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Alcohol Abuse and Dependence Symptoms: A Multidimensional Model of Common and Specific Etiology

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Abstract

This study tested a theoretical model hypothesizing differential pathways from five predictors to alcohol abuse and dependence symptoms. The participants were college students (*N*= 2,270) surveyed on two occasions in a 6-month prospective design. Social norms, perceived utility of alcohol use, and family history of alcohol problems were indirectly associated with Time 2 (T2) abuse and dependence symptoms through influencing level of alcohol consumption. Poor behavioral control had a direct effect on alcohol abuse but not dependence symptoms at T2, whereas affective lability exhibited a direct prospective effect on alcohol dependence but not abuse symptoms. A multigroup analysis showed that high levels of poor control increased the strength of paths from both consumption level and affective lability to abuse symptoms. Implications for prevention of alcohol problems among college students are discussed.

Keywords: Alcohol abuse and dependence, self-control, affect regulation, etiology

This study tests a theoretical model positing that alcohol abuse and dependence have differential relationships with five conceptual domains of variables: affective lability, poor behavioral control, social norms, personal utility of alcohol use, and family history of alcohol problems. Alcohol-related problems are multifaceted with a complex etiology and course (American Psychiatric Association, 2000; Baer, 2002; Schulenberg & Maggs, 2002). Understanding alcohol use and misuse requires identifying factors that support the initiation and maintenance of alcohol use pat-

terns, account for the plasticity of behavior while intoxicated (e.g., type and likelihood of abuse symptoms, negative consequences), and contribute to progression to alcohol dependence (Edwards, 1974, 1986; Jackson, Sher, & Park, 2005; Leonard & Blane, 1999; Zucker, 1994). Our theory suggests that alcohol consumption and symptoms of abuse and dependence arise, in part, from theoretically distinct psychological mechanisms. Examining these mechanisms in a multivariate model can improve our understanding of these constructs, their etiology, and course.

Alcohol Abuse and Dependence

Alcohol-related problems have historically been divided into abuse symptoms (e.g., associated adverse consequences, conduct problems) and dependence symptoms (e.g., compulsive use behavior, craving, withdrawal, tolerance). The distinction between abuse and dependence as qualitatively distinct constructs has an extensive background (Edwards, Gross, Keller, & Moser, 1976) and this qualitative distinction has been supported by factor analytic studies in adolescent and adult samples (Grant et al., 2007; Harford & Muthen, 2001; Wills et al., 2002). Furthermore, though results have been mixed, the dimensions have been differentially associated with variables such as alcohol consumption and family history of alcohol problems, both being more strongly related to dependence than to abuse (Hasin, van Rossem, McCloud, & Endicott, 1997; Knight et al., 2002; Schuckit & Smith, 2001).

However, recent studies using item response theory have focused on identifying a unidimensional model of alcohol problem severity (Martin, Chung, Kirisci, & Langenbucher, 2006; Saha, Chou, & Grant, 2006). This research indicates that some symptoms are not uniformly predictive of degree of alcohol involvement across demographic groups, suggesting that they are indicators of another related process instead of alcohol problem severity. For example, abuse symptoms of hazardous use, legal problems, and getting in physical fights are more likely to be endorsed by men than women, and represent less severe alcohol involvement for men (Kahler & Strong, 2006; Martin et al., 2006). These symptoms may be indicators of self-control deficits rather than specific symptoms of alcohol use disorder (Martin et al., 2006; Mason & Windle, 2002). Recently, Martin and colleagues have suggested that alcohol use disorder be defined by a single dimension of alcohol dependence and that clinically significant, but non-criterion, associated consequences be described and possibly shifted to V codes in the DSM-V (Martin, Chung, & Langenbucher, 2008). They argue that these associated consequences (e.g., some problem behavior symptoms) should not be considered a disorder or criterion symptoms. This differentiation between alcohol use disorder and associated consequences is particularly relevant to the study of alcohol-related problems in college students. For many college students, heavy alcohol use and associated consequences appear to be a time-limited developmental process that may be a function of the unique social and environmental factors of college (Schulenberg et al., 2001; Schulenberg & Maggs, 2002). Students often have new-found levels of freedom, limited social responsibilities, and are in an environment where alcohol is often readily available, and its use (at times heavy use) is socially promoted.

In summary, both multidimensional factor models and unidimensional item response models suggest that some abuse symptoms represent a potentially different type of alcohol-related problem that may be qualitatively different from level of alcohol involvement and dependence. College students may exhibit patterns of hazardous drinking and associated problems that, while risky, may

not be signs of an alcohol disorder per se. This underscores the importance of examining psychological mechanisms specific to the development of alcohol abuse (i.e., negative consequences) or dependence, independent of their common association with heavy alcohol consumption.

Affective Lability and Alcohol Dependence

Affective lability may be differentially associated with alcohol abuse and dependence. Affective lability refers to the frequency, speed, and range of changes in affective states. The affective processing model of negative reinforcement posits that fluctuation in negative affect is the core mechanism driving substance dependence (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). This model integrates the role of both "external" (e.g., stress) and "internal" (e.g., withdrawal) sources of negative affect in influencing cognitive processing, creating response biases, and as a stimulus for drug consumption. The model suggests that subtle withdrawal symptoms (e.g., hangover) can occur relatively early in a use trajectory and are important initial determinants of continued use behavior. Repeated use and withdrawal cycles sensitize the individual to stimuli that signal negative affect. At low levels of negative affect, early signals of impending negative affect or signs of dropping levels of the drug may be detected and motivate drug use outside of conscious awareness. In contrast, high levels of negative affect promote "hot" information processing (Metcalfe & Mischel, 1999). The "hot" system is characterized as an emotion-based, reflexive system, optimized for quick processing and stimulus response. High negative affect is posited to reduce deliberative cognitive control processes and create strong response biases, which greatly increase the incentive value of drug use and decrease the incentive value of non-drug alternatives. Importantly, negative affect driving this process and influencing cognitive processing and responses may arise from either acute withdrawal or other external or internal determinants of negative affective states.

Given the importance of sensitization to negative affective states, high levels of negative affect, and rapid changes in negative affect in this model, we propose that individuals characterized by high levels of affective lability are at increased risk for the compulsive drinking patterns characteristic of dependence. Although lability per se is not central to Baker and colleagues' model, lability is positively associated with negative affectivity and fluctuation in negative affective states that are central to the negative reinforcement model. Indeed, affective lability was significantly associated with alcohol dependence but not abuse symptoms in an inpatient substance use treatment sample (Simons, Oliver, Gaher, Ebel, & Brummels, 2005c). For college students, affective lability is frequently unrelated or negatively associated with level of drinking but is positively associated with composite measures of alcohol-related problems (Simons & Carey, 2006; Simons, Gaher, Correia, Hansen, & Christopher, 2005a). We hypothesized that affective lability is a factor contributing specifically to the development of a pattern of consumption indicative of alcohol dependence.

Poor Control and Alcohol Abuse

Poor control of behavior (e.g., impulsivity) has been linked to alcohol use in several populations (Brody & Ge, 2001; Miller & Brown, 1991; Patock-Peckham, Cheong, Balhorn, & Nagoshi, 2001), and here we propose that it will increase the conduct problems characteristic of alcohol abuse. Poor behavioral control is characterized by lack of restraint, planning, and forethought; exhibiting premature responding and poor delay of gratification; difficulty in fitting behavior to situational

demands; and failure to consider risks (Daruna, Barnes, McCown, Johnson, & Shure, 1993; Plutchik & Van Praag, 1995; Wills, Walker, Mendoza, & Ainette, 2006). Poor control may lead to a variety of deleterious outcomes as behavior may be governed more by immediate "hot" cognition and relatively less by deliberative controlled processing of distal consequences (Carver, 2005; Metcalfe & Mischel, 1999). Problems such as interpersonal conflict, assault and other illegal behavior, and neglecting social responsibilities all occur at increased rates among people characterized by impulsiveness and poor control (Carver, 2005; Mason & Windle, 2002; Sher & Trull, 1994; Wills & Dishion, 2004). Whereas alcohol intoxication may increase the likelihood of these behaviors, poor control may also increase the likelihood of these behaviors independent of consumption. We hypothesized that poor control is a factor contributing to abuse symptoms, independent of level of alcohol consumption.

Moderation Effects of Poor Control

Both alcohol intoxication and the heightened emotional arousal associated with labile affect can reduce deliberative cognitive control processes (Baker et al., 2004; Curtin & Fairchild, 2003; Fillmore, Carscadden, & Vogel-Sprott, 1998; Metcalfe & Mischel, 1999; Steele & Josephs, 1990). Consequences of such reduced deliberative control effects may be most pronounced among individuals with a high level of poor control. The affective processing model of dependence discussed above posits that heightened negative affect reduces conscious cognitive control processes leading to compulsive consumption. Poor control may amplify this process due to pre-existing deficits in deliberative cognitive processing. For example, though support has been mixed, some research suggests that poor control may increase associations between affective lability and alcohol problems as well as negative affect and both heavy use and problems (Hussong, Hicks, Levy, & Curran, 2001; Simons, Carey, & Gaher, 2004; Simons et al., 2005b). Affective lability and poor behavioral control are moderately to strongly correlated and thus may be expected to have some common effects on both abuse and dependence (Simons & Carey, 2006; Wills et al., 2006). Thus, we are not proposing that there should be a clear demarcation of associations. For example, in addition to poor control, lability may be relevant to interpersonal conflicts or assault characteristic of abuse. Such effects may be most pronounced in individuals with a higher level of poor control.

Similarly, some alcohol-related problems including both abuse and dependence symptoms may derive, in part, from an effect of alcohol for reducing information processing and cognitive control processes, hence narrowing response options and increasing focus on immediate cues (Baker et al., 2004; Curtin & Fairchild, 2003; Fillmore et al., 1998; Metcalfe & Mischel, 1999; Steele & Josephs, 1990). Poor control may amplify this process as well. Poor control has been found to moderate the relationship between alcohol use and related problems, increasing the strength of associations (Neal & Carey, 2007; Simons et al., 2005b), and research has provided some evidence of greater moderating effects of poor control on the relationship between substance use and abuse rather than dependence symptoms (Wills et al., 2002).

Indirect Predictors of Problems via Alcohol Consumption

While affective lability and poor behavioral control may be particularly relevant for the development of abuse and dependence symptoms, it should be recognized that alcohol consumption among college students is a highly social activity (Schulenberg & Maggs, 2002). Socio-cultural factors such as social norms, fraternity/sorority membership, or perceived utility of alcohol for attaining personal goals are important factors supporting the initiation and maintenance of drinking patterns (Park, Sher, & Krull, 2008; Read, Wood, Kahler, Maddock, & Palfai, 2003; Simons & Carey, 2006). The associations of these factors with problems are often indirect, through influencing level of consumption, rather than directly predictive of use-related problems (Read et al., 2003; Simons & Carey, 2006; Wills et al., 2002).

In addition, several studies have observed elevated rates of alcohol use disorders and alcohol-related problems among children of alcoholics (Jackson, O'Neill, & Sher, 2006; Kushner & Sher, 1993; Perkins & Berkowitz, 1991; VanVoorst & Quirk, 2003). Among adolescents, the association between family history and alcohol problems is mediated by level of alcohol use (Stice et al., 1998). Recent longitudinal research with adolescents revealed that family history of alcoholism was positively associated with trajectory groups defined by either adolescent-limited problem drinking or escalating (into adulthood) problem drinking, compared with a low- or no-problem-drinking group (Warner, White, & Johnson, 2007). However, family history did not differentiate the two problem drinking groups. Family history may be associated primarily with rates of drinking rather than a specific risk for developing a persistent alcohol use disorder.

Present Research

This prospective study tested antecedents and moderators of alcohol consumption, abuse, and dependence. Some variables (alcohol norms, utility, and family history) were hypothesized to act primarily by promoting a higher level of alcohol consumption, itself a risk factor for abuse or dependence. Other variables (affective lability, poor behavioral control) were hypothesized to have direct effects to abuse or dependence symptomatology. In addition, poor control was hypothesized to moderate the effects of several other variables. A heuristic model of the primary hypotheses is presented in Figure 1.

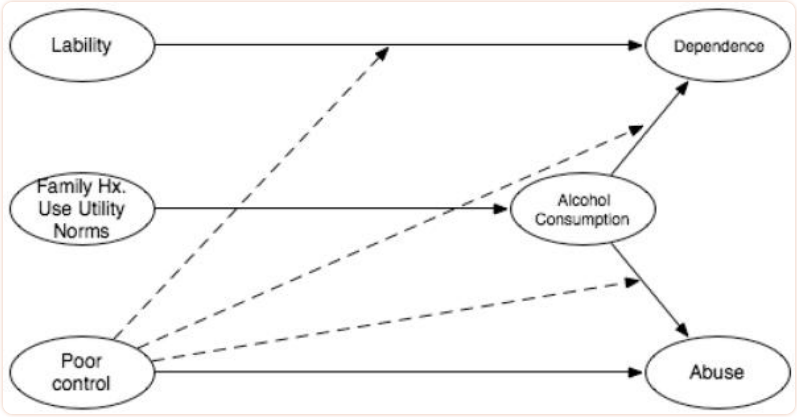

Figure 1

Heuristic model depicting primary hypotheses. Dashed lines are moderation effects.

Methods

Participants

Participants were 2270 college students recruited from a state university. Women made up 64% of the sample. The sample ranged in age from 18-25 years (M = 19.59, SD = 1.51). Ninety-four percent of the participants were White, 1% Asian, 1% Black, 1% Native American/Alaskan Native, and 3% other or did not respond. Ninety-eight percent were non-Hispanic. These demographic characteristics are comparable to the university population (Regents Information Systems, 2006). Participants were recruited through e-mail, fliers, and advertisements in the student newspaper. The study was described as a study of emotions, personal goals, and alcohol and other risk behavior. All enrolled undergraduates were eligible for recruitment. Eighty-seven percent of the baseline sample returned for a 6-month follow-up (T2), and 94% of returning participants were successfully matched to their baseline (T1) data. Missing data were replaced as needed by best subsets regression imputation using the Stata 9.0 impute module using existing Time 1 (T1) or Time 2 (T2) data as predictors (StataCorp, 2005). Specifically, partially completed scales had items imputed before creating total scores and scale scores required for defining the analysis groups (i.e., poor control and T2 six-month drinking) were imputed. However, the structural equation models were conducted with missing data modeled through Full Information Maximum Likelihood (FIML; (Enders & Bandalos, 2001; Muthen & Muthen, 2006). One previous manuscript has been published from this dataset (Simons, Dvorak, & Batien, 2008).

Procedure

Participants completed on-line questionnaires under the supervision of a research assistant. Participants had adequate space to protect their privacy and provided informed consent for participation. Participation took approximately 45 minutes. The study was approved by the human

subjects institutional review board. Responses were identified by a user-defined code, ensuring participants' anonymity. Participants received $20 for the baseline assessment and $30 for a 6-month follow-up assessment. Average time interval was 203 days (*SD* = 50.35).

Measures

Alcohol consumption was assessed by three indicators. First, the Modified Daily Drinking Questionnaire (DDQ-M; Dimeff, Baer, Kivlahan, & Marlatt, 1999) consisted of a grid representing the seven days of the week. The grid assessed participants' typical daily alcohol consumption for a typical week during the last six months. Weekly consumption was calculated by summing the number of standard drinks (one standard drink is equal to 12 oz. beer, 5 oz. wine, or 1.5 oz. liquor) across the number of drinking days reported by the participant. Second, frequency of use of alcohol in the past six months was assessed with a 9-point anchored rating scale (0 = no use, 8 = more than once a day). Third, quantity of alcohol consumed per drinking day in the last six months was assessed by an 11-point scale (0 = no drinks [i.e., non-drinker], 10 = 19 or more drinks). This scale was adapted from (Dimeff, Baer, Kivlahan, & Marlatt, 1999). The DDQ-M, the frequency rating scale, and the quantity rating scale were indicators of an alcohol consumption latent variable.

Abuse and dependence symptoms in the last 6 months were assessed by a DSM-IV alcohol abuse and dependence symptom checklist (Knight et al., 2002). The 10-item abuse checklist assesses each abuse symptom with multiple items, resulting in a summary score that ranges from 0 – 4 indicating the number of DSM-IV abuse symptoms endorsed. Dependence symptoms were assessed by a 7-item checklist corresponding to DSM-IV criteria (Knight et al., 2002). These have been used previously with college students and have expected associations with alcohol-related variables (Knight et al., 2002).

Affective lability was a latent variable assessed by three indicators derived from subscales of the Affective Lability Scale – Short form (Oliver & Simons, 2004). Items are rated on 4-point scales (1) Very Undescriptive - (4) Very Descriptive; sample item: "I switch back and forth between being extremely energetic and having so little energy that it's a huge effort just to get where I am going." The three subscales assess affective lability in respect to depression/elation (8 items, α = .87), anxiety/depression (5 items, α = .86) and anger (5 items, α = .83).

Poor control was a latent variable assessed by three indicators. First, seven items from the Eysenck impulsivity scale I7 (Eysenck, Pearson, Easting, & Allsopp, 1985) assessing difficulty in controlling behavior (α = .75). Sample item; "Do you generally do and say things without stopping to think". In addition, two scales were derived from a measure of self-regulation (Kendall & Williams, 1982); impatience (three items, e.g., "I have to have everything right away," α = .52) and distractibility (6 items, e.g., "I like to switch from one thing to another," α = .81). Items are rated on 5-point scales (1) = Not At All True Of Me, (5) = Very True Of Me. Previous studies indicate that these measures of impulsivity, impatience, and distractibility form a replicable subscale structure and load significantly on a factor of poor control (Wills et al., 2001; Wills et al., 2002; Wills & Stoolmiller, 2002). High scores indicate poor behavioral control.

Social norms was a latent variable assessed by three indicators: (a) Number of friends who drink alcohol (7-point anchored rating scale [0] none – [6] all); (b) Friends' attitude toward participant using alcohol once a week or more (9-point anchored rating scale [1] Strongly disapprove – [9] Strongly approve); (c) Friends' attitude toward participant getting drunk (9-point anchored rating scale [1] Strongly disapprove – [9] Strongly approve).

Alcohol use utility was a latent variable defined by five indicators. Personal strivings are "goals that lie directly behind individuals' behavioral choices" (Emmons & King, 1988), and are relatively stable in college students (Emmons, 1989). In the assessment, the participant first lists 10 personal strivings, each representing "an objective you are typically trying to accomplish." The remainder of the assessment focuses on the five strivings that the participant identifies as most descriptive. The five strivings were entered into a matrix that included five columns that represent levels of alcohol use (1) abstinence - (5) most every day. The participant rates the extent to which each level of use would help or hinder the attainment of each personal striving using a 5-point scale (−2= very harmful effect, +2 = very helpful effect). An alcohol use utility score was created for each personal striving (reverse scoring the abstinence column) and these five scores were indicators of the latent alcohol use utility variable. The resulting utility score reflects both individual differences in types of valued goals as well as individual differences in perceived utility of substance use for goal attainment (Simons & Carey, 2003; Simons, Christopher, Oliver, & Stanage, 2006).

Family history of alcohol problems was assessed by 9-item versions of the Short Michigan Alcohol Screening Test (SMAST) that have been adapted to assess participants' biological father's (F-SMAST) and mother's (M-SMAST) alcohol abuse (Crews & Sher, 1992). The correlation between the 9-item version and parent SMAST scores are .73 (F-SMAST) and .76 (M-SMAST). These scales have demonstrated good reliability and validity in college student samples, and the recommended cut score of 3 was used (Crews & Sher, 1992). Alcohol problems for participants' maternal and paternal grandparents were assessed by four single-item questions (e.g., Do you think your paternal grandfather (i.e., your biological father's father) has or had problems because of drinking?). Single-item assessments of college students' parent's alcoholism have demonstrated reasonably good psychometrics in regard to sensitivity, specificity, and test-retest reliability (Crews & Sher, 1992). The family history density (FHD) method was used to code family history. This method is recommended when both men and women are being assessed (Stoltenberg, Mudd, Blow, & Hill, 1998). FHD is based upon degree of family relatedness including both parents and all four grandparents. Relatives without history of alcohol problems receive a score of zero. Each parent with evidence of alcohol problems contributes 0.5 and each grandparent with evidence of alcohol problems contributes 0.25 to the total score, which can range from 0-2.

Analysis Plan

Structural equation models were estimated using Mplus 5.1 (Muthén & Muthén, 2008) with Full Information Maximum Likelihood (FIML) estimation, which models the complete dataset allowing the inclusion of cases with missing data. Missing data are assumed to be missing at random (MAR) but not necessarily missing completely at random (MCAR; Enders & Bandalos, 2001). Covariances among all exogenous variables were included in the model. All variables were measured at T1 and alcohol consumption and problems were measured at both T1 and T2. Correlations between er-

ror variances for paired T1 and T2 indicators were specified in the model. We tested two measurement models, one for the continuous predictors and one to examine the factor structure of the binary DSM criteria. We used maximum likelihood estimation for the former and the WLSMV estimator (Muthén & Muthén, 2008) for the latter. We then combined these into an overall measurement model and estimated it using WLSMV. Incremental fit indexes (e.g., CFI) greater than or equal to .95 represent acceptable fit (Hu & Bentler, 1999). SRMR values less than or equal to .08 and RMSEA values less than or equal to .06 represent acceptable fit (Hu & Bentler, 1999). For modeling categorical data with WLSMV, CFI ≥ .96 and WRMR of approximately 1.0 indicate good fit (Yu, 2002). To our knowledge, there are not clear guidelines for fit indices for models with a combination of continuous and categorical data.

We tested two structural models. All participants who reported alcohol use during the follow-up period were included (N = 2084). The first model examined the hypothesized direct effects of affective lability and poor control, and indirect effects of alcohol use utility, family history, and social norms. Gender was included as a covariate. The second model was a multigroup analysis that examined the moderating effects of poor control. Poor control was removed from the previous model and used as a grouping variable, comparing effects across the upper (n = 1042) and lower (n = 1042) halves. In the multigroup analysis, factor loadings were constrained to be equal across groups and the fit of this model was compared to the fit of an unconstrained model. All observed variable error variances and covariances, the variances and covariances of exogenous latent variables, and the disturbance term variances and covariances of endogenous latent variables were freely estimated across groups

Results

Attrition Analyses

Attrition analyses indicated that participants with matched T2 assessments (n = 1851) reported slightly less alcohol use and problems. For example, they tended to report fewer drinks per week on the DDQ-M (M = 13.14, SD =13.73 vs. M = 15.92, SD =16.37), lower scores on the abuse symptoms checklist (M = 0.70, SD =0.92 vs. M = 0.89, SD =1.04), and lower scores on the dependence symptom checklist (M = 0.78, SD =1.23 vs. M = 0.96, SD =1.34). A logistic regression analysis indicated that all study variables accounted for approximately 3% of the variance in attrition, with LR $\chi 2$ (30) = 68.36, $p < .001$, Cragg-Uhler R^2 = .03. This magnitude of differential attrition is comparable to that found in other longitudinal studies conducted in this age range (e.g., Newcomb & Bentler, 1988) and the longitudinal sample was similar in basic characteristics to the baseline sample.

Descriptive Statistics

At T2, approximately 92% of the total sample reported drinking at least once in the past six months. Table 1 presents summary statistics for the analytic sample (i.e., reported drinking at least once in the past six months). At T2, approximately 52% of drinkers (47% of the full T2 sample) reported one or more abuse symptoms, 47% reported one or more abuse symptoms and less than

three dependence symptoms, and 11% reported 3 or more dependence symptoms in the past 6-months. Abuse and dependence symptoms were moderately correlated at T1 (r = .48, p < .001) and T2 (r = .50, p < .001). Participants in the analytic sample reported drinking a mean of 14.27 (SD = 13.33) drinks per week and 7-8 drinks per drinking day (rating scale M = 3.84, SD = 2.36) at T2. The sample is thus characterized by rates of drinking and related problems that are relatively high compared to national samples and congruent with the elevated rates of heavy drinking among young adults in this region (Knight et al., 2002; Wright & Sathe, 2006).

Table 1

Descriptive Statistics for the Sample of Drinkers

	M	SD	Skew	Range
Alcohol use utility 1	−0.50	0.68	0.44	−2 - 2
Alcohol use utility 2	−0.50	0.74	0.45	−2 – 2
Alcohol use utility 3	−0.47	0.74	0.39	−2 – 2
Alcohol use utility 4	−0.47	0.75	0.46	−2 – 2
Alcohol use utility 5	−0.47	0.73	0.44	−2 – 2
Social norms 1	4.47	1.16	−1.49	0 – 6
Social norms 2	5.68	2.31	−0.32	1 - 9
Social norms 3	6.14	2.25	−0.64	1 - 9
Family history	0.21	0.33	1.88	0 - 2
Lability (anx./dep.)	8.75	3.62	0.86	5 – 20
Lability (dep./ela.)	16.70	5.34	0.28	8 – 32
Lability (anger)	7.98	3.25	1.25	5 – 20
Eysenck – I7 items	2.68	2.13	0.42	0 – 7
Impatience	5.95	2.13	0.81	3 – 15
Distractability	15.18	4.86	0.34	6 – 30
Weekly drinks (T1)	14.77	14.25	1.87	0 - 100
Alcohol freq. (T1)	3.06	1.38	−0.51	0 - 8
Drinks/DD (T1)	3.98	2.71	0.81	0 -10
DSM-Abuse (T1)	0.80	0.96	1.08	0 - 4
DSM-Dep. (T1)	0.88	1.28	1.70	0 – 7
Weekly drinks (T2)	14.27	13.33	2.09	0 - 121
Alcohol freq. (T2)	3.25	1.20	−0.30	1 - 8
Drinks/DD (T2)	3.84	2.36	1.02	0 - 10
DSM-Abuse (T2)	0.84	0.99	1.04	0 - 4
DSM-Dep. (T2)	0.81	1.25	1.91	0 – 7

Note. T1 *N*'s 2043 – 2084, T2 *N*'s 1659 – 1664, 64% women. Anx. = anxiety, dep = depression, ela. = elation. See measures section for description of Social norms 1-3. DD = drinking day. DSM-Dep. = DSM dependence checklist, DSM-Abuse= DSM Abuse checklist.

Alcohol Problem Analyses

Measurement model for abuse and dependence symptoms The hypothesized 2-factor measurement model (based on 4 items for abuse and 7 items for dependence) provided good fit to the data, with $\chi 2$ (116, N =2,080) = 222.88, $p < .001$; RMSEA = 0.021; CFI = .98; and WRMR = 1.028. Standardized factor loadings ranged from .45 - .81 (p's < .001). A single-factor alcohol problem model had adequate fit ($\chi 2$ (118, N =2,080) = 252.70, $p < .001$; RMSEA = 0.023; CFI = .97; and WRMR = 1.108). However, the two-factor model had significantly better fit than the one-factor model ($\Delta\chi 2$ (5, N =2,080) = 35.32, $p < .0001$). The chi-square difference and degrees of freedom are estimated by Mplus for comparison of models estimated by WLSMV. Abuse and dependence latent factors were highly correlated (r = .87 at T1 and r = .85 at T2).

Measurement model for continuous predictors The hypothesized measurement model provided a good fit to the data, with $\chi 2$ (152, N =2,084) = 831.98, $p < .001$; RMSEA = 0.046; CFI = .96; and SRMR = 0.038. Standardized factor loadings ranged from .60 - .89 (p's < .001).

Measurement model respecification Error term covariances with modification indices greater than 50 were sequentially freed and the model re-estimated (there were no modification indices greater than 20 in the problems analysis). This resulted in two correlated errors (distractability with ALS-depression/elation, social norms 1 with T1 alcohol use frequency). For the final measurement model, $\chi 2$ (150, N =2,084) = 645.38, $p < .001$; RMSEA = 0.040; CFI = .97; and SRMR = 0.036. Standardized factor loadings ranged from .61 - .89 (p's < .001). Table 2 presents the correlations between the constructs in the measurement model.

Table 2

Correlations among Latent Constructs in the Sample of Drinkers

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Alcohol Use Utility	1.00									
2. Social Norms	.43	1.00								
3. Affective Lability	.03^{ns}	.02^{ns}	1.00							
4. Poor Control	.21	.16	.61	1.00						
5. T1 Consumption	.52	.64	.00^{ns}	.22	1.00					
6. T2 Consumption	.46	.57	.01^{ns}	.18	.82	1.00				
7. T1 Abuse	.40	.60	.19	.45	.86	.70	1.00			
8. T2 Abuse	.38	.55	.18	.39	.75	.82	.69	1.00		
9. T1 Dependence	.33	.49	.29	.42	.71	.58	.87	.74	1.00	
10. T2 Dependence	.25	.36	.32	.38	.52	.68	.65	.85	.71	1.00

Note. N = 2084. ^{ns} = not significant. All other correlations significant at $p < .001$.

Full measurement model The measurement model combining all latent constructs fit well $\chi 2$ (258, N =2,084) = 875.10, $p < .001$; RMSEA = 0.034; CFI = .94; and WRMR = 1.35.

Structural model The hypothesized structural model fit the data well, with $\chi 2$ (278, N =2,084) = 977.70, $p < .001$; RMSEA = 0.035; CFI = .93; and WRMR = 1.40.

Model respecification We considered adding non-hypothesized structural paths to the model based on the modification indices and substantive interpretation. A path from T1 dependence symptoms to T2 alcohol consumption was added. In the initial model, neither affective lability or poor control was prospectively associated with T2 abuse. We thus dropped the weaker and non-hypothesized lability path. For the final model, $\chi 2$ (278, N =2,084) = 973.22, $p < .001$; RMSEA = 0.035; CFI = .93; and WRMR = 1.39.

The model is presented in Figure 2. As shown, the analysis tests the hypothesized structural associations at T1, models the stability of the criterion variables over the follow-up period, and then tests prospective associations of the T1 predictors on T2 consumption, abuse and dependence symptoms. It is thus a test of prospective associations that partials out the cross-sectional associa-

tions and explicitly models the stability of the criterion variables as well as the errors of their indicators. Abuse and dependence symptoms are modeled with a correlated disturbance term to account for their residual covariation.

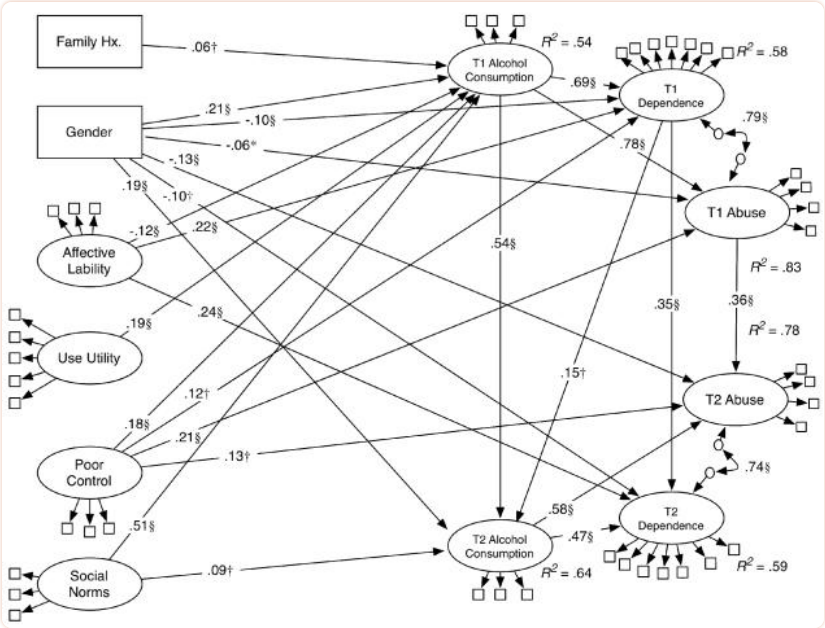

Figure 2

Structural model for the full alcohol use sample (N = 2084). All values are standardized coefficients. Gender is coded 1 = Men, 0 = Women. *p < .05, †p < .01, §p < .001. Error variances between paired T1 and T2 indicators were allowed to covary. Nonsignificant paths from family history, lability, poor control, and use utility to T2 consumption, poor control to T2 dependence, lability to T1 abuse are omitted for clarity but are included in model estimation.

As hypothesized, family history of alcohol problems, alcohol use utility, and social norms were each associated with T1 alcohol consumption but only indirectly associated with T1 abuse and dependence symptoms via this association. In contrast, affective lability exhibited cross-sectional associations with dependence symptoms that did not go through level of use, but was unrelated to abuse symptoms at T1. Poor control exhibited a significant cross-sectional association with both dependence and abuse symptoms. These effects represent significant associations between these constructs after partialing out the strong effect of consumption on both abuse and dependence symptoms as well as the common variance with the other exogenous variables. The model thus shows unique cross-sectional associations between both affective lability and poor control and the criterion variables, abuse and dependence symptoms, independent of the rate of alcohol consumption or the other covariates.

Furthermore, prospective paths indicated that affective lability had a direct effect on T2 dependence symptoms and poor control had a direct effect on T2 abuse symptoms. These effects are significant after partialing out all of the cross-sectional associations depicted in the model. Male

gender, social norms, and T1 dependence symptoms exhibited significant positive prospective associations with level of alcohol consumption at T2. The significant effects of the other variables on T2 consumption were all indirect via cross-sectional associations with T1 consumption. Total and indirect effects of the exogenous variables on T2 consumption, abuse, and dependence are presented in Table 3.

Table 3

Standardized Indirect and Total Effects in Predicting T2 Alcohol Consumption, Abuse, and Dependence

	T2 Consumption		T2 abuse		T2 Dependence	
	Indirect	**Total**	**Indirect**	**Total**	**Indirect**	**Total**
1. Gender	.13†	.31†	.14†	.09*	.11†	.06
2. Alcohol use utility	.12†	.16†	.13†	.15†	.10†	.12†
3. Social norms	.33†	.42†	.35†	.40†	.28†	.32†
4. Family history	.04*	.05	.04*	.05*	.03*	.04*
5. Affective lability	−.08	−.04	−.08	−.03	−.07	.26†
6. Poor control	.11*	.10*	.12*	.31†	.10*	.12

Note: *N* = 2084. Gender is coded 0 = Female, 1 = Male. Indirect effects are all of the indirect effects via T1 alcohol consumption. Bias-corrected bootstrapped confidence intervals were used to determine the significance of effects (MacKinnon, Lockwood, & Williams, 2004). Superscripts denote significant effects based on

*= 95% confidence interval or

†= 99% confidence interval.

In respect to differential direct predictors of alcohol abuse and dependence symptoms, affective lability was significantly associated with T2 dependence but not abuse symptoms. In contrast, poor control exhibited significant direct prospective associations with abuse but not dependence. At T1, affective lability was associated with dependence symptoms but not abuse symptoms, while poor control was associated with both abuse and dependence symptoms at T1.

Multigroup Analysis

Measurement model

The hypothesized measurement model with factor loadings constrained to be equal across groups (defined by median split on poor control) provided a good fit to the data, with $\chi 2$ (320, N =2084) = 677.24, $p < .001$; RMSEA = 0.033; CFI = .96; and WRMR = 1.59.[1]

Structural model We first tested the hypothesized model with paths unconstrained across groups $\chi 2$ (345, N =2084) = 723.18, $p < .001$; RMSEA = 0.032; CFI = .96; WRMR = 1.60. We estimated a model with four specific paths constrained to test the hypothesized moderating effects of poor control on associations between T1 consumption and T1 abuse, T2 consumption and T2 abuse, and T1 lability and T1 and T2 abuse symptoms. These constraints significantly worsened the model fit, $\Delta\chi 2$ (4, N = 2084) = 13.33, p = .0098. As hypothesized, in each case the effects were stronger in the high poor control group. We then tested the four hypothesized moderating effects of poor control on the associations with dependence symptoms (i.e., T1 lability to T1 and T2 dependence symptoms, T1 consumption to T1 dependence, T2 consumption to T2 dependence). These constraints did not significantly worsen the model fit (compared to the initial unconstrained model) $\Delta\chi 2$ (4, N = 2084) = 3.56, p = .4694. To create a more parsimonious model, we constrained all structural paths aside from the four moderation effects identified above. This partially constrained model did not fit significantly worse than the fully unconstrained model $\Delta\chi 2$ (15, N = 2084) = 17.23, p = .305; Final model $\chi 2$ (338, N = 2084) = 694.38, $p < .001$; RMSEA = 0.032; CFI = .96; WRMR = 1.62.[2] See Figure 3.

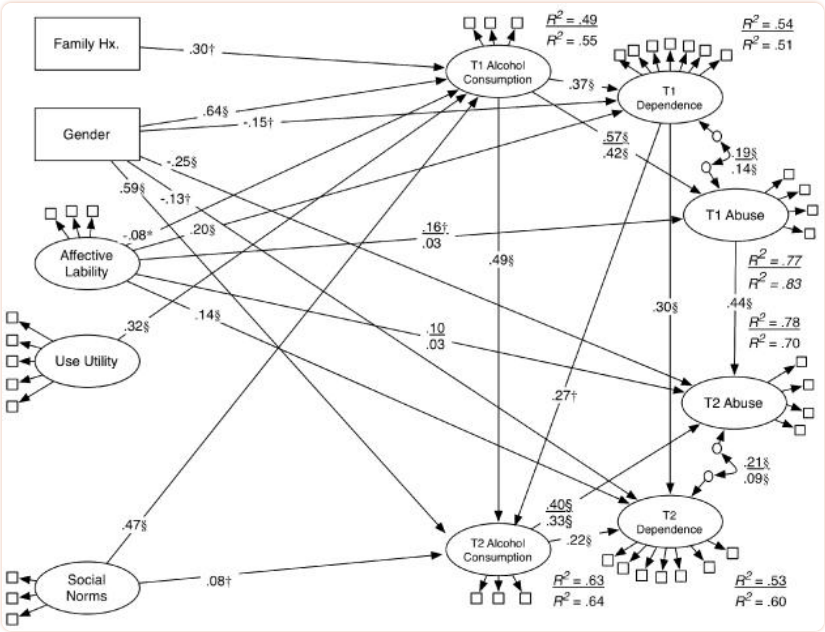

Figure 3

Structural model for the multigroup analysis (N = 2084). All values are unstandardized coefficients. Gender is coded 1 = Men, 0 = Female. *$p < .05$, †$p < .01$, §$p < .001$. Error variances between paired T1 and T2 indicators were allowed to covary. Path coefficients that were significantly different across groups are depicted with the high poor control group above, and the low poor control group below the horizontal lines. Nonsignificant paths from lability, use utility, and family history to T2 consumption, and gender to T1 abuse are omitted in the figure for clarity but are included in model estimation.

Discussion

The purpose of this prospective study was to test a theoretical model that generated several hypotheses about the etiology of alcohol abuse and dependence symptoms. The results were consistent with both main-effect and moderation hypotheses in almost all respects. Alcohol abuse and dependence symptoms exhibited a two-factor structure with both common and unique antecedents. In addition, the identification of predicted antecedents for abuse and dependence provided support for the proposed etiological processes manifesting in meaningfully distinct classes of alcohol-related outcomes.

Associations between Affective Lability and Dependence Symptoms

In the main-effect model, affective lability exhibited significant direct cross-sectional and prospective associations with dependence but not abuse and this did not occur through level of use. This finding lends support for the proposed theoretical mechanism based on the affective processing model of negative reinforcement (Baker et al., 2004). We propose that the speed, frequency, and intensity of affective shifts experienced by individuals with labile affect places them at increased risk for the process of the development of drug addiction. Thus the dynamics of affect regulation become central to addiction etiology, contrasting with earlier formulations of self-medication models that posit that negative affective states act simply as aversive stimuli to be coped with (Khantzian, 1985). This perspective is consistent with recent research on smoking, which indicated that negative mood variability predicted escalation in smoking among adolescents (Weinstein, Mermelstein, Shiffman, & Flay, 2008). Importantly, Weinstein and colleagues demonstrated that negative mood variability predicted escalation independent of effects of mean negative mood level. Dvorak and Simons (2008) similarly demonstrated that lability, but not negative affect, differentiated daily from occasional smokers. Affect lability is significantly associated with negative affectivity. Future research jointly examining the role of mean levels of negative affect as well as affect variability in predicting substance dependence and accounting for associations between internalizing disorders and substance dependence is warranted.

Consistent with previous research, affective lability did not correlate significantly with level of alcohol consumption and was in fact negatively associated with T1 consumption in the structural model, indicating a suppression effect (Simons & Carey, 2006). It did, however, have direct effects to dependence symptoms at T1 and T2 when consumption was controlled. This suggests that labile affect may be specifically associated with the development of a pattern of drinking that is compulsive and characterized by the hallmark symptoms of dependence. The direct unique relation between affective lability and dependence symptoms, combined with the lack of a significant total effect on T2 abuse symptoms, supports its proposed role specifically in the development of alcohol dependence, and the findings emphasize the importance of this pathway to the development of alcohol problems. Affective lability may thus be a marker variable that can help to identify persons who will be at risk for alcohol problems even after they leave the college environment.

Although affective lability plays a potentially important role in the etiology of dependence, it is not necessarily more influential than other variables. For example, social norms, due to its strong relation to T1 consumption, ultimately had a large indirect effect on T2 dependence symptoms be-

cause of the linkage between norms, consumption, and dependence. In this environmental context, social influences can present a significant adverse effect through promoting frequent and unconstrained drinking, which sets up a pathway to dependence among the more vulnerable individuals in the college population. Thus we would emphasize the implications of social context factors for prevention of alcohol problems among college students. The findings, we think, support programs to address both the subtle influence of normative factors (through correcting erroneous perceptions about the prevalence and acceptability of binge drinking) and situational factors that directly encourage high levels of consumption (e.g., parties with large amounts of alcohol easily available). Programs using motivational interviewing to reduce consumption among current heavy drinkers can also help achieve this goal through counseling at the individual level (Carey, Henson, Carey, & Maisto, 2007; Carey, Scott-Sheldon, Carey, & DeMartini, 2007).

Poor Control and Alcohol Abuse

Main-effect results showed that poor behavioral control had a prospective direct effect on abuse (but not dependence) symptoms, in addition to its relations with baseline drinking and symptomatology. Abuse symptoms include failure to fulfill social responsibilities, reckless hazardous behavior, socially disruptive illegal activity, and interpersonal conflicts such as physical fights (American Psychiatric Association, 2000). The results of the current study demonstrate that poor control contributes to these problems indirectly, through its positive relation to alcohol consumption, and also contributes directly to these problems even after controlling for consumption. Assessment measures typically ask whether these behaviors have occurred “while drinking” or “because of drinking.” Without explicitly modeling the occurrence of these behaviors while sober as well as intoxicated it is difficult to determine the extent to which such behaviors are a symptom of alcohol abuse or due to preexisting personal characteristics correlated with consumption (Neal & Fromme, 2007). Our results are consistent with recent item-response studies that suggest some symptoms of “alcohol problems” may be indexing the existence of another correlated process, in this case, poor behavioral control (Martin et al., 2006). Edwards’ (1974, 1986) concept of plasticity and the biaxial model of dependence and disability (e.g., abuse symptoms) is a useful heuristic for conceptualizing the effects of poor control. Poor control may be conceived of as a pathoplastic agent that affects intoxicated behavior, promoting a range of socially deleterious and risky behavior commonly grouped under the rubric of alcohol abuse.

Moderation Effects of Poor Control

Moderation hypotheses were mostly supported, as poor control interacted with both affective lability and alcohol consumption in the prediction of abuse symptoms. Consistently, high levels of poor control increased the strength of the associations. This may occur because poor control is associated with decreased deliberative cognitive processing and an increased emphasis on immediate salient cues guiding behavior (Carver, 2005; Wills & Dishion, 2004). Alcohol intoxication itself is associated with a decrease in information processing speed and inhibition, and an overall narrowing of cognitive focus (Fillmore, 2003; Fillmore et al., 1998; Steele & Josephs, 1990). Also, the intense emotional arousal characteristic of labile affect is associated with a decrease in deliberative “cool” cognitive processing (Baker et al., 2004; Metcalfe & Mischel, 1999). The “cool” system is posited to be associated with declarative knowledge, slower acting, reflective, and contrib-

utes to the inhibition of prepotent responses (Baker et al., 2004; Lieberman, 2007; Metcalfe & Mischel, 1999). Thus, we believe these interactions represent a similar process whereby preexisting poor control combined with intoxication or affective lability leads to enhanced alcohol-related problems. Moderating effects of poor control may result from an over-reliance on immediate affective and environmental cues and a decreased awareness of alternative behaviors, solutions, and potential consequences (Curtin & Fairchild, 2003; Steele & Josephs, 1990).

Clinical Implications

Heavy drinking among college students is associated with both acute and long-term negative consequences (Jackson et al., 2005), so prevention programs during the college years are clearly warranted (Baer et al., 2002). Though students in college may drink more than their same aged non-college peers, college attendance overall is associated with less risk for alcohol dependence in later life (Harford, Yi, & Hilton, 2006; SAMHSA 2006), so consideration should be given to identifying which students are more at risk for long-term alcohol problems. The results of the present study suggest that affective lability may be a factor that differentiates students who will develop long-standing problems with alcohol dependence from persons who are experiencing a time-limited period of heavy drinking. Both secondary and tertiary interventions may benefit from including components designed to reduce volatile negative affect and poor behavioral control as well as to reduce alcohol consumption. Poor control had multiple direct, indirect, and interactive effects on alcohol outcomes. The direct effects on alcohol abuse suggest that there may be value in conceptualizing such problems as reflecting general deficits in self-control rather than solely a problem of excessive alcohol use. The prospective association between social norms and alcohol consumption as well as the strong cross-sectional associations with both alcohol use utility and social norms are supportive of the emphasis on these or related constructs in college alcohol interventions (e.g., Dimeff et al., 1999). Interventions to foster improved self-control across the lifespan may be beneficial in not only reducing alcohol use but also reducing a range of problematic social behaviors, using either targeted programs for high-risk students or broader general-population approaches (Miller, Leckman, Delaney, & Tinkcom, 1992; Rehm, Kaslow, & Rabin, 1987; Watson & Tharp, 2002).

Structure of Alcohol Use Disorder

The better fit of a 2-factor model to the data, the differential associations with lability and poor control, and the unique prospective association from T1 dependence symptoms to T2 consumption, each support the differentiation of alcohol abuse and dependence symptoms in this population. However, the factors were highly correlated and a parsimonious 1-factor model fit the data adequately. Though our results support the differentiation of abuse and dependence symptoms, this is not to suggest that the current diagnostic categories are optimal, or that the negative consequences indicative of abuse are best conceived of as a mental disorder per se. Continued efforts to refine the conceptualization and diagnosis of alcohol use disorder are clearly warranted (cf. Babor, 2007; Martin et al., 2008; Midanik, Greenfield, & Bond, 2007).

Limitations and Suggestions for Research

Limitations of the present study should be noted. First, the sample for the research was a predominantly White college population with some under-representation of males, and generalization to other populations should be tested. There was slightly greater attrition for heavier drinkers, as is typical in longitudinal studies of alcohol and other drug use (Wills, Walker, & Resko, 2005). Second, the participants were studied in one developmental stage, and the theoretical model used here could be evaluated at earlier and later ages. Third, the follow-up period was relatively short and more extended designs with additional time points suitable for examining trajectories of use and abuse would be beneficial.

In summary, the results showed that alcohol abuse and dependence are correlated constructs with unique antecedents beyond a large shared association with alcohol consumption. The significant role of affective factors in the model suggests further attention to the interrelation of affect and other risk factors for drinking, both in between-person studies and in within-person analyses (Neal & Carey, 2007; Simons & Carey, 2006; Simons et al., 2005b). Further research is suggested to explore the cognitive as well as affective concomitants of alcohol problems, through understanding the explicit and implicit cognitive processes that underlie drinking motivations and decisions, and the role of controlled vs. automatic processes in compulsive behaviors (Baker et al., 2004; Gibbons, Gerrard, Reimer, & Pomery, 2006; Stacy & Wiers, 2006; Wiers et al., 2007).

Moderating effects of poor control on both affective lability and consumption relationships are congruent with a proposed mechanism of decreased deliberative cognitive processing strengthening the associations between these predictors and use-related problems. Studies to integrate cognitive concepts with measures of personality and self-regulation may also help to understand linkages of these areas and implications for alcohol abuse and other addictive behaviors (Cervone, Orom, Artistico, Shadel, & Kassel, 2007; Sussman et al., 2004). Finally, in view of the significant role of social aspects in college alcohol use, detailed research on how drinking is embedded in social networks can help to better understand the role of normative and social factors in drinking behavior (Valente, Gallaher, & Mouttapa, 2004). Research on alcohol consumption and problems can benefit from testing theoretical models that reflect the multidimensional nature of alcohol problems and the multiple processes involved in their development, presentation, and course.

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Footnotes

[1]The initial measurement model with correlated errors across time for all indicators resulted in a non-positive definite matrix for the high poor control group. This was corrected by constraining a correlated error across time on the abuse and dependence factors in each group. Following the recommendations of Hoyle and Smith (1994), we examined the assumption of measurement invariance in factor loadings. We tested a model with factor loadings freely estimated across groups. The unconstrained model did not fit significantly better than the constrained model ($\Delta\chi 2$ (18, N = 2084) = 8.11, p = .98. Chi-square difference and degrees of freedom are estimated by Mplus for WLSMV.

[2]We examined the moderated paths across alternative groupings by conducting a tertile split of the sample based on poor control scores. The tertile analysis ($\Delta\chi 2$ (8, N = 2084) = 17.46, p = .026) indicated that the paths significantly varied across groups. Inspection of the coefficients revealed a pattern basically consistent with the median split analysis. Thus the observed moderation effects are consistent across multiple splits of the data.

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