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INTERNALIZING AND EXTERNALIZING DYSFUNCTION: AN INTEGRATIVE MODEL OF ADOLESCENT DRINKING

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INTERNALIZING AND EXTERNALIZING DYSFUNCTION: AN INTEGRATIVE MODEL OF ADOLESCENT DRINKING

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor in Philosophy in the
College of Arts and Sciences
at the University of Kentucky

By

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2017

ABSTRACT OF DISSERTATION

INTERNALIZING AND EXTERNALIZING DYSFUNCTION: AN INTEGRATIVE MODEL OF ADOLESCENT DRINKING

Separate externalizing and internalizing pathways to problem drinking have been described. However, internalizing and externalizing are substantially correlated, thus, there is good reason to believe that these two forms of dysfunction behaviors do not operate independently.

We tested an integrative developmental model of transactions among internalizing symptomatology, externalizing personality, and psychosocial learning in the prediction of both drinking problems and future internalizing symptoms. To do so, we studied a large sample (n = 1910, 49.9% female) of children over a critical developmental period, from the spring of 5th (last year elementary school) grade through the spring of 9th grade (first year of high school). Using a battery of self-report questionnaires, we assessed demographics, pubertal status, negative urgency, depressive symptoms, positive drinking expectancies, and drinking behavior. Specifically, the present study tested whether internalizing symptomatology (depressive symptoms) in elementary school predicts a classic externalizing pathway (to problem drinking in middle school, and whether problem drinking in middle school predicts increased depressive symptomatology in highschool.

Structural equation modeling yielded significant findings for hypothesized direct and indirect pathways, with overall good model fit (CFI = .94; SRMR = .05; RMSEA = .05, 90% CI .04-.05): elementary school depressive symptomatology predicted middle school drinking problems (mediated by negative urgency and psychosocial learning) and middle school drinking problems predicted increased risk for depressive symptoms in high school, pointing to a reciprocal relationship between internalizing and externalizing dysfunction.

The present study incorporated internalizing symptomatology into a traditional externalizing model of drinking risk, and demonstrated a reciprocal relationship between internalizing and externalizing dysfunction during adolescence. These findings are particularly noteworthy when considered in a developmental framework. The present

study highlights the need to integrate both internalizing and externalizing forms of dysfunction into models of substance use risk.

KEYWORDS: Depression, Alcohol, Internalizing, Externalizing, Expectancies, Adolescence

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May 22nd, 2017

INTERNALIZING AND EXTERNALIZING DYSFUNCTION: AN INTEGRATIVE MODEL OF ADOLESCENT DRINKING

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Chapter 1: Introduction

Historically, researchers have described two different forms of dysfunction. Externalizing dysfunction reflects disorders involving conflict with the external environment, and internalizing dysfunction reflects disorders involving problems within the self (Achenbach, 1966). This distinction has been replicated empirically among children (Achenbach, 1966), adults (Krueger, 1999), and adolescents (Cosgrove et al., 2011). It has also proven useful, as researchers have identified common genetic and psychosocial risk factors for externalizing disorders, such as substance use and delinquency (Kendler, Prescott, Myers, & Neale, 2003; Settles, Fischer, Cyders, Combs, Gunn, & Smith, 2012); and for internalizing disorders, such as depression and anxiety (Kendler et al., 2003; Krueger & Markon, 2006; Settles et al., 2012). With respect to substance use (SU), it has led to models delineating separate externalizing (Zucker, Donovan, Masten, Mattson, & Moss, 2008) and internalizing (Hussong, Jones, Stein, Baucom, & Boeding, 2011) pathways of risk.

However, it is also true, though under-appreciated, that the two dimensions are in fact highly correlated (Cosgrove et al., 2011; Krueger, 1999; Lahey et al., 2004). Just as there is evidence to support unique genetic underpinnings to internalizing and externalizing, there is also evidence for common genetic underpinnings, which may help explain their overlap (Cosgrove et al., 2011; Kendler et al., 2003). Thus, individuals who tend to be high in externalizing dysfunction tend also to be high in internalizing dysfunction. Despite the ubiquity of the two-factor model in psychopathology research, the two dimensions do not reflect separate pathways to, or forms of, distress.

This dissertation presents and tests empirically one model of risk for adolescent SU (specifically, alcohol consumption) that integrates externalizing and internalizing processes. The model specifies the following: internalizing psychopathology measured in elementary school predicts what is traditionally considered an externalizing pathway to harmful behaviors and alcohol-related problems in middle school. This pathway, in turn, predicts increased internalizing dysfunction in high school. That is, we tested a transactional model describing mutual, reciprocal predictive influence between classic internalizing and externalizing forms of dysfunction. We did so over the multiple developmental transitions from elementary school through middle school to high school. To introduce this empirical test, we briefly review evidence pertaining to the two factors and their overlap. We then describe the rationale for, and the specifics of, the transactional model tested. We then describe the empirical test we conducted.

The Two-Factor Model of Psychopathology: Internalizing and Externalizing

In the mid to late 20th century, Achenbach (1966) and Achenbach and Edelbrock (1978) published seminal work on the classification of child psychiatric symptoms into two higher-order dimensions, which they labeled with the terms externalizing and internalizing. The idea was that symptoms of externalizing disorders (substance use, delinquency) describe conflict with the external environment or behaviors that disrupt the lives of others, whereas symptoms of internalizing disorders (depression, anxiety) describe affective reactions and behaviors that reflect disruption in the personal sense of well-being (Achenbach, 1966). Subsequently, Krueger (1999) replicated this two-factor structure with adults and described it in similar ways: externalizing as a general predisposition toward disorders characterized by disinhibition, antisocial behaviors, and

substance use; and internalizing as a general predisposition toward negative-affect-laden mood and anxiety disorders. The internalizing-externalizing structure has been supported in child and adolescent populations as well (Lahey et al., 2004; Cosgrove et al., 2011).

An important point to appreciate about the two-factor model is that the two factors share a sizeable portion of variance; specifically, correlations between internalizing and externalizing latent factors are estimated to be $r = .51$ in adults (Krueger, 1999), $r = .72$ in adolescents (Cosgrove et al., 2011), and $r = .66$ in children (Lahey et al., 2004). In general, those who are experiencing one form of distress (such as depression) are likely to also be experiencing other forms of distress (such as SU). Indeed, considerable overlap between depression/anxiety and SU has been documented (Chen et al., 2011; Jané-Llopis & Matytsina, 2006; Kessler et al., 1994; Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Regier et al., 1990).

Applying the Two-Factor Model to Substance Use

To date, applications of this perspective to SU have emphasized the distinction between the two dimensions, not their overlap. Externalizing models of risk (Zucker et al., 2008) have identified overlap among SU, aggression, and delinquency. Risk models have identified contributors to externalizing behaviors that include both personality traits, such as the trait of negative urgency (the tendency to act rashly or impulsively when distressed); and psychosocial learning mechanisms, such as learned expectancies for reinforcement from SU and motives to engage in SU (Guller, Zapolski, & Smith, 2015a). Those models have led to successful prediction of the onset of, and increases in, alcohol and tobacco use among adolescents (Guller, Zapolski, & Smith, 2015b). Theoreticians have identified a separate risk pathway involving internalizing dysfunction (Hussong et

al., 2011). The focus of this pathway is on the experience of subjective distress and negative mood; SU behaviors are thought to provide distraction from, or relief from, one's internal distress. Support for this model includes evidence that depressive symptoms predict SU and SU predicts subsequent depressive symptoms (Birkley, Zapolski, & Smith, 2015; Saraceno, Heron, Munafò, Craddock, & van den Bree, 2012). Similarly, there is evidence to suggest that the trait negative urgency predicts depressive symptomatology during early adolescence (Smith, Guller, & Zapolski, 2013).

A New, Integrative Model

The model we developed and tested integrates the two pathways by specifying reciprocal influence between internalizing and externalizing behaviors. There is considerable evidence for the presence of what is thought of as an externalizing pathway to risk, which is referred to as the acquired preparedness (AP) model of risk. The AP model includes the following components. The trait of negative urgency reflects a disposition to act rashly when distressed and predicts engagement in numerous behaviors that provide negative reinforcement in the form of distraction from distress (Smith & Cyders, 2016). Therefore, those high on the trait find drinking reinforcing, and thus form strong expectancies for reinforcement from drinking. Elevations in drinking reinforcement expectancies, in turn, predict drinking behavior (Davis, Riley, & Smith, in press; Smith & Anderson, 2001). This model has received longitudinal support in youth making the transition from elementary to middle school (Settles, Zapolski, & Smith, 2014) and in college students (Settles, Cyders, & Smith, 2010). The AP model has also received support from longitudinal work in the areas of smoking (Doran et al., 2013) and binge eating (Pearson, Combs, Zapolski, & Smith, 2012).

We tested whether longitudinal data are consistent with the possibility that elevations in a classic form of internalizing dysfunction, depressive symptomatology, influence the operation of the AP pathway. Those with a heightened frequency of depressive symptoms may, over time, become more and more disposed to engage in action designed to alleviate their distress, even if that action does not serve their long-term interests. Thus, depressive symptomatology may predict increases in negative urgency prospectively. As a result, internalizing dysfunction may set in motion the AP externalizing risk process.

Next, because SU in general, and adolescent alcohol use in particular, are associated with so many negative consequences (Chung et al., 2012), we tested whether drinking problems following the AP risk process in turn predicted yet further increases in depressive symptomatology. Thus, the overall integrative model we tested holds that depressive symptoms predict increases in negative urgency, which predicts increases in expectancies for reinforcement from drinking, which predicts increased drinking, which then predicts further increases in depressive symptomatology. Because of transactional influences between these forms of internalizing and externalizing dysfunction, the two types of dysfunction tend to co-occur.

The Current Study

We chose to conduct this model test on an early adolescent sample for two reasons. First, we wanted to test this model in youth, before the presence of scar effects from ongoing psychopathology muddy the waters as we seek to ascertain prospective, reciprocal prediction over time (Widiger & Smith, 2008). Second, we wanted to study transactions among these risk factors during the earliest drinking years, in order to assess

the value of the model with respect to risk for very early drinking. We focused on early drinking because it predicts both concurrent and future harms, including diagnosable alcohol disorders in adulthood (Guttmannova et al., 2012).

Accordingly, in a sample of 1,910 youth, we tested a model that included the following sequence of predictions: (1) depressive symptoms present in the last year of elementary school (spring of 5th grade) predict increases in negative urgency 18 months later (fall of 7th grade); (2) negative urgency in the fall of 7th grade predicts increases in expectancies for reinforcement from drinking 6 months later (spring of 7th grade); (3) alcohol expectancies in the spring of 7th grade predict increased drinking frequency 6 months later (fall of 8th grade); (4) drinking frequency in the fall of 8th grade predicts increases in drinking problems 6 months later (spring of 8th grade); and (5) drinking problems in the spring of 8th grade predicts increases in depressive symptoms 12 months later, in the spring of 9th grade (end of the first year of high school).

Although pubertal onset is not a focus of our model, it is important to consider it in any model concerning the transition into middle school and high school. Early pubertal onset, often defined as occurring before 75% of one's peers (Lynne-Landsman, Graber, & Andrews, 2010), predicts early alcohol use and other addictive behaviors (Dick, Rose, Viken, & Kaprio, 2000 Lanza & Collins, 2002; Westling, Andrews, & Peterson, 2012). The influence of puberty is thought to reflect biological, social and contextual factors, and even parental psychopathology (Dick et al., 2007; Ellis, 2004; Ellis & Garber, 2000). Thus, we control for early pubertal onset in the empirical test of our integrative model.

Chapter 2: Method

Sample

The sample included 1910 students, recruited from local public schools as part of a larger longitudinal study funded by NIAAA. As part of this study, students were assessed at six time points: spring of 5th grade, or the last year of elementary school (Wave 1); fall and spring 7th and 8th grades, or the last two years of middle school (Waves 2-5); and spring of 9th grade, or the first year of high school (Wave 6). The mean age of the participants was just under 11 years old at study onset. The split by gender was roughly even (50.1% male; 49.9% female). Ethnicities are broken down as follows: 61.6% European American, 17% African American, 6.9% Hispanic/Latino, 3% Asian American, and 11.5% reporting other backgrounds.

Measures

Demographic and background questionnaire. This measure provided assessment of the demographic information reported above. Participants were asked the circle their gender, write in their current age (in years), and indicate which label(s) best described their ethnic background.

Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). This scale consists of five questions for boys ("do you have facial hair yet?") and five questions for girls ("have you begun to have your period?") Evidence for reliability and validity are strong (Brooks-Gunn**,** Warren**,** Rosso**,** & Gargiulo, 1987; Coleman & Coleman, 2002). We used the common dichotomous classification of the PDS (Culbert, Burt, McGue, Iacono, & Klump, 2009) as pre-pubertal or pubertal, with mean scores above 2.5 indicative of pubertal onset.

UPPS-P-Child Version. The child version of the UPPS-P (Zapolski et al., 2010), based on the UPPS-P measure of impulsivity-related traits in adults (Cyders et al., 2007; Whiteside & Lynam, 2001), assesses several impulsogenic personality traits. For the current study, we used the trait of negative urgency, the tendency to act rashly when distressed. The 8 negative urgency items are assessed on a four-point Likert-type scale, ranging from "not at all like me" to "very much like me." The internal consistency estimate of reliability was $\alpha = .85$ at wave 1 and slightly higher at subsequent waves. There is considerable evidence for the validity of the scale, as summarized in Smith and Cyders (2016).

The Drinking Styles Questionnaire (DSQ). The DSQ (Smith, McCarthy, & Goldman, 1995) was used to assess drinking behavior. We assessed drinking frequency with a single item, the values of which range from "Never engaged in the behavior" to a maximum level reflecting engagement in the behavior "daily or almost daily." Specifically, for drinking, 0 = "I have never had a drink of alcohol," 1 = "I have only had 1, 2, 3, or 4 drinks of alcohol in my life," 2 = "I only drink alcohol 3 or 4 times a year," 3 = "I drink alcohol about once a month," 4 = "I drink alcohol once or twice a week," and 5 = "I drink alcohol almost daily." We used drinking frequency because, for adolescents, the simple assessment of drinking frequency is the best predictor of the presence of alcohol use disorder symptoms (Chung et al., 2012). The drinking frequency item used in the current study has proven stable and valid in prior studies (Riley, Rukavina, & Smith, 2016; Settles et al., 2014).

We assessed drinking problems with a 14-item scale that assesses symptoms of alcohol abuse, including blackouts, work- or school-related difficulties from drinking,

legal problems as a result of drinking, experience of physical injury related to drinking, and problems with friends and family as a result of drinking. Among young adolescents, a 10 item version of the scale proved internally consistent (Alphas ranging from .66 to .80), stable (test-retest r = .81), and in agreement with collateral reports (*r*'s ranging from .36 to .53: Smith et al., 1995). The current study included the addition of 4 items to reflect common adolescent negative consequences from drinking, such as doing embarrassing things (Kahler, Strong, & Read, 2005).

Center for Epidemiological Studies-Depression Scale (CES-D). (CES-D: Radloff, 1977) is used to measure individual differences in depressive symptomology, and has previously been used in this age group (Clarke et al., 2005). The scale has demonstrated good reliability (internal consistency estimates ranging from .85 to .90) and validity in numerous studies; it is frequently used with children, adolescents, and adults (Clarke et al., 2005; Radloff, 1977, 1991; Roberts, Lewinsohn, & Seeley, 1991). We use CES-D scores as interval scale indicators of depressive symptomology. The internal reliability estimate for this measure was .85 at initial assessment in current sample and higher in later waves.

Memory Model-Based Expectancy Questionnaire (MMBEQ). The MMBEQ (Dunn & Goldman, 1996) provides an extensive assessment of alcohol expectancies in children. For the current study, we focused on the expectancy that alcohol increases positive social experiences, because that expectancy has proven most predictive of adolescent drinking behavior in prior studies (Smith, Goldman, Greenbaum, & Christiansen, 1995). The scale begins with the stem, "Drinking alcohol makes people ____." Children then read items that complete the stem (e.g., "active," "friendly," "wild,"

"mean") and then circle one of four responses: "never," "sometimes," "usually," or "always." Thus, items are scored on a Likert-type scale. Each of the subscales is correlated with drinking levels (Dunn & Goldman, 1996, 1998). Sample items and internal consistency reliability estimates from Wave 1 data are as follows: ("friendly," "fun," "outgoing"), $\alpha = .84$; internal consistency was slightly higher at later waves.

Procedure

Questionnaire administration. Participants attending local public schools were recruited using a passive consent procedure. Each family was sent a letter introducing the study. Families were asked to return an enclosed, stamped letter or call a phone number if they did not want their child to participate. Out of 1,988 5th graders in the participating schools, 1,910 participated in the study at Wave 1. A total of 72 students did not participate due to one of the following reasons: families declined to participate, students declined assent, or a variety of other reasons, such as language-based learning disabilities that precluded completing the questionnaires.

Questionnaires were administered during regular school hours in school classrooms or cafeterias. Data collections took place at 23 public elementary schools at Wave 1, then 15 public middle schools at Waves 2 through 5, and finally, 7 public high schools at Wave 6 of the longitudinal study.

At each data collection, it was made clear to the students that their responses on the questionnaire packet were to be kept confidential and no one outside of the research team would see them. The research team introduced the federal certificate of confidentiality for the project and emphasized that they were legally bound to keep all responses confidential. After each participant signed the assent form, the researchers

then passed out packets of questionnaires. Participants who moved out of the study's school districts were contacted and asked to complete the forms by mail and were paid $30 for doing so. The questionnaire administration took 60 minutes or less. This procedure was approved by the University's IRB and by the participating school systems and was utilized at all six waves of the study.

Data Analysis

Pearson product moment correlations among all the study variables were calculated (for the dichotomous variables, point biserial correlations were calculated) both within waves and across waves. Additionally, we tested for school-specific effects in order to determine if membership in individual school cohorts is related to any study variables. To do so, intraclass correlation coefficients were calculated for each study variable, using elementary school membership, $n = 23$, as the nesting variable.

The main hypothesis of the study was tested using structural equation modeling (SEM). We conducted the SEM model test in two ways. First, because of the positive skew and moderately high number of zero values for drinking frequency and drinking problems, we used a negative binomial model, which does not make distributional assumptions of normality and which handles a high number of zero values with minimal bias. However, because negative binomial modeling does not permit testing of mediational pathways, and mediational path hypotheses were important to the study (the AP model is a mediational model), we ran the analyses a second time using traditional maximum likelihood estimation, robust to violations of normality. We compared the results of the two methods. Because, as we describe below, the pattern of direct path findings was the same in the two cases, we report the results of both approaches,

including the mediation tests using maximum likelihood estimation. We used Mplus for all analyses (Muthén & Muthén, 2004). The model specified autoregressions in which each variable at one wave predicted the same variable the following wave (e.g. prediction of fall of 8th grade drinking frequency included autoregressive effects from spring of 7th grade drinking).

The structural model tested whether: (a) depressive symptomatology measured in elementary school predicted the AP pathway (urgency indirectly predicts drinking frequency through its influence on positive expectancies) in middle school, (b) drinking frequency predicts subsequent drinking problems, and (c) drinking problems, in turn, predict yet higher levels of depressive symptomatology in high school.

We tested several direct and indirect pathways involving transactions among depressive symptoms, urgency, psychosocial learning, and drinking behaviors. Specifically, direct paths included: (a) depressive symptoms in the spring of 5th to increased negative urgency by the fall of 7th grade; (b) negative urgency in the fall of 7th grade to increased positive drinking expectancies by the spring of 7th grade; (c) positive drinking expectancies in the spring of 7th grade to increased drinking frequency by the fall of 8th grade; (d) drinking frequency in the fall of 8th grade to increased drinking problems in the spring of 8th grade; and (e) drinking problems in the spring of 8th grade to increased depressive symptoms in spring of 9th grade.

Indirect paths included: (a) depressive symptoms in the spring of 5th grade to positive drinking expectancies in the spring of 7th grade (mediated by negative urgency in the fall of 7th grade); (b) negative urgency in the fall of 7th grade to drinking frequency in the fall of 8th grade (mediated by positive drinking expectancies in the spring of 7th

grade); (c) positive drinking expectancies in the spring of 7th grade to drinking problems in the spring of 8th grade (mediated by drinking frequency in the fall of 8th grade); and (d) drinking frequency in the fall of 8th grade to depressive symptoms in the spring of 9th grade (mediated by drinking problems in the fall of 9th grade). Results presented in the negative binomial model are in the form of unstandardized beta weights. Results presented in the maximum likelihood estimation model are in the form of standardized beta weights.

For the negative binomial model, we report values of the Akaike information criterion (AIC) and Bayesian information criterion (BIC) for each model. Because these indices of fit are most useful for comparing models to each other, and because we tested a single, omnibus model of our hypothesized process, these fit indices provide little information in this context.

To measure the fit of the standard, maximum likelihood estimation model, four fit indices were calculated: (a) the root mean square error of approximation (RMSEA), which reflects discrepancy between the covariances implied by the model and the observed covariances per degree of freedom; (b) the standardized root mean square residual (SRMR), which reflects the average discrepancy between the correlation matrices of the observed sample and the hypothesized model; (c) the comparative fit index (CFI); (d) and the nonnormed fit index (NNFI), both of which are based on a comparison of the chi-square value for the model with the chi-square value for a baseline model in which all variables are independent. General guidelines are that RMSEA values of .06 or lower are thought to indicate a close fit, .08 a fair fit, and .10 a marginal fit (Browne & Cudeck, 1993; Hu & Bentler, 1999); SRMR values of approximately .09 or

lower are thought to indicate good fit (Hu & Bentler, 1999); and CFI and NNFI values above either .90 or .95 are thought to represent very good fit (Hu & Bentler, 1999; Kline, 2005).

Chapter 3: Results

Participant Retention

Retention between successive waves of the study (i.e. spring of 5th to fall of 6th grade; fall of 6th to spring of 6th grade… spring of 8th to spring of 9th) ranged from 94.2% and 99.3% of prior wave participants. Retention over the entirety of the study, that is, retention between the first and final waves of the study, was: spring of 5th grade, n = 1,910; spring of 9th grade, n = 1,428 (74.8% of the overall sample). Retained and non-retained participants did not differ on any demographic, criterion, or trait variable. Therefore, we inferred that data were missing at random. Under that assumption, we imputed values for missing data using the expectation maximization procedure, which has been shown to produce more accurate estimates of population parameters than have other methods, such as deletion of missing cases or mean substitution (Enders, 2006).

Possible Effects due to School Membership

In order to determine whether there was significant covariance among the study variables due to participants attending the same school, we calculated intraclass coefficients for each variable (using elementary school membership, n = 23, as the nesting variable). Intraclass coefficients ranged from .03 (negative urgency) to .00. We therefore concluded that school membership was essentially unrelated to study variables.

Sample Characteristics

We first investigated the distributions of each variable. CES-D scores were mildly positively skewed (skew values ranged from 1.52 to 1.64) and moderately kurtotic (kurtosis values ranged from 2.72 to 3.47). For that reason, we conducted all analyses on both the original CES-D scores and on square root–transformed CES-D scores

(transformed scores were appreciably closer to normally distributed: skew values for the transformed scores were 1.23 or below, and kurtosis values ranged from 1.33 to 1.77). Because we found no difference in results between the two approaches, we report results using original CES-D scores. There was neither significant skew nor kurtosis for other study variables.

In Table 1, we report drinker status, and mean levels of depressive symptomatology at each wave of data collection. Consistent with prior work, rates of drinking increased over time: for example, the percentage of students who identified as drinkers increased from 10.5% to 46.9% between the first and final waves of the study. Mean scores for depression additionally increase over the four-year period.

Bivariate Correlations

Table 2 provides estimates of the uncorrected bivariate relationships among study variables. For ease of presentation, the correlation matrix presents variables at time points that correspond with those represented in this study's primary structural model. For example, the bivariate correlation matrix includes depression in the spring of 5^{th} and 9^{th} grades only. This was done to remain consistent with variable time points emphasized in the main reciprocal model tested in this study. As seen in Table 2, negative urgency, depressive symptoms, and drinking behaviors were correlated at each wave.

Table 1

Descriptives of Drinker Status and CES-D Scores

	Spring 5th	Fall 7th	Spring 7th	Fall 8th	Spring 8th	Spring 9th
Drink N (%)	10.5%	18.1%	22.1%	28.8%	29.7%	46.9%
CES-D Mean (SD)	14.70 (8.43)	14.16 (9.92)	13.68 (8.60)	12.80 (8.25)	13.97 (8.96)	16.28 (9.61)

Table 2

Bivariate Correlations of Key Study Variables

	Sex	PU 5s	DE 5s	DE 8s	DE 9s	NU 5s	NU 7f	EX 7f	EX 7s	DF 7s	DF 8f	DP 8f	DP 8s
PU s5	.00	-											
DE s5	.00	.14**	-										
DE s8	.00	.15**	.31**	-									
DE s8	.00	.07*	.28**	.54**	-								
NU s5	.00	.11**	.40**	.22**	.21**	-							
NU f7	-.02	.12**	.27**	.30**	.30**	.43**	-						
EX f7	-.01	.09**	.11**	.19**	.19**	.22**	.35**	-					
EX s7	.03	.10**	.11**	.22**	.19**	.16**	.27**	.54**	-				
DF s7	.02	11**	.06*	.25**	.19**	.16**	.25**	.25**	.34**	-			
DF f8	-.01	.13**	.07**	.26**	.19**	.18**	.23**	.23**	.29**	.53**	-		
DP f8	.03	.12**	.07**	.19**	.17**	.17**	.23**	.19**	.24**	.49**	.62**	-	
DP s8	.04	.12**	.07*	.29**	.21**	.13**	.21**	.18**	.20**	.41**	.44**	.48**	-
DP s9	.04	.13**	.11**	.25**	.32**	.17**	.25**	.22**	.24**	.31**	.35**	.31**	.40**

Note. **p < .001; *p < .01; PU = Puberty; DE = Depression; NU = Negative Urgency; EX = Expectancies; DF = Drinking Frequency, DP = Drinking Problems; f = Fall, s = spring; Numbers = grade (e.g. 5 = 5th grade)

Test of Reciprocal Model

The central test of this study was whether internalizing and externalizing symptomatology transact in a reciprocal manner across a four-year period marked by critical developmental transitions from elementary school to middle school to high school. Again, we tested our hypothesized process with structural equation modeling. Figure 1 presents the primary results of this model test, using negative binomial estimation and unstandardized beta weights. Figure 2 presents the same results, using standard maximum likelihood estimation, robust to violations of normality. Comparison of the two models shows that the pattern of statistical significance is the same using the two approaches. Significant predictive effects reported were present above and beyond prediction from prior behavior and pubertal status. For the negative binomial model, AIC = 74,492.04 and BIC = 74,891.99. Fit indices using maximum likelihood estimation provide a means for judging overall model fit, and model fit was good (CFI = .94; SRMR = .05; RMSEA = .05, 90% CI .04-.05).

Direct. Each of the direct pathways hypothesized to be present were found to be significantly greater than 0. Depression in the spring of 5th grade predicted increases in negative urgency beyond prior levels of the trait 18 months later, in the fall of 7th grade. Negative urgency in the fall of 7th grade predicted increased endorsement of positive drinking expectancies in the spring of 7th grade. Positive drinking expectancies in the spring of 7th grade predicted increased drinking frequency in the fall of 8th grade. Drinking frequency in the fall of 8th grade predicted increased problems related to

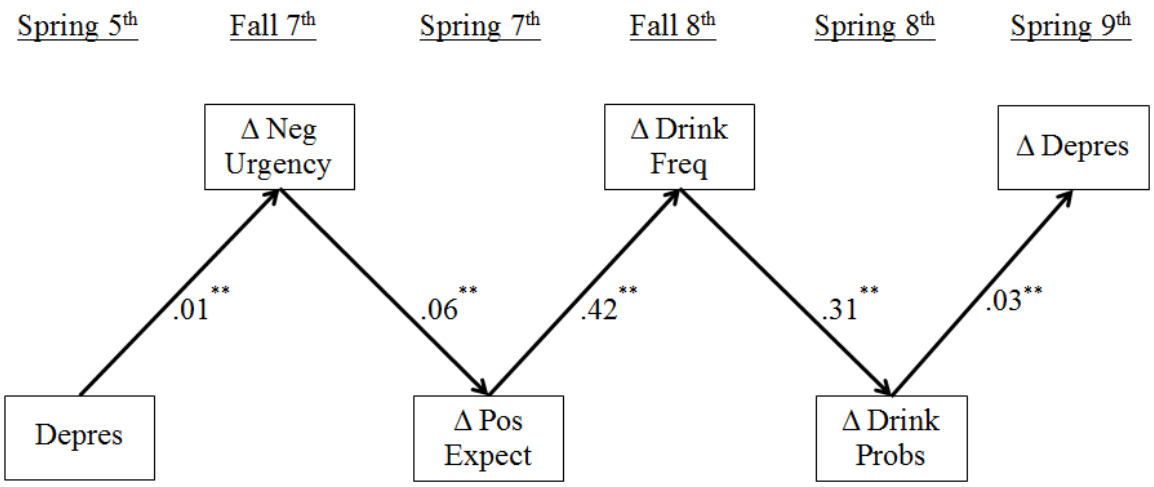

Figure 1. Negative Binomial Model (simplified). ** p < .001; Values are unstandardized beta weights; Δ = prediction above and beyond autoregressive effects; Depres = depression; Neg Urgency = negative urgency; Positive Expect = positive expectancies; Drink Freq = drinking frequency; Drink Probs = problems from drinking.

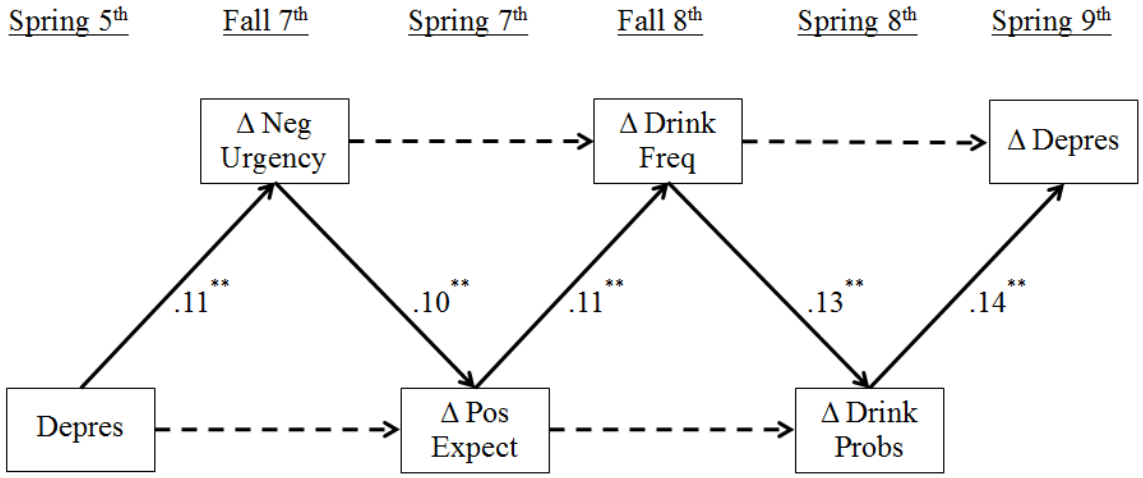

Figure 2. Maximum Likelihood Model (simplified). ** p < .001; Values are standardized beta weights; Δ = prediction above and beyond autoregressive effects; Depres = depression; Neg Urgency = negative urgency; Positive Expect = positive expectancies; Drink Freq = drinking frequency; Drink Probs = problems from drinking; - - - -► = mediated pathways.

drinking in the spring of 8th grade. Finally, drinking problems in the spring of 8th grade predicted increased depressive symptomatology in the spring of 9th grade.

Indirect. All but one of the indirect (mediational) pathways yielded significant values: depression in the spring of 5th grade predicted positive drinking expectancies in the spring of 7th grade through an indirect path mediated by negative urgency in the fall of 7th grade ($\beta = .01$, $p < .001$). Negative urgency in the fall of 7th grade predicted drinking frequency in the fall of 8th grade through an indirect path mediated by positive drinking expectancy formation in the spring of 7th grade ($\beta = .01$, $p < .001$). Significant findings from this indirect test offer support for and are consistent with previous research on the acquired preparedness model of drinking risk. Positive drinking expectancies in the spring of 7th grade predicted drinking-related problems in the spring of 8th grade through an indirect path mediated by drinking frequency in the fall of 8th grade ($\beta = .01$, $p < .01$). The final indirect test, the path from drinking frequency in the fall of 8th grade to depression in the spring of 9th grade mediated by drinking problems in the spring of 8th grade, did not quite reach statistical significance ($\beta = .02$, $p = .06$).

Chapter 4: Discussion

Elementary school endorsement of depressive symptomatology predicted middle school drinking problems, mediated by an established externalizing risk process, which in turn, predicted heightened depressive symptomatology in the first year of high school. Prior research in both child and adult samples has offered support for an AP model of drinking, a process by which personality increases risk due to its influence on the psychosocial learning process of expectancy formation (Settles et al., 2010; Corbin, Iwamoto, & Fromme, 2011), however, these studies only looked at externalizing personality. The present study not only incorporated internalizing symptomatology into the externalizing AP pathway to drinking, but also demonstrated a reciprocal relationship between the two forms of dysfunction. The findings of the study provided clear support for the model underlying the empirical test. It thus seems possible that models specifying transactions between internalizing and externalizing forms of dysfunction may help explain the co-occurrence of the two. That is, subjective distress can lead to actions that disrupt the lives of others, which, in turn, can further heighten subjective distress.

Support for this transactional model is noteworthy for several reasons. First, depression in 5th grade predicted subsequent externalizing behavior above and beyond several other key predictors, including pubertal status and autoregressions. Second, this pathway emerged early in development (mean participant age at wave 1 was 11), which reduces the likelihood that the depression-externalizing relationship was an artifact of scar effects, i.e. effects reflecting ongoing psychopathology and life disruption. Third, this model was supported during a crucial period of development, marked by two important life transitions – the transition from elementary school into middle school, and

the transition from middle school into high school – so prediction of reciprocal relationships between internalizing symptomatology and externalizing behaviors across these transitions may lead to insight regarding maladaptive transitions during this stage of development. Fourth, these findings emerged over a four-year period spanning late childhood and early/mid-adolescence, which is particularly striking, given the extensive degree of change that takes place during these crucial stages of development.

These findings provide clear support for the utility of using an integrative approach to the development of risky behavior like problem drinking, as well as mood pathology like depression. This approach may be particularly beneficial for future intervention and prevention efforts aiming to promote healthy development among individuals entering the critical stage of adolescence, which is considered particularly high-risk in terms of potential emergence and stabilization psychopathology (Hudziak & Novins, 2013; Settles & Smith, 2015).

There are at least two possible explanations for the extensive overlap between internalizing and externalizing forms of dysfunction: (a) common etiological underpinnings shared by both forms of dysfunction, and (b) transactional relationships between the two forms of dysfunction over time. The present study models reciprocal effects between depression and an externalizing path to drinking, thus supporting the second explanation. Other researchers, however, have sought to understand the overlap between internalizing and externalizing dysfunction using the former, etiological approach. Caspi and colleagues (2014) identified what is referred to as the p-factor: a one-factor solution to psychopathology, which subsumes both internalizing and externalizing factors from the two-factor solution. Using a factor analytic approach

similar to that used in the two-factor model, researchers demonstrated that the higher-order p-factor is associated with greater life impairment, worse developmental histories, and more compromised early-life brain function.

Taking a cognitive neuroscience perspective, Carver, Johnson, and Joorman (2008) identified the same brain process as underlying both externalizing dysfunction and depression, specifically low levels of serotonin in a functional brain system relating the orbitofrontal and ventromedial prefrontal cortex to the amygdala and striatum (Carver et al., 2008; Cyders & Smith, 2008). Davidson (2003) makes a similar observation, i.e. that low serotonin in this brain system is associated with deficits in affect-guided planning, or the capacity to stay affectively connected to one's long-term goals override intense emotions in order to meet these goals.

It is likely that both types of explanation have merit: shared etiology together with transactional risk processes can both occur. Both explanations have implications for prevention and treatment. To the degree there is a common cause for the two types of dysfunction, as argued by Carver et al. (2008), interventions that address that common cause may have transdiagnostic efficacy. To the degree the two types of dysfunction transact, as shown in the current study, interventionists may need to consider and plan for anticipated downstream effects of a given problem behavior.

These findings should be viewed in the context of the limitations to the study. Although the focus on the elementary, middle, and high school years is strength of this research, it is also a weakness. The emphasis on late childhood and early/mid-adolescence prevented us from collecting information the relationship between internalizing and externalizing dysfunction at other important periods of development

(e.g. early adulthood). In addition, although there is good evidence for the reliability and validity of the self-report items used in this study, reliance on pen-and-paper questionnaires limited our ability to clarify items and content, as would have been possible in an interview-style assessment. Studying a subset of transactions, i.e. those limited to depression, personality, psychosocial learning, and drinking, does not offer information on other potentially important contributors to risk. Furthermore, studying narrow forms of internalizing (depression) and externalizing (drinking) symptomatology limits the applicability of this research to other forms of dysfunction within the two factors (e.g. anxiety on the internalizing factor, aggression on the externalizing factor), and beyond. Last, although the model was prospective in design and included tight controls, our predictive findings are not a rigorous test of causality. The model underlying this test involves reciprocal causality; the current correlational findings should be viewed as consistent with, but not a demonstration of, a causal risk process.

Despite these limitations, the findings of this study do support the possibility that drinking and depression transact as early as the elementary, middle, and high school years. More broadly, the findings of this study indicate the value of risk models that incorporate the overlap between the internalizing and externalizing domains. Future research can add to the current demonstration to develop more comprehensive transactional models of risk.

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PUBLICATIONS

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Zapolski, T. C. B., **Guller, L.**, Smith, G. T. (2013). On the valid description of personality dysfunction. In P. T. Costa, Jr. & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (3rd ed., pp. 29-42). American Psychological Association.

MacPherson, L., Calvin, N. T., Richards, J. M., **Guller, L.**, Mayes, L. C., Crowley, M., Daughters, S. B., & Lejuez, C. W. (2012). Development and preliminary validation of a behavioral task of negative reinforcement underlying risk taking and its relation to problem alcohol use in college freshmen. *Alcoholism: Clinical and Experimental Research*, *36*, 426-433.

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Chen, K. W., Banducci, A. N., **Guller, L.**, Macatee, R. J., Lavelle, A., Daughters, S. B., & Lejuez, C. W. (2011). An examination of psychiatric comorbidities as a function of gender and substance type within an inpatient substance use treatment program. *Drug and Alcohol Dependence, 118,* 92-99.

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CONFERENCE PRESENTATIONS

Guller, L., & Smith, G. T. (2017, April). *Internalizing and externalizing: A transactional model of depression and drinking across the elementary, middle, and high school years.* Poster presented at the Kentucky Psychological Association Spring Academic Conference, Louisville, Kentucky.

Guller, L., & Smith, G. T. (2016, June). *Personality and the overlap between drinking and internalizing symptoms: An integrative approach.* Poster presented at the 39th annual Research Society on Alcoholism, New Orleans, Louisiana.

Guller, L., & Smith, G. T. (2015, June). *Individual differences and the prediction of drinking during the transition from middle school to high school*. Poster presented at the 38th annual Research Society on Alcoholism, San Antonio, Texas.

Guller, L., & Smith, G. T. (2015, April). *A reciprocal model of externalizing risk and depression across adolescence*. Poster presented at the 5th annual Children at Risk Conference, Lexington, Kentucky.

Guller, L., & Smith, G. T. (2014, June). *Integrating externalizing and internalizing pathways to problem drinking across adolescence*. Paper presented at the 37th annual Research Society on Alcoholism, Bellevue, Washington.

Guller, L., & Smith, G. T. (2014, June). *Integrating externalizing and internalizing pathways to problem drinking across adolescence*. Poster presented at the 37th annual Research Society on Alcoholism, Bellevue, Washington.

Davis, H. A., **Guller**, L., & Smith, G. T. (2014, April). *Developmental trajectories of fasting behavior and excessive exercise across the middle school years*. Poster presented at the 4th annual Children at Risk Conference, Lexington, Kentucky.

Guller, L., Han, D. Y., Harp, J. P., & Anderson, A. J. (2014, February). *Cognitive phenotype of PARK2-associated early-onset Parkinson's disease in two siblin*gs. Poster presented at the 42nd annual International Neuropsychological Society, Seattle, Washington.

Guller, L., Zapolski, T. C. B., & Smith, G. T. (2013, June). *Predicting the rate of increase in drinking across four years of early adolescence from elementary school personality*. Paper presented at the 36th annual Research Society on Alcoholism, Orlando, Florida.

Guller, L., Zapolski, T. C. B., & Smith, G. T. (2013, June). *Predicting the rate of increase in drinking across four years of early adolescence from elementary school personality*. Poster presented at the 36th annual Research Society on Alcoholism, Orlando, Florida.

Guller, L., Zapolski, T. C. B., & Smith, G. T. (2013, March). *The dual process model of impulsivity and depression*. Poster presented at the 3rd annual Children at Risk Conference, Lexington, Kentucky.

Guller, L., Zapolski, T. C. B., & Smith, G. T. (2012, June). *Longitudinal prediction of risky behaviors during the transition to middle school*. Poster presented at the 35th annual Research Society on Alcoholism, San Francisco, California.

Guller, L., Zapolski, T. C. B., & Smith, G. T. (2012, March). *Predicting risky behaviors during the transition to middle school*. Poster presented at the 2nd annual Children at Risk Conference, Lexington, Kentucky.

MacPherson, L., Reynolds, E.K., Wang, F.L., Dachman, L., **Guller, L.**, Lejuez, C.W. (2010, November*). Predictive and time-varying relationships between risk taking propensity and risky behaviors in adolescence*. Paper presented at the 44th annual convention of the Association for Behavioral and Cognitive Therapies, San Francisco, California.

MacPherson, L., Calvin, N.T., Richards, J.F., **Guller, L.**, & Lejuez, C.W. (2010, June). *Development of a behavioral task of negative reinforcement underlying risky alcohol use*. Paper presented at the 33rd Annual Scientific Meeting of the Research Society on Alcoholism, San Antonio, Texas.

FELLOWSHIPS AND GRANTS

July 2012 - June 2014 — **National Institute of Drug Abuse**
T32 Pre-doctoral Training Grant (T32DA035200)
$22,032 plus tuition

August 2010- July 2012 — **University of Kentucky**
Lyman T. Johnson Fellowship
$15,000 plus tuition

HONORS AND AWARDS

April, 2017 — **Graduate Student Poster Award, Second Place Winner**
Kentucky Psychological Foundation Spring Academic Conference
Louisville, Kentucky

June, 2015 — **Student Merit Award**
38th Annual Research Society on Alcoholism
San Antonio, Texas

June, 2014 — **Enoch Gordis Research Recognition, Pre-Doctoral Finalist**
37th Annual Research Society on Alcoholism
Bellevue, Washington