (i.e., age-) specific experiences of family stressors. We also examined whether these associations reflected confounding effects of shared risk factors or a mediational process. Our findings are consistent with the notion that family stressors affect adolescent alcohol use predominantly by producing time-specific boosts in drinking. Moreover, we found support for the marker hypothesis, in that the effects of family stressors on growth in drinking were partially accounted for by shared risk factors. In addition, our data support the hypothesis that some of the shared risk factors (parental alcoholism, male gender and poor parental support) seem to act as launching factors, setting adolescents onto increasing trajectories of alcohol use. However, there was little support for the hypotheses that trait family stress acts as a launch factor, or that trait stressors mediate the effects of distal shared risk factors on alcohol use. These findings highlight the utility of a state-trait conceptualization of the occurrence of stressors, and they demonstrate how the experience of negative family stressors in adolescence may be related to alcohol use at multiple levels of time.

The current findings were consistent with the boost hypothesis: time-specific elevations in stressors had a direct effect on adolescent alcohol use. Similar to Aseltine & Gore (2000), time-specific variation in negative family stressors explained intra-individual variation in drinking beyond the adolescent's average trajectory of drinking. Specifically, when an adolescent experienced family stressors in a year that exceeded their "usual load," short-term escalations in drinking during that year were more likely, suggesting that exposure to stressors during that year "boosted" alcohol use. To some degree, these findings address the time scale of the effects of stressors. The effects of stressors on observed problem behaviors (such as drinking in adolescence) seemed to be more time-specific than general, affecting deviations from trajectories of drinking more than accelerations in drinking across adolescence.

These time-specific effects seem to reflect an immediate response to the stressors over and above the adolescent's "usual load." However, the mechanism of this effect remains unclear, and indeed this effect could reflect multiple mechanisms. Exposure to stressors in the short term could lead to increases in drinking via affective dysregulation and irritability, gravitating towards deviant peers for support (e.g. Chassin et al., 1996; Wills et al., 2001), or by increasing drinking for those adolescents who perceive stress-reducing benefits (Windle & Windle, 1996). Moreover, these time-specific effects could be even stronger for more experienced adolescent drinkers, such as for those who present for treatment, due to their established experience with the perceived beneficial and stress-dampening effects of alcohol. It is unlikely

that the time-specific relations are due to reverse directions of effect (i.e., effects of adolescent drinking on stress) because of the uncontrollable nature of the stress items[7].

On the other hand, the launch hypothesis was not supported. Although previous research (e.g. Chassin et al., 1996; Wills et al., 2001) had suggested that stressful life events launch adolescents on higher trajectories of growth in drinking, in the current study the effect of trait family stressors on growth in alcohol use was significantly attenuated by the addition of shared risk factors to the model, reducing its effect to marginal significance (i.e. $p < .10$). This suggests that a high level of stress-exposure across adolescence marks adolescents who are at high risk for escalations in alcohol use. In other words, teenagers who tend to be exposed to more family stressors also increase the quantity and frequency of their alcohol use more quickly over time largely because shared risk factors are associated with both stress exposure and changes in drinking over time. This suggests that considering *who* is exposed to stressors is as important as understanding the effects of being exposed to stress, and that not all of the observed effects of stressors are due solely to stress processes.

The fact that trait stressors act as a marker of risk for greater growth in adolescent drinking likely reflects several sources of influence. First, there may be a common genetic vulnerability (shared by parents and offspring) that leads to both alcohol use (e.g. Krueger et al., 2002) and a more chaotic family environment that exposes adolescents to higher numbers of negative family stressors. In addition, it could be that this covariation reflects interactive processes that occurred earlier in childhood that shaped both the family dynamic and an adolescent's trajectory of problems that in turn lead to drinking. Finally, it could reflect an interactive process between family stressors and the parenting environment, where the experience of stressors such as those assessed by the current study (e.g. parental financial and social problems, etc.) impairs parenting such as monitoring or consistency of discipline, in turn leading to offspring alcohol use (e.g. Steinberg, Fletcher & Darling, 1994; Chassin et al., 1993).

Finally, our results confirmed the findings of other studies, suggesting that family variables including familial alcoholism, gender and poor parental support launch adolescents onto higher trajectories of alcohol use over time (Chassin et al., 2003). On the other hand, we did not find support for a mediating role of trait family stressors. In the current study the effect of trait stressors on growth in alcohol use approached but did not reach significance when the effects of shared risk factors on growth in alcohol use were taken into account. Given that the effects approached significance, we cannot conclusively reject a mediating role of family stressors. However, additional research is needed to more conclusively test these effects.

Limitations of the current study

Although the current study has many strengths, including its prospective design, its use of a high risk community sample, and its utilization of advanced latent-variable modeling techniques, it has some limitations that should be noted. First, our measures of alcohol use and life events were relatively coarse, in that they required the adolescent to retrospect about their experiences in the past year, and we are thus unable to know the degree to which exposure to life events preceded changes in alcohol use or vice-versa. Future studies with shorter time frames and/or more fine-grained assessment techniques such as time-line follow-back procedures will be better able to ascertain the temporal precedence of acute escalations in life events and alcohol use, and to determine the length of the effect of the "boost" on alcohol use. Second, the putative effect of trait stressors on growth in alcohol use only approached significance (and thus so did the mediational effects); future studies should attempt to replicate

[7]We also tested whether modeling the relations between family stressors and adolescent drinking as a correlation would provide different results. Results indicated that this more conservative model did not differ substantively from those presented in the current study.

these effects to attempt to more conclusively test the mediational hypothesis. Moreover, the current study utilized a count measure of the occurrence of family stressors, which assumes that the effects of family stressors are additive, and this assumption may not always be appropriate. It may also be that individuals may differ in the degree to which stress exposure produces boosts in their drinking. Future studies should explore the degree to which the time-varying association between stress and drinking is moderated by individual differences. In addition, we utilized a measure of impulsivity that averaged across four constructs that are thought to be relatively distinct; future research should attempt to consider the individual dimensions of impulsivity more closely. Finally, the current study utilized a measure of stressors that mainly captured stressors in the family environment. Future studies should attempt to extend the state-trait framework to the occurrence of different types of stressors, and better examine the degree to which the effects of stressors are additive or not.

In conclusion, the current study demonstrated the utility of a state-trait conceptualization of family stressors across adolescence by using it to predict global and time-specific variation in alcohol use across four years of adolescence. The current results suggest that to some degree, the association between number of family stressors experienced by adolescents and growth in their drinking is due to shared risk factors, but that time-specific elevations in stressors, over and above an individual's own average, produce "boosts" in drinking at that same age. As such, the findings bring us closer to understanding the true extent to which stressor events contribute to alcohol use among adolescents.

Acknowledgments

This study was supported by Grants AA016213 from the National Institute on Alcohol Abuse and Alcoholism to Laurie Chassin, Grant AA011873 to Brooke Molina, and a National Research Service Award from the National Institute on Drug Abuse to Kevin King, Grant DA019753, We thank Patrick Curran, Andrea Hussong, Amee Epler and Stephanie Stepp for their comments and feedback on earlier drafts of this manuscript. A version of these analyses was awarded an Enoch Gordis Research Recognition Award at the 2007 Annual Meeting of the Research Society on Alcoholism. Portions of these analyses were also presented at the Annual WPIC Research Day Conference in 2007.

References

Agnew R. Foundation for a General Strain Theory of Crime and Delinquency. Criminology 1992;30:47–87.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed. Washington DC: 1980. Author

Andersen SL, Teicher MH. Desperately driven, no brakes: Developmental stress exposure and subsequent risk for substance abuse. Neuroscience & Biobehavioral Reviews. 2008 doi:10.1016/j.neubiorev.2008.09.009.

Aseltine RH, Gore SL. The variable effects of stress on alcohol use from adolescence to adulthood. Alcohol Use and Misuse 2000;35:643–668.

Baumrind D. The influence of parenting style on adolescent competence and substance use. Journal of Early Adolescence 1991;11:56–95.

Bergeman CS, Plomin R, Pedersen NL, McClearn GE, Nesselroade JR. Genetic and environmental influences on social support: the Swedish Adoption Twin Study of Aging (SATSA). Journal of Gerontology 1990;45:101–106.

Bollen KA, Curran PJ. Autoregressive latent trajectory (ALT) models: A synthesis of two traditions. Sociological Methods and Research 2004;32:336–383.

Buss, AH.; Plomin, R. Temperament: Early developing personality traits. Erlbaum; Hillsdale, NJ: 1984.

Cerbone FG, Larison CL. A bibliographic essay: The relationship between stress and alcohol use. Alcohol Use and Misuse 2000;35:757–786.

Champion L. The relationship between social vulnerability and the occurrence of severely threatening life events. Psychological Medicine 1990;20:157–161. [PubMed: 2320693]

Chassin L, Barrera M, Bech K, Kossak-Fuller J. Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. Journal of Studies on Alcohol 1992;53:316–319. [PubMed: 1619925]

Chassin L, Curran PJ, Hussong AM, Colder CR. The relation of parental alcoholism to adolescent alcohol use: A longitudinal follow-up study. Journal of Abnormal Psychology 1996;105:70–80. [PubMed: 8666713]

Chassin L, Flora D, King KM. Trajectories of Alcohol and Drug Use and Dependence from Adolescence to Adulthood: The Effects of Familial Alcoholism and Personality. Journal of Abnormal Psychology 2004;113:483–498. 2004. [PubMed: 15535782]

Chassin L, Pillow DR, Curran PJ, Molina BSG, Barrera M. The relation of parent alcoholism to adolescent alcohol use: A test of three mediating mechanisms. Journal of Abnormal Psychology 1993;102:3–19. [PubMed: 8436697]

Chassin L, Pitts S, DeLucia C, Todd M. A longitudinal study of children of alcoholics: Predicting young adult alcohol use disorders, anxiety and depression. Journal of Abnormal Psychology 1999;108:106–119. [PubMed: 10066997]

Chassin, L.; Ritter, J.; Trim, RT.; King, KM. Adolescent Substance Use Disorders. In: Mash, EJ.; Barkley, RA., editors. Child Psychopathology. 2nd Edition. Guilford Press; New York: 2003. p. 199-230.

Chassin L, Rogosch F, Barrera M. Alcohol use and symptomatology among adolescent children of alcoholics. Journal of Abnormal Psychology 1991;100:449–463. [PubMed: 1757658]

Cloninger CR. Neurogenetic adaptive mechanisms in alcoholism. Science 1987;236:410–416. [PubMed: 2882604]

Colder, CR.; Chassin, L.; Lee, MR.; Villalta, IK. Developmental Perspectives: Affect and Substance Use. In: Kassel, JD., editor. Substance Use and Emotion. American Psychological Association; (in press)

Cole DA, Nolen-Hoeksema S, Girgus J, Paul G. Stress exposure and stress generation in child and adolescent depression: A latent state-trait-error approach to longitudinal analyses. Journal of Abnormal Psychology 2006;115:40–51. [PubMed: 16492094]

Endicott, J.; Anderson, N.; Spitzer, RL. Family History Diagnostic Criteria. Biometrics Research, New York Psychiatric Institute; New York: 1975.

Foley DL, Neale MC, Kendler KS. A longitudinal study of family stressors assessed at personal interview with an epidemiologic sample of adult twins: the basis of individual variation in event exposure. Psychological Medicine 1996;26:1239–1252. [PubMed: 8931170]

Freedman LS, Graubard BI, Schatzkin A. Statistical validation of intermediate endpoints for chronic diseases. Statistics in Medicine 1992;11:167–178. [PubMed: 1579756]

Fulker DW, Eysenck SBG, Zuckerman M. A genetic and environmental analysis of sensation seeking. Journal of Personality Research 1980;14:261–281.

Furman W, Buhrmester D. Children's perceptions of the personal relations in their social networks. Developmental Psychology 1985;21:1016–1024.

Grant KE, Compas BE, Stuhlmacher AF, Thurm AE, McMahon SD, Halpert JA. Stressors and child/adolescent psychopathology: Moving from markers to mechanisms of risk. Psychological Bulletin 2003;129:447–466. [PubMed: 12784938]

Grant KE, Compas BE, Thurm AE, McMahon SD. Stress and child and adolescent psychopathology: Measurement issues and prospective effects. Journal of Child and Adolescent Psychology 2004;2:412–425.

Gunnar MR, Vazquez DM. Low cortisol and flattening of expected daytime rhythm: Potential indices of risk in human development. Development and Psychopathology 2001;13:515–538. [PubMed: 11523846]

Hawkins JD, Catalano R, Miller J. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for alcohol abuse prevention. Psychological Bulletin 1992;112:64–105. [PubMed: 1529040]

Helzer JE, Pryzbeck TR. The co-occurrence of alcoholism with other psychiatric disorders in the general population and its impact on treatment. Journal of Studies on Alcohol 1988;49:219–224. [PubMed: 3374135]

Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structural analysis: Conventional criteria versus new alternatives. Structural Equation Modeling 1999;6:1–55.

Hussong AM, Curran PJ, Moffitt TE, Caspi A, Carrig MM. Substance abuse hinders desistance in young adults' antisocial behavior. Development and Psychopathology 2004;16:1029–1046. [PubMed: 15704826]

Jackson KM, Sher KJ, Wood PK. Prospective analysis of comorbidity: Tobacco and alcohol use disorders. Journal of Abnormal Psychology 2000;109:679–694. [PubMed: 11195992]

Jackson KM, Sher KJ. Alcohol use disorders and psychological distress: A prospective state-trait analysis. Journal of Abnormal Psychology 2003;112:599–613. [PubMed: 14674872]

King KM, Meehan BT, Trim RT, Chassin L. Marker or Mediator? The relation between adolescent alcohol use and young adult educational attainment. Addiction 2006;101:1630–1640.

King KM, Molina BSG, Chassin L. A state-trait model of negative life event occurrence in adolescence: Predictors of stability in the occurrence of stressors. Journal of Clinical Child and Adolescent Psychology 2008;37:848–859. [PubMed: 18991134]

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior and personality: Modeling the externalizing spectrum. Journal of Abnormal Psychology 2002;111:411–424. [PubMed: 12150417]

Langrock AM, Compas BE, Keller G, Merchant MJ, Copeland ME. Coping with the stress of parental depression: Parents' reports of children's coping, emotional, and behavioral problems. Journal of Clinical Child and Adolescent Psychology 2002;31:312–324. [PubMed: 12149969]

Little, R.; Rubin, D. Statistical analysis with missing data. Wiley; New York: 1987.

MacKinnon DP, Fritz MS, Williams J, Lockwood CM. Distribution of the product confidence limits for the indirect effect: Program PRODCLIN. Behavior Research Methods 2007;39:384–389. [PubMed: 17958149]

MacKinnon DP, Krull JL, Lockwood CM. Equivalence of the mediation, confounding, and suppression effect. Prevention Science 2000;1:173–181. [PubMed: 11523746]

Mackinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V. A comparison of methods to test mediation and other intervening variable effects. Psychological Methods 2002;7:83–104. [PubMed: 11928892]

MacKinnon, DP.; Yoon, M.; Ryu, E.; Fairchild, A. Evaluation of the proportion mediated measure of mediation. (in preparation)Manuscript in preparation

Marsh HW, Hau KT, Wen Z. In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) findings. Structural Equation Modeling 2004;11:320–341.

Muthén, LK.; Muthén, BO. Mplus user's guide. Muthén & Muthén; Los Angeles: 1998.

Muthén, LK.; Muthén, BO. Mplus, 4.0. 2005. http://www.statmodel.com

Phillips SD, Erkanli A, Keeler GP, Costello EJ, Angold A. Disentangling the risks: Parent criminal justice involvement and Children's exposure to family risks. Criminology and Public Policy 2006;5:677–702.

Pillow DR, Barrera M, Chassin L. Using cluster analysis to assess the effects of stressful life events: Probing the impact of parental alcoholism on child stress and substance use. Journal of Community Psychology 1998;26:361–380.

Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. Archives of General Psychiatry 1981;38:381–389. [PubMed: 6260053]

Sandler, IN.; Ramirez, R.; Reynolds, KD. Life stress for children of divorce, bereaved and asthmatic children. Paper presented at the American Psychological Association Convention; Washington, DC. Aug. 1986

Satorra, A. Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In: Heijmans, RDH.; Pollock, DSG.; Satorra, A., editors. Innovations in multivariate statistical analysis. A Festschrift for Heinz Neudecker. Kluwer Academic Publishers; London: 2000. p. 233-247.

Schmitt MJ, Steyer R. A latent state-trait model (not only) for social desirability. Personality and Individual Differences 1993;14:519–529.

Sher, KJ. Children of Alcoholics: A Critical Appraisal of Theory and Research. University of Chicago Press; Chicago: 1991.

Sher KJ, Gershuny BS, Peterson L, Raskin G. The role of childhood stressors in the intergenerational transmission of alcohol use disorders. Journal of Studies on Alcohol 1997;58:414–427. [PubMed: 9203123]

Sinha R. How does stress increase risk of drug abuse and relapse? Psychopharmacology 2001;158:343–359. [PubMed: 11797055]

Smith GT, Fischer S, Cyders MA, Annus AM, Spillane NS, McCarthy DM. On the validity and utility of discriminating among impulsivity-like traits. Assessment 2007;14:155–170. [PubMed: 17504888]

Spear LP. The adolescent brain and the college drinker: Biological basis of propensity to use and misuse alcohol. Journal of Studies on Alcohol 2002;S14:71–81.

Tarter RE, Kirischi L, Mezzich A, Cornelious JR, Pajer K, Vanyukov M, Gardner W, Blackson T, Clark DB. Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. American Journal of Psychiatry 2003;160:1078–1085. [PubMed: 12777265]

Steinberg L, Fletcher A, Darling N. Parent monitoring and peer influences on adolescent alcohol use. Pediatrics 1994;93:1060–1064. [PubMed: 8197008]

Wills TA, Dishion TJ. Temperament and adolescent substance use: A transactional analysis of emerging self control. Journal of Clinical Child and Adolescent Psychology 2004;33:69–81. [PubMed: 15028542]

Wills TA, Sandy JM, Yaeger AM. Temperament and adolescent substance use: An epigenetic approach to risk and protection. Journal of Personality 2000;68:1127–1151. [PubMed: 11130735]

Wills TA, Sandy JM, Yaeger AM, Cleary SD, Shinar O. Coping dimensions, life stress, and adolescent alcohol use: A latent growth analysis. Journal of Abnormal Psychology 2001;110:309–323. [PubMed: 11358025]

Wills TA, Filer M. Stress-coping model of adolescent alcohol use. Advances in Clinical Child Psychology 1996;18:91–132.

Windle M, Windle RC. Coping strategies, drinking motives, and family stressors among middle adolescents: associations with emotional and behavioral problems and with academic functioning. Journal of Abnormal Psychology 1996;105:551–560. [PubMed: 8952188]

Windle M, Windle RC. Depressive symptoms and cigarette smoking among middle adolescents: Prospective associations and intrapersonal and interpersonal influences. Journal of Consulting and Clinical Psychology 2001;69:215–226. [PubMed: 11393599]

Zhou Q, King KM, Chassin L. The relation of familial alcoholism and adolescent family environment to young adults' alcohol and drug dependence: Mediated and moderated effects. Journal of Abnormal Psychology 2006;115(2):320–331. [PubMed: 16737396]

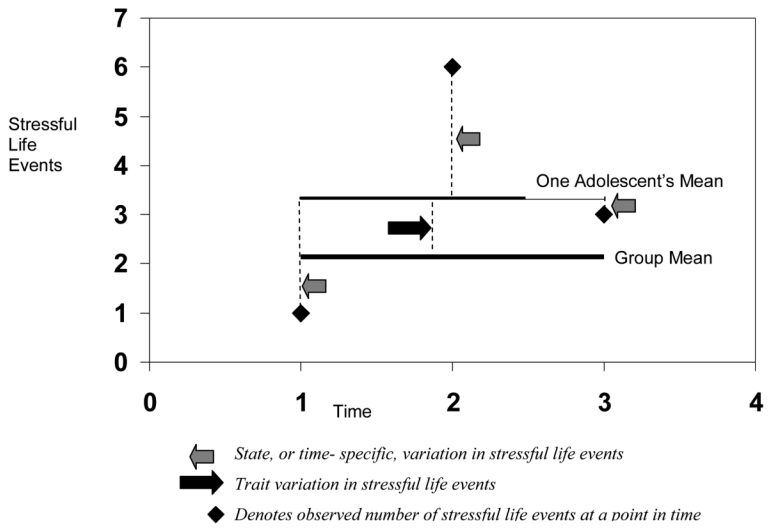

Figure 1.
Conceptual Diagram of the State-Trait Model of Family stressors.

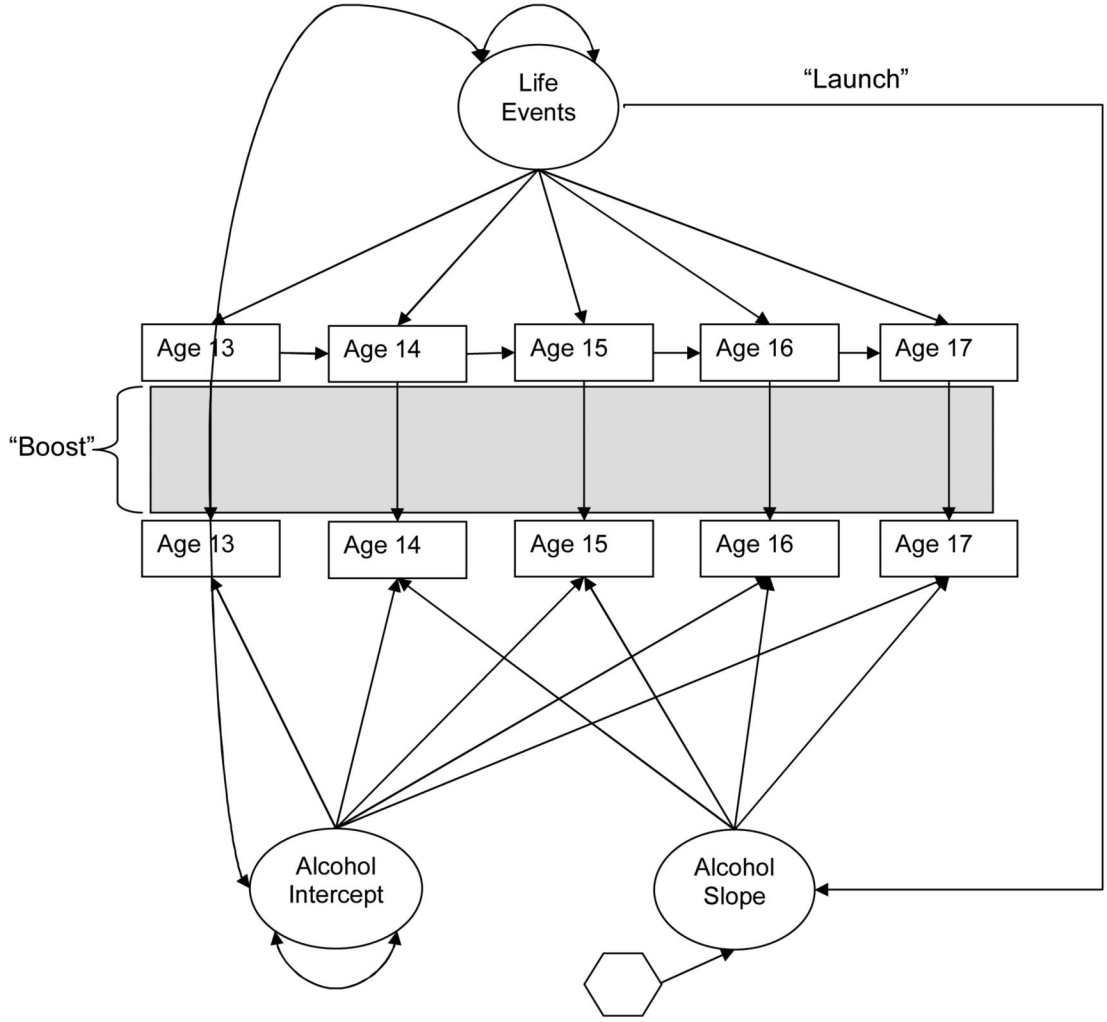

Figure 2. The Unconditional Parallel Models of State-Trait Family Stressors and Growth in Drinking Across Adolescence

This structural model illustrates the association between the parallel models of growth in drinking and state-trait family stressors. The "boost" hypothesis is tested in the effects of residualized stressors at each time point on residualized alcohol use at each time point. The "launch" hypothesis is tested by examining the effect of trait stressors on growth in alcohol use, and the "marker" hypothesis is tested by examining how much that effect is reduced by the addition of shared risk factors.

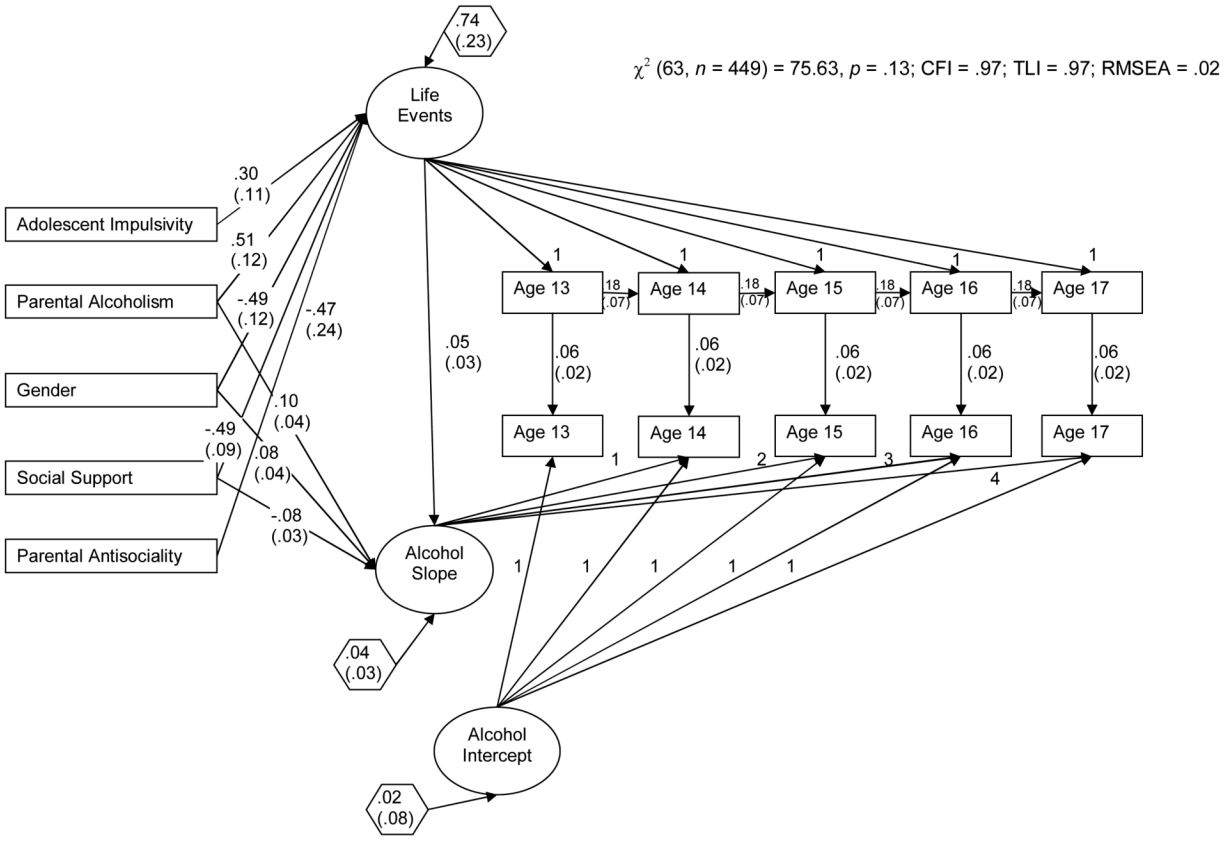

Figure 3. Relations Among Negative Family Stressors and Drinking Across Adolescence

This illustrates the final model simultaneously testing the launch, boost, marker and mediator hypotheses, displaying the unstandardized coefficients for estimated effects and residual variances for the latent variables, with standard errors in parentheses. Residual variances for the observed variables (family stressors and drinking) were estimated but are not displayed. All paths shown as solid lines were significant ($p < .05$); non-significant direct effects of the exogenous predictors on the latent variables are displayed in Table 4

Table 1

Sample Size by Cohort

	Age					
Cohort	**13**	**14**	**15**	**16**	**17**	**N**
1	X					68
2	X	X				82
3	X	X	X			101
4		X	X	X		102
5			X	X	X	76
6				X	X	22
N	250	283	275	199	96	

Table 2

Family stressors and Drinking by Age

	Number of Family stressors in the Past Year				
	Age 13	**Age 14**	**Age 15**	**Age16**	**Age 17**
n	250	283	275	199	96
M	1.82	2.05	2.03	2.13	2.10
SD	1.68	1.86	1.74	1.85	1.80

	Number of Drinks Per Month in the Past Year (untransformed)				
	Age 13	**Age 14**	**Age 15**	**Age16**	**Age 17**
n	250	283	275	199	96
M	.14	1.95	3.54	8.04	11.23
SD	.72	17.06	14.04	26.92	36.07

Table 3

Family stressors 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.

Table 4

Effects of Predictors on Latent Factors.

	Slope of Alcohol Use (log-transformed)				**Intercept of Alcohol Use (log-transformed)**			
	β	**σ 2**	**Z**	**p**	**β**	**σ 2**	**Z**	**p**
Parental Alcoholism	0.10	0.04	2.44	0.02	0.03	0.03	1.19	0.24
Parental Antisociality	0.09	0.09	0.97	0.33	0.02	0.10	0.21	0.84
Gender	0.08	0.04	1.99	0.05	−0.02	0.03	−0.55	0.58
Adolescent Impulsivity	0.04	0.03	1.11	0.27	0.01	0.02	0.62	0.54
Social Support	−0.08	0.03	−2.62	0.01	0.01	0.03	0.44	0.66

Intercept	**Latent Trait Family Stressors**			
	β	**σ 2**	**Z**	**p**
Parental Alcoholism	0.51	0.12	4.21	0.00
Parental Antisociality	0.47	0.24	2.01	0.05
Gender	−0.49	0.12	−4.17	0.00
Adolescent Impulsivity	0.30	0.11	2.86	0.00
Social Support	−0.49	0.09	−5.55	0.00

Table 5

Mediated Effects of Shared Predictors on Growth in Alcohol Use via Trait Stressors

	α*β	**σ²**	**UCL**	**LCL**
Parental Alcoholism	0.025+	0.014	0.058	−0.00079
Parental Antisociality	0.023	0.018	0.065	−0.00178
Adolescent Impulsivity	0.015	0.009	0.037	−0.00025
Social Support	−0.024+	0.013	0.001	−0.05371

+Coefficient approached significance, $p < .10$