

Test of a Clinical Model of Drinking and Suicidal Risk

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Background: There are few data on the role of drinking patterns in suicidal thoughts or behavior among alcohol-dependent individuals (ADIs) and meager data on variables that may influence the role of drinking in suicidal thoughts and behavior. This study tested a heuristic model that predicts that drinking promotes suicidal thoughts and behavior, the association is mediated (accounted for) by depressive symptoms, and that anger moderates (increases) the risk associated with intense drinking.

Methods: Data from Project MATCH, a multisite alcohol use disorders treatment trial, were analyzed using structural equation modeling. There were 1,726 participants including 24% women and a mean age of 40.2 ± 11.0 years. Subjects were assessed at baseline and at 3-, 9-, and 15-month follow-up. Two categorical measures (presence/absence) of suicidal ideation (SI) were used that were analyzed in separate models. Predictors of interest were continuous assessments of average drinking intensity (i.e., drinks per drinking day or DDD), drinking frequency (i.e., percent days abstinent or PDA), depression, and anger.

Results: Both DDD and PDA were associated with SI at a statistically significant level, with PDA showing an inverse association. Depression scores served as a partial mediator or a full mediator of the drinking–SI relationship depending on the measure of SI used in the analysis. The models testing anger scores as a moderator fit the data poorly and did not support that anger serves as a moderator of the drinking–SI association.

Conclusions: Greater drinking intensity and drinking frequency predict SI among ADIs and depression serves as a mediator of these associations, but anger does not appear to serve as a moderator. Further research is required to clarify whether depression serves as a partial or full mediator and to see whether the results herein extend to suicidal behavior (i.e., suicide attempt, suicide).

Key Words: Suicide, Alcohol Dependence, Drinking, Depression.

ALCOHOL-DEPENDENT INDIVIDUALS (ADIs) are at elevated risk for suicide attempts (Kessler et al., 1999) and suicide (Yoshimasu and Miyashita, 2008), and more severe clinical populations are especially vulnerable (Wilcox et al., 2004). Suicidal ideation (SI) is critical to address in the prevention of suicidal behavior among treated ADIs. First, clinical suicide risk assessment protocols (American Association of Suicidology, 2006; Center for Substance Abuse Treatment, 2009) emphasize the need to screen for SI, assess SI (suicide plan? intent to act?), and track SI in the management of suicide risk. Second, SI is 1 of only 2 preconditions for suicidal behavior, the other being the availability of a method. Therefore, SI provides a common pathway through which all patients pass prior to carrying out a suicidal behavior, inspiring research on SI as a pathway to suicidal behavior (Conner et al., 2007; Joe et al., 2006; Kessler et al., 1999; Nock et al., 2009).

Third, SI has consistently been demonstrated to confer prospective risk for suicidal behavior in adult clinical populations (Brown et al., 2000) including studies of alcohol and drug dependence (Britton and Conner, 2010; Darke et al., 2005, 2007; Ilgen et al., 2007).

Drinking and Suicidal Thoughts and Behavior

ADIs are most vulnerable to suicide during active drinking periods (Conner et al., 2000); ADIs with more severe drinking patterns are at higher long-term risk (Roy and Janal, 2007; Roy et al., 1990) and are at higher short-term risk for suicidal behavior compared to less severely drinking ADIs (Cornelius et al., 1996; Murphy et al., 1992). These data lead to the conclusion that “more drinking is worse,” yet a more refined understanding of drinking and suicidal thoughts and behavior will require the use of more specific measures of drinking. Two well-researched and relatively independent measures of drinking are the frequency of drinking and the average intensity of drinking (Babor et al., 1994). An analysis of Project MATCH data (Project MATCH Research Group, 1993) suggests that drinking intensity is more robustly associated with SI than drinking frequency (Conner et al., 2003). As well, an analysis of a large cohort of men followed prospectively for 16 years showed that high drinking intensity, defined in the study as > 2 standard drinks per drinking day (DDD),

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conferred risk for suicide, whereas other measures of drinking including alcohol use frequency did not contribute to risk (Mukamal et al., 2007), although the generalization of these results to ADIs is unclear. Overall, studies on patterns of drinking and suicidal thoughts and behavior are few, but available research suggests that drinking intensity more so than drinking frequency confers risk.

Clinical Model of Drinking and Suicidal Thoughts and Behavior

Beyond identification of the drinking variables (e.g., intensity, frequency) that promote suicidal thoughts and behavior, a comprehensive understanding of the role of drinking in suicidal thoughts and behavior requires the understanding of the interrelationship of drinking with other risk factors, including potential mediating and moderating relationships (Bagge and Sher, 2008). Studies of ADIs have firmly established that drinking promotes depressive symptoms and depressive disorders (Conner et al., 2009) and that depression stands out as a highly prevalent and potent risk factor for suicidal thoughts and behavior (Conner et al., 2003; Murphy et al., 1992; Pless et al., 2002). These findings lead to a hypothesis that drinking promotes depression, which in turn promotes SI (and suicidal behavior), consistent with a conceptualization of mediation (Baron and Kenny, 1986). However, the extent to which depression mediates suicidal thoughts and behavior among ADIs is unknown, with implications for prevention and treatment. If, on the one hand, depression fully mediates the association between drinking and suicidal thoughts and behavior, it would suggest a dominant role of depression in the development of drinking-related risk among ADIs. On the other hand, if depression is a partial mediator, then it would suggest a more complex relationship and the need to explicate other mediators.

Like its counterpart depression in the internalizing spectrum, aggression stands out in the externalizing spectrum for the consistency with which it is shown to confer suicide-related risk in a variety of populations (Brezo et al., 2006; Conner et al., 2001), including clinical samples of ADIs (Koller et al., 2002; Oquendo et al., 2006; Sher et al., 2005; Windle, 2004). Conceptually, anger is a particularly promising aspect of aggression for suicide research studies, because it represents the affective component of aggression (Buss and Perry, 1992), and negative affect is presumed to be a hallmark of an acutely suicidal state (Shneidman, 1985). We hypothesize that the propensity for anger serves to moderate (increase) risk for suicidal thoughts and behavior conferred by drinking. We offer this hypothesis because suicidal behavior is known to occur reactively during intense drinking bouts (Berman et al., 2009; Hufford, 2001), and ADIs with heightened anger are conceptually more vulnerable to this scenario. In addition, anger has been shown to enhance the association between drinking and aggression (Giancola, 2002). Whether these findings generalize to suicidal thoughts or behaviors among ADIs (or other populations) is unknown. Note that

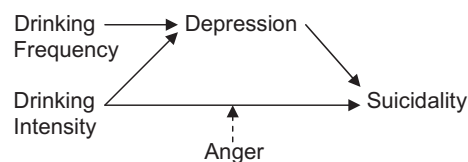


Fig. 1. Clinical model of drinking and suicidality.

the data showing associations of aggression and anger with suicidal thoughts and behavior are based overwhelmingly on general measures of aggression and anger and not on measures of “self-directed” aggression/anger (Brezo et al., 2006; Conner et al., 2001), suggesting that the findings are not circular; in other words, they do not merely reflect a propensity for suicidal thoughts and behavior tapped by assessments of “self-directed” aggression/anger.

Integrating these ideas, we propose a heuristic model of drinking and suicidal thoughts and behavior among treated ADIs that proposes that drinking promotes risk and that the association is mediated by depressive symptoms and it is moderated by anger. The model is based in part on the findings of an early study of suicide attempts among ADIs that described suicidal acts preceded by a chronic process marked by steady drinking and depression as well as suicidal acts that occurred during intense drinking bouts that were triggered by acute interpersonal conflict (Mayfield and Montgomery, 1972). These observations suggest the importance of integrating depression and steady alcohol use (i.e., drinking frequency) as well as intense drinking and anger proneness into a model of suicidal thoughts and behavior among ADIs. They further suggest that anger may serve as a moderator of the relationship between intense drinking in particular and suicidal thoughts and behavior. Figure 1 depicts a conceptual model that incorporates these ideas that depicts direct or mediating paths using solid lines and moderating paths using broken lines. Although research studies have consistently implicated drinking, depression, and anger in suicidal thoughts and behavior among ADIs as we have discussed, to our knowledge, no suicide research study has examined the interrelationship of these variables in an adult alcohol-dependent sample.

Purpose

The study’s goals are addressed through a reanalysis of Project MATCH data (Project MATCH Research Group, 1993). MATCH assessed SI (but not suicidal behavior) prospectively, allowing for the longitudinal examination of SI. The first goal of the current study is to test depression as a mediator of the drinking–SI association, with a focus on determining whether there is partial or full mediation of drinking frequency and drinking intensity, respectively. The second goal is to test anger as a moderator of the relationship between these drinking variables and SI, and we hypothesized that anger moderates the association between drinking

intensity and SI. Although an earlier analysis of drinking and SI using the MATCH sample examined drinking and SI (Conner et al., 2003), the study did not consider the mediating role of depression or the moderating role of anger.

MATERIALS AND METHODS

Procedure

Project MATCH (Project MATCH Research Group, 1993) was a large, multisite, randomized clinical trial. Substance abuse patients were eligible if they were ages 18 and older, had a primary alcohol use disorder, read English at a sixth grade level of higher, and did not have co-occurring intravenous drug use, drug dependence (except cannabis), psychosis, organic impairment, or acute suicide risk. Participants entered the study through inpatient rehabilitation programs, representing the aftercare arm, or came directly to outpatient treatment, representing the outpatient arm. They were randomly assigned to 1 of 3 treatments that were provided on an outpatient basis over 3 months: cognitive behavioral treatment, motivational enhancement, and 12-step facilitation. We refer to the sample as ADIs because 96% of participants met DSM-III-R criteria for alcohol dependence with or without alcohol abuse (American Psychiatric Association, 1987), with the remaining meeting criteria for alcohol abuse only. We analyzed the Project MATCH Public Use dataset that includes a pretreatment interview (baseline) and 3 reassessments at 3-, 9-, and 15 months. There are 1,726 participants including 24% women and a mean age of 40.2 ± 11.0 years.

Measures

Suicidal Ideation. SI is the outcome in all analyses. We derived 2 measures of SI to provide the ability to examine the consistency of results. The Beck Depression Inventory (BDI; Beck et al., 1961) item #9 was used to create a categorical assessment of SI ("I have thoughts of killing myself but I would not carry them out" or "I would like to kill myself" or "I would kill myself if I had the chance") and no SI ("I don't have any thoughts of killing myself"). This item, that we refer to as BDI-SI, has been used to assess SI in several secondary analysis studies and is correlated with the detailed, multi-item Beck Scale for Suicide Ideation (Beck and Steer, 1991; Beck et al., 1997), supporting validity.

We also assessed SI using a modified item from the Addiction Severity Index (ASI; McLellan et al., 1980) that asks "During the past 30 days, have you had a period (even during a time when you were using drugs/alcohol) in which you experienced serious thoughts of suicide?" Responses were coded yes or no. We refer to this item as ASI-SI. Supporting validity, an SI item taken from the ASI has been shown to predict suicide attempts in treated substance abusers (Britton and Conner, 2010).

Drinking. We calculated average DDD and percent days abstinent (PDA) to quantify drinking intensity and frequency, respectively (Babor et al., 1994). DDD and PDA were derived from data collected on the Form 90, a validated retrospective interview of daily alcohol use (Miller and Del Boca, 1994). Baseline drinking was based on a summary assessment of DDD and PDA in the 3 months prior to study entry. DDD and PDA for each of the follow-up assessments were calculated using the preceding 3 months of drinking data. We derived square-root transformed measures of DDD and arcsin-transformed measures of PDA to stabilize the variance as recommended (Babor et al., 1994).

Depressive Symptoms. The BDI (Beck et al., 1961) provided a continuous measure of depression severity. We removed the suicide item from the BDI scores to create a 20-item measure.

Anger. Total scores on the 44-item State-Trait Anger Expression Inventory provided a validated, summary measure of general anger (STAXI; Spielberger, 1996). STAXI total scores are widely used to assess anger including in research of ADIs (Waldron et al., 2001; Witkiewitz and Villaruel, 2009).

Covariates. All analyses are adjusted for age, sex, race (white, nonwhite), treatment assignment (cognitive behavioral treatment, motivational enhancement, and 12-step facilitation), and study arm (aftercare, outpatient). In addition, the analyses of the DDD-SI association covary baseline PDA, and the analyses of the PDA-SI association covary baseline DDD.

Analyses

General Description. The analyses were performed using a constrained longitudinal structural equation model (SEM) in MPlus (Muthen and Muthen, 2006). We assumed multi-normality of the error terms. We formed latent measures of DDD such that a latent construct DDD_j ($j = 3, 9, 15$) at each follow-up assessment was formed from the respective prior 3 months of DDD variables using confirmatory factor analyses (Roberts, 1999). For example, DDD_3 (at 3 months) was formed from DDD month 1, DDD month 2, and DDD month 3. A comparable latent construct was formed for the PDA_j ($j = 3, 9, 15$) variables. The factor variance was fixed at one for all of the latent constructs. We generated standardized estimates for all predictors in the models as follows: covariate X with raw estimate β and standard deviation = $SD(X)$, with standard deviation of the response $Y = SD(Y)$, was computed as

$$\beta_{\text{standardized}} = \frac{\beta \times SD(Y)}{SD(X)}$$

so that the standard error of $\beta_{\text{standardized}}$ is close to zero.

Each 3-month time period represented a time point (3, 9, and 15 months). If any covariate or response for a model at a given time point was missing, the subject was omitted from an analysis at that particular time point. The final estimates used in the models are constrained to be the same at each time point. Owing to the SEM structure of the models, multiple equations were run simultaneously. Therefore, a subject could be missing from 1 equation, but observed in another. To describe missing data (through counts of observations), we focus on the model with the response (outcome), either BDI-SI or ASI-SI, as this is the most informative. Model fits were assessed using the chi-square test of model fit, comparative fit index, CFI (Bentler, 1990), Tucker-Lewis index, TLI (Tucker and Lewis, 1973), and root mean square error of approximation, RMSEA (Browne and Cudeck, 1993).

Specific Models. We assessed the direct associations between each drinking variable (DDD, PDA) with each outcome (BDI-SI, ASI-SI) in separate models, after adjusting for the covariates. The analyses examining the mediating role of depression on the drinking variables were performed for 4 causal structures: (i) $DDD \rightarrow BDI \rightarrow BDI-SI$, (ii) $DDD \rightarrow BDI \rightarrow ASI-SI$, (iii) $PDA \rightarrow BDI \rightarrow BDI-SI$, and (iv) $PDA \rightarrow BDI \rightarrow ASI-SI$. First, a path diagram was drawn to clarify the relationship. Three mediation models were run for each of the 3-month periods, under the constraint that all respective parameters should have the same estimate. Four models examining the moderating role of anger on the drinking variables were performed as follows: (i) $\text{logit}(BDI-SI) = DDD + \text{Anger} + DDD * \text{Anger}$, (ii) $\text{logit}(ASI-SI) = DDD + \text{Anger} + DDD * \text{Anger}$, (iii) $\text{logit}(BDI-SI) = PDA + \text{Anger} + PDA * \text{Anger}$, and (iv) $\text{logit}(ASI-SI) = PDA + \text{Anger} + PDA * \text{Anger}$. For these models, latent constructs of DDD and PDA were formed as previously described, with the factor variance fixed to one and under the

Table 1. Descriptive Data on Suicidal Ideation (SI) and Demographic Characteristics at Baseline Assessment

SI status	Male (%)	Female (%)	Age (mean ± SD)	White (%)	Nonwhite (%)
BDI-SI results					
Present	10.5	13.0	38.6 ± 10.3	11.8	9.3
Absent	89.5	87.0	40.5 ± 11.0	88.2	90.7
ASI-SI results					
Present	9.9	14.6	39.3 ± 10.6	11.3	9.9
Absent	89.1	85.4	40.4 ± 11.0	88.7	90.1

BDI, Beck Depression Inventory; ASI, Addiction Severity Index.

constraint that all the parameters would have the same estimates for all 3 time points (consisting of each 3- month period). Anger was observed at each assessment time and was also included under the constraint that all the parameters would have the same estimates for all 3 time points.

Table 2. Descriptive Data on SI and Predictors of Interest

Measure	Baseline	3 months	9 months	15 months
BDI-SI item (%)	11.1%	9.3%	11.7%	13.8%
ASI-SI item (%)	11.0%	5.2%	3.9%	4.7%
DDD (Mean, SD)	16.6 (10.6)	4.4 (7.1)	4.5 (7.1)	4.6 (7.0)
PDA (Mean, SD)	0.31 (0.30)	0.80 (0.31)	0.79 (0.32)	0.78 (0.32)
BDI (Mean, SD)	10.1 (8.1)	7.5 (7.7)	7.7 (7.8)	7.8 (8.2)
STAXI (Mean, SD)	30.1 (7.5)	27.7 (7.1)	27.0 (7.2)	26.4 (7.2)

DDD and PDA raw scores are shown here, but they are transformed (log, arcsine) in analyses.

BDI-SI item, Suicidal ideation (SI) based on Beck Depression Inventory (BDI) suicide item; ASI-SI item, SI based on Addiction Severity Index (ASI) suicide item; DDD, drinks per drinking day; PDA, percent days abstinent; BDI, BDI score minus suicide item; STAXI, State-Trait Anger Expression Inventory score.

RESULTS

Descriptive Data

Of 1,726 participants, BDI-SI data are available at baseline for 96.3% of the sample, at 3 months 90.0%, at 9 months 87.4%, and at 15 months 89.2%. ASI-SI data are available at baseline for 100% of the sample, at 3 months 91.5%, at 9 months 89.2%, and at 15 months 90.1%. Demographic characteristics of participants stratified by SI status (present or absent) at baseline assessment are presented in Table 1. Using both outcomes (BDI-SI, ASI-SI), participants with SI are more likely to be female, younger, and white ($p < 0.05$). The percentage of subjects with SI at each assessment is shown in Table 2 (denominator is based on subjects with available data on SI at a given assessment). These measures show a similar prevalence of SI at baseline, and the prevalence of SI at follow-ups is higher based on the BDI-SI than the ASI-SI. Descriptive data on the predictors of interest (DDD, etc.) at each wave are also shown in Table 1.

DDD Analyses

The basic SEM models (i.e., that do not consider mediation or moderation) that examine DDD as a predictor of SI over follow-up are shown in Fig. 2. Both models adequately fit the data, with model fit information provided in the figure. In the models, the standardized coefficients between DDD scores (log transformed) and SI were positive and statistically significant ($p < 0.001$), supporting the association between DDD and SI. In the models, baseline DDD is also predictive of DDD over follow-up ($p < 0.001$), and baseline SI is predictive of SI over follow-up ($p < 0.001$). The BDI-SI models

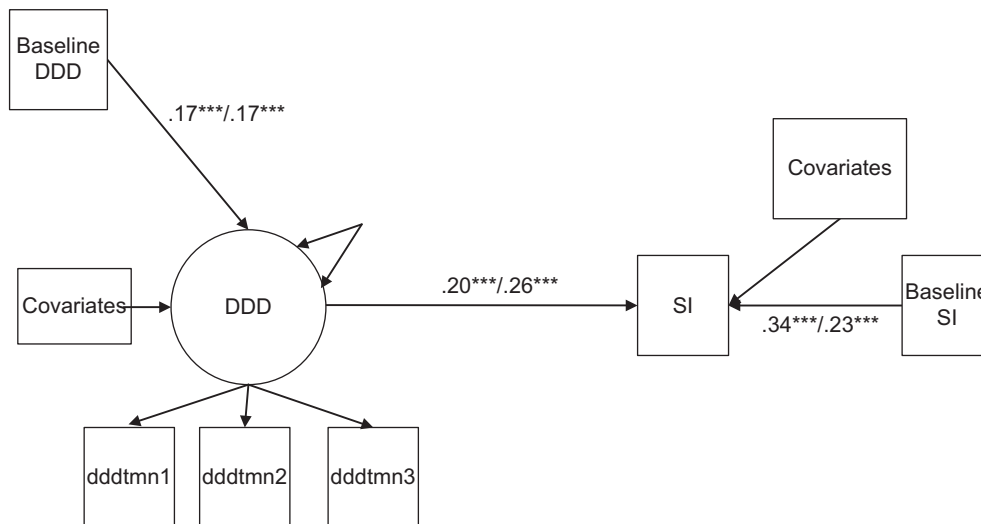


Fig. 2. Constrained longitudinal logistic structural equation models examining drinks per drinking day and suicidal ideation (SI). Standardized estimates of the coefficients for the BDI-SI outcome are presented before the slash; estimates for the ASI-SI are presented after the slash. Covariates include gender, age, race, treatment assignment, study arm and baseline percent days abstinent. χ^2_{BDI} (47) = 189.113, $p \leq 0.001$; comparative fit index (CFI) = 0.924; Tucker–Lewis index (TLI) = 0.906; root mean square error of approximation (RMSEA) = 0.044. χ^2_{ASI} (47) = 129.157, $p \leq 0.001$; CFI = 0.952; TLI = 0.941; RMSEA = 0.033. *** $p < 0.001$.

are based on 87.1% of the total sample at 3-month follow-up, 84.4% at 9 months, and 85.7% at 15 months. The ASI-SI models are based on 90.9% of the total sample at 3 months, 89.0% at 9 months, and 90.4% at 15 months.

The models that examine depression (BDI score) as a mediator of the DDD–SI association are shown in Fig. 3. Both models adequately fit the data as shown in the figure. In the models, there are statistically significant paths from DDD over follow-up to depression over follow-up ($p < 0.001$) and from depression over follow-up to SI over follow-up ($p < 0.001$). There are somewhat different mediation results when depression is examined as a mediator in the BDI-SI model compared to the ASI-SI model. In the BDI-SI model, the path from DDD to BDI-SI is greatly reduced and is non-significant ($p = 0.209$) compared to the basic model. These results suggest that when depression is added to the model, it acts as a full mediator of the association between DDD and SI. However, in the ASI-SI model, the path from DDD to SI is somewhat reduced compared to the basic model, but nonetheless it remains statistically significant ($p < 0.001$), suggesting that when depression is added to the model, it acts as a partial (but not full) mediator of the association between DDD and SI. The BDI-SI mediation models are based on 86.0% of the total sample at 3-month follow-up, 82.7% at 9 months, and 83.7% at 15 months. The ASI-SI mediation models are based on 87.8% of the total sample at 3 months, 85.1% at 9 months, and 86.3% at 15 months.

We also examined anger (STAXI score) as a moderator of the DDD–SI association by use of M-Plus (models not shown). The interaction term for DDD and anger in the

BDI-SI model (estimate = 0, $p = 0.006$) does not support statistical moderation, because an estimate of “0” in MPlus is not consistent with an interaction effect. Moreover, the statistically significant result in this instance is misleading because of the poor model fit [$\chi^2_{\text{DDD}}(53) = 13272.642, p \leq 0.001$; CFI = 0.07; TLI = -0.211; RMSEA = 0.419].

Similarly, the interaction term for DDD and anger in the ASI-SI model (estimate = 0, $p = 0.432$) analyzed using MPlus does not support statistical moderation, and the model fit is poor [$\chi^2_{\text{DDD}}(54) = 13663.149, p \leq 0.001$; CFI = 0.042; TLI = -0.241; RMSEA = 0.420]. There are a similar number of observations (i.e., similar missing data) in these models compared to the meditational models.

PDA Analyses

The basic SEM models (i.e., that do not consider mediation or moderation) that examine PDA as a predictor of BDI-SI and ASI-SI, respectively, over follow-up are shown in Fig. 4. Both models show adequate fit as shown in the figure. In the models, the coefficients between PDA scores (arc sine transformed) and SI are negative and statistically significant ($p < 0.001$), supporting that PDA is inversely associated with SI. In both models, baseline PDA is also predictive of PDA over follow-up ($p < 0.001$), and baseline SI is predictive of SI over follow-up ($p < 0.001$). These models contain the same number of observations as the basic DDD models.

The models that examine depression (BDI score) as a mediator of the PDA–SI association are shown in Fig. 5. Both models adequately fit the data as shown in the figure. In the

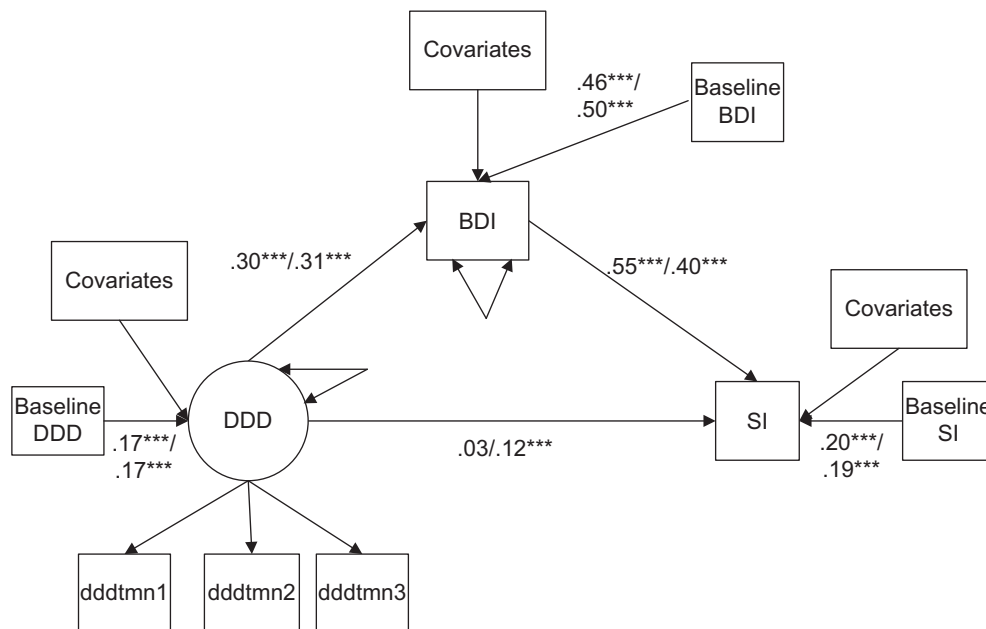


Fig. 3. Constrained longitudinal logistic structural equation models examining Beck Depression Inventory (BDI) scores, with the BDI suicide item removed, as a mediator of the association between drinks per drinking day and suicidal ideation (SI). Standardized estimates of the coefficients for the BDI-SI outcome are presented before the slash; estimates for the ASI-SI are presented after the slash. Covariates include gender, age, race, treatment assignment, study arm and baseline percent days abstinent. $\chi^2_{\text{BDI}}(59) = 218.288, p \leq 0.001$; CFI = 0.947; TLI = 0.933; RMSEA = 0.042. $\chi^2_{\text{ASI}}(65) = 240.790, p \leq 0.001$; CFI = 0.941; TLI = 0.928; RMSEA = 0.042. *** $p \leq 0.01$ / **** $p < 0.001$.

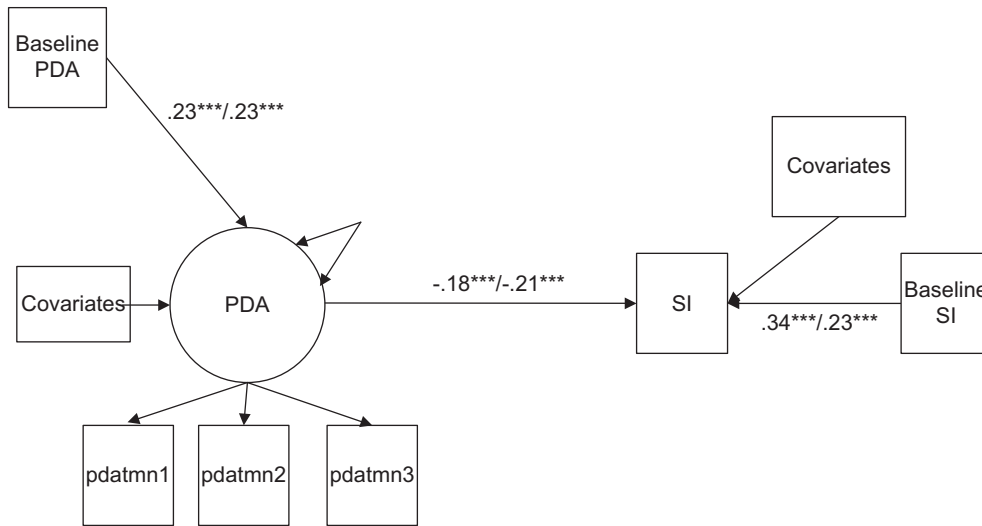


Fig. 4. Constrained longitudinal logistic structural equation models examining percent days abstinent and suicidal ideation (SI). Standardized estimates of the coefficients for the BDI-SI outcome are presented before the slash; estimates for the ASI-SI are presented after the slash. Covariates include gender, age, race, treatment assignment, study arm and baseline drinks per drinking day. $\chi^2_{BDI} (39) = 162.563, p \leq 0.001$; CFI = 0.939; TLI = 0.920; RMSEA = 0.045. $\chi^2_{ASI} (39) = 116.996, p \leq 0.001$; CFI = 0.959; TLI = 0.947; RMSEA = 0.035. *** $p < 0.001$.

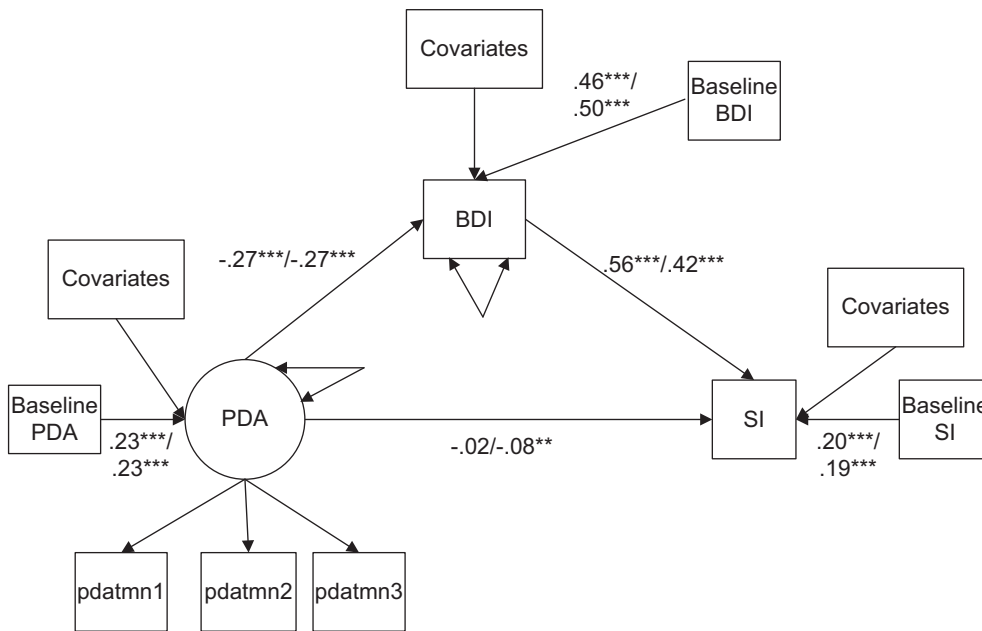


Fig. 5. Constrained longitudinal logistic structural equation models examining Beck Depression Inventory (BDI) scores, with the BDI suicide item removed, as a mediator of the association between percent days abstinent and suicidal ideation (SI). Standardized estimates of the coefficients for the BDI-SI outcome are presented before the slash; estimates for the ASI-SI are presented after the slash. Covariates include gender, age, race, treatment assignment, study arm and baseline drinks per drinking day. $\chi^2_{BDI} (52) = 175.256, p \leq 0.001$; CFI = 0.960; TLI = 0.947; RMSEA = 0.039. $\chi^2_{ASI} (57) = 208.896, p \leq 0.001$; CFI = 0.951; TLI = 0.937; RMSEA = 0.042. ** $p \leq 0.01$ /** $p < 0.001$.

models, there are statistically significant negative correlations from PDA over follow-up to depression over follow-up ($p < 0.001$), and there are positive correlations from depression over follow-up to SI over follow-up ($p < 0.001$). Similar to the findings for the analyses of DDD, there are somewhat different mediation results in the BDI-SI and the ASI-SI models. In the BDI-SI model, the absolute correlation from PDA

to SI is greatly reduced compared to the basic model, and it is statistically nonsignificant ($p = 0.303$). The results suggest that when depression is added to the model, it acts as a full mediator of the association between PDA and SI. However, in the ASI-SI model, the absolute correlation from PDA to SI is somewhat reduced compared to the basic model, but nonetheless a statistically significant, inverse association between

PDA and SI remains ($p = 0.010$); this suggests that when depression is added to the model, it acts as a partial (but not full) mediator of the association between PDA and SI. These models contain the same number of observations as the DDD models that consider mediation.

We also examined anger (STAXI score) as a moderator of the PDA–SI association (models not shown). Analyses using M-Plus showed that the interaction term for PDA and anger in the BDI-SI model (estimate = 0, $p = 0.055$) does not support statistical moderation. Moreover, there is a poor model fit [$\chi^2_{\text{PDA}}(53) = 3249.439, p \leq 0.001$; CFI = 0.262; TLI = 0.068; RMSEA = 0.206]. Similarly, the interaction term for PDA and anger in the ASI-SI model (estimate = $-0.02, p = 0.252$) analyzed using MPlus does not support statistical moderation, and the model fit is also poor [$\chi^2_{\text{PDA}}(53) = 3180.494, p \leq 0.001$; CFI = 0.227; TLI = 0.023; RMSEA = 0.203]. These models contain the same number of observations as the DDD models that consider mediation.

DISCUSSION

In the simple models without mediation or moderation, higher average drinking intensity as measured by DDD predicted greater likelihood of SI, and less frequent drinking as measured by PDA predicted lower likelihood of SI. The findings implicate both greater drinking intensity and greater drinking frequency in the promotion of suicidal thoughts and behavior among treated ADIs.

The results also showed that depression mediates the relationship between drinking frequency and drinking intensity with SI. Moreover, the degree of mediation (partial or full) by depression was sensitive to the assessment of SI. When BDI-SI was the outcome, depression fully mediated the association between the drinking variables and SI. However, when ASI-SI was the outcome, depression served as a partial mediator. These somewhat differing results are most likely attributable to measurement. The BDI-SI outcome variable was created using the suicide item from the BDI, and the BDI served as the measure of depression to test mediation. Therefore, although we removed the BDI suicide item from the BDI when we examined the latter as a mediator, greater similarity between the modified BDI (with the suicide item removed) and the BDI-SI outcome may be expected than with the use of a measure of SI derived using another scale (i.e., ASI-SI). Moreover, SI is a cognitive variable, and the BDI is weighted to assess the cognitive features of depression, consistent with Beck's cognitive theory of depression (Beck, 1967). In retrospect, these measure characteristics made it more likely that depression would more strongly mediate the relationship between drinking and SI assessed using the BDI-SI than the ASI-SI, consistent with the findings.

This study did not support the hypothesis that anger serves as a moderator of the relationship between drinking intensity (or drinking frequency) and SI in treated ADIs. Several features of the study make it ideal for examining this hypothesis, including the large sample size; reliable and valid

assessments of drinking intensity, drinking frequency, and anger; and the use of 2 assessments of SI that maximized the opportunity to uncover moderation. Although anger does not appear to moderate the drinking–SI relationship, it is important to note that the generalizability of these results to suicidal *behavior* (i.e., suicide attempt, suicide) is unclear. Whether anger (and related constructs) potentiates the risk for suicidal behavior associated with drinking requires further study. For example, anger may be hypothesized to promote unplanned (impulsive) acts of suicidal behavior during acute drinking bouts. In this context, suicidal behavior may occur with little forethought, a process that may be insensitive to detection with assessments of SI that are presumably better at capturing more persistent ideation. Along these lines, a study of ADIs showed that a measure of alcohol-related aggression was uniquely related to a history of unplanned suicide attempts but was not associated at a statistically significant level with SI or planned attempts (Conner et al., 2007), although the study was limited by the retrospective research design.

LIMITATIONS

There are limitations of the study that are important to note. Although prior studies support the validity of the BDI-SI and ASI-SI as assessments of SI, neither measure is considered a gold standard. The BDI-SI is taken from an item on the BDI, which likely served to increase the association between depression (assessed with the BDI) and SI as well as the mediating role of depression in the models using the BDI-SI outcome. Given our hypothesis that anger moderates the relationship between drinking and suicidal thoughts and behavior, it would have been ideal to also analyze a measure of anger while drinking, although the fact that individuals who are prone to anger in general also appear to be most prone to anger while drinking tempers this limitation somewhat (Giancola, 2002). The generalizability of results to suicidal behavior is unclear. Patients at acute risk for suicidal behavior, a group of keen interest, were excluded in MATCH, although we do not know how many patients were excluded for this reason. Although a model of drinking and suicidal thoughts and behavior featuring depression and anger in mediation and moderation is a logical starting point, additional mediators and moderators should be examined as guided by theory and future studies. We also did not exhaustively examine all potential directional pathways. In this regard, depression may also lead to drinking consistent with a negative affect model of drinking (Cooper et al., 1995). Finally, the results consider drinking *pattern*, and relevance to drinking in the event is unclear.

CONCLUSION AND FUTURE DIRECTIONS

We tested a clinical model of drinking and suicidal thoughts and behavior among ADIs (see Fig. 1). As hypothesized, measures of average drinking intensity and drinking

frequency predicted SI, and these associations were mediated by depression. However, inconsistent with our hypotheses, anger did not moderate the association between drinking intensity and SI, suggesting the need for refinement of the model. Unresolved questions raised by the analyses can be addressed in future studies including use of a gold standard measure of SI such as the Scale for Suicidal Ideation (Beck et al., 1979) to more definitively determine whether depression partially or fully mediates the association between drinking and SI. As well, the conceptual model should be tested with suicidal behavior (i.e., suicide attempt, suicide). More definitive results may be obtained with the use of experience sampling (Hussong, 2007) or a related strategy to gather real-time assessments of drinking, affect, and suicidal thoughts or behavior. Until such data become available, it is essential to take advantage of available resources including secondary datasets to advance the field.

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