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Abstinence self-efficacy, mood, and treatment outcomes in emerging adults with substance dependence

Brenna L. Greenfield

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_____
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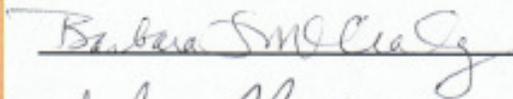
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**ABSTINENCE SELF-EFFICACY, MOOD,
AND TREATMENT OUTCOMES
IN EMERGING ADULTS
WITH SUBSTANCE DEPENDENCE**

BY

BRENNA L. GREENFIELD

B.A., ST. OLAF COLLEGE, 2006

THESIS

Submitted in Partial Fulfillment of the
Requirements for the Degree of

**Master of Science
Psychology**

The University of New Mexico
Albuquerque, New Mexico

May, 2010

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ABSTRACT

This study examined the relationship between abstinence self-efficacy (ASE), depression, psychological distress and treatment outcomes in 18 to 24 year olds with substance dependence. Three hundred and three participants completed questionnaires and interviews at three time points during a 28-day twelve-step based inpatient program, and at 1-month and 3-months post-discharge. ASE was measured with the Alcohol and Drug Use Self-Efficacy scale; psychological distress was assessed with the Brief Symptom Inventory 18; and depression diagnoses were determined from the Structured Clinical Interview for the DSM-IV. We tested the association between intake ASE, distress, and depression. Random coefficient regression analyses focused on changes in ASE during and after treatment, with distress and depression included as moderators. We used distress and depression to predict post-treatment outcomes using ordinary least squares regression. At intake, individuals with depression and high distress had significantly lower ASE. During treatment, depression and distress did not moderate changes in ASE, although individuals with depression and high distress had consistently lower ASE

throughout treatment. Depression and distress did not predict treatment outcomes, in part because of invariability in 1- and 3-month outcomes. Post-treatment, ASE did not change in a consistent manner, and changes for the most part were not moderated by depression or distress. Among the abstinent, changes in post-treatment ASE did depend on distress, in that those with above average distress at end of treatment increased in ASE from end of treatment to the 3-month follow-up. ASE is a potential byproduct of 12-step based treatment, but on the whole does not behave differently for the depressed versus non-depressed.

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INTRODUCTION

"Unless people believe they can produce desired effects by their actions they have little incentive to act or to persevere in the face of difficulties." (Bandura, 2004, pp. 621-622)

Although the transition to adulthood in the United States can be a time of great change and opportunity, it also has a dark side: levels of risky behaviors peak. Eighteen to 25-year-olds are more likely than any other age group to use illicit drugs, drive while under the influence of drugs, and binge drink (Substance Abuse and Mental Health Services Administration [SAMHSA], 2005, 2007). These risk behaviors correspond with the period of change and exploration termed emerging adulthood (Arnett, 2000). Spanning from 18 to 25 years of age, this period is characterized by a lack of demographic stability. Frequent variation in romantic partners, residence, and employment is common; few obligations exist. It is a time of lifestyle experimentation that often includes alcohol and drug use. This use does not come without consequences - emerging adults have the highest rates of past-year alcohol and drug use disorders (SAMHSA, 2005, 2007). Yet research focusing on this age group is scarce; 18 to 25 year olds are regularly grouped with adults in studies. While this may be appropriate for some research, it is not the case for substance misuse. Emerging adults have substance use patterns similar to adolescents – they tend to be poly-substance users, engage in sporadic but heavy drinking, and experience less withdrawal and physical consequences because their using careers are recent (Arnett, 2005; Chung & Maisto, 2006). Like adolescents, emerging adults often present for substance abuse treatment at the behest of someone

else, which makes it important to consider individual motivation when studying treatment outcomes (Chung & Maisto, 2006).

When substance use becomes problematic, some adolescents will seek treatment to resolve those problems. Treatment outcomes are variable, but most adolescents do reduce their substance use post-treatment (Chung & Maisto, 2006). Multiple factors predict how individuals will fare post-treatment. One salient predictor is self-efficacy. Specific self-efficacy (i.e., abstinence self-efficacy; ASE) to abstain from drinking or using drugs is a key predictor of better substance abuse treatment outcomes (McKellar, Ilgen, Moos, & Moos, 2008). Depression also predicts outcomes (albeit poorer ones), and is of particular interest because 32.75% of adults with alcohol use disorders and 44.26% of adults with drug use disorders who present for substance use treatment also meet criteria for past-year Major Depression (Grant et al., 2004). The current study aims to integrate findings on abstinence self-efficacy and depression by investigating both variables in a sample of emerging adults with substance dependence. We begin with an introduction to self-efficacy theory, followed by a discussion of comorbid substance dependence and depression, and conclude by linking the two.

Self-Efficacy

Social cognitive theory maintains that individuals act as agents in their own lives. It presupposes that people can self-regulate and self-reflect, actively shaping their environment. Levels of self-efficacy are central to this theory. Self-efficacy expectancies are beliefs individuals have about their ability to exercise control over life events (Bandura, 1989). Bandura found that, "Perceived self-efficacy was shown to be the common pathway through which different modes of influence promote change in

different spheres of functioning" (2004, p. 622). Self-efficacy expectancies govern the extent to which people feel capable of self-action and change (Bandura, 2004). Individuals change when their efficacy and sense of personal mastery increases. Theoretically, increases in self-efficacy should be followed by increases in specific behavior (Maddux, 1991).

Self-efficacy theory holds that individuals will persevere at certain behaviors depending on three factors: (1) outcome value (importance), (2) outcome expectancy (likelihood of occurring), and (3) self-efficacy expectancy (estimate of personal capability; Maddux, 1991). Self-efficacy expectancies are the most relevant to individual behavior because they influence commencement of and persistence in adaptive behaviors and coping (Maddux, 1991). Outcome expectancies are less important because they depend on self-efficacy expectancies and do not add significant utility in studies (Maddux, 1991).

Self-efficacy expectancies are relevant to multiple aspects of life, including health, work achievement, political system improvement, and clinical settings. Bandura (2004) cites four ways in which self-efficacy may increase. The first, and most influential, involves mastery or performance experiences (e.g., personal successes in overcoming obstacles, such as repeatedly turning down a drink at a party). The second method is social modeling or vicarious experiences, in which an individual observes others overcoming obstacles (e.g., Alcoholics Anonymous [AA], group therapy, inpatient role models). The effect of this second method depends on whether a person thinks he or she is similar to the person being observed (Maddux, 1991). The third involves social and verbal persuasion, when others convince someone that he or she is capable (e.g.,

affirmations from therapist). In this case, the persuader must be trusted by the person or seen as an expert. Finally, self-efficacy may vary depending on an individual's particular physical or emotional state (Bandura, 2004). Emotional arousal leads a person to associate aversive emotional states with poor performance and failure. Those who feel bad are more likely to doubt their capabilities and choose environments in which they are more likely to fail (Maddux, 1991). In this way, those who feel depressed have less self-efficacy, and are less likely to succeed in the tasks they face.

Abstinence self-efficacy. For Bandura, self-efficacy is specific: it is most useful when applied to particular situations (e.g., social gatherings, emotional states) and behaviors (e.g., substance use). High levels of drug and alcohol abstinence self-efficacy predict better substance abuse treatment outcomes across genders and treatment settings (McKellar et al., 2008; Witkiewitz & Marlatt, 2004). Witkiewitz and Marlatt include ASE as a key interpersonal, cognitive determinant of relapse in their relapse prevention model. When individuals face high-risk relapse situations, low ASE may inhibit their capability to cope with that situation successfully and not drink or use substances (Gwaltney, Metrik, Kahler, & Shiffman, 2009). Twelve-step based models do not focus explicitly on increased ASE during treatment, but attending AA meetings has been associated with higher ASE (Moos, 2008).

Independent of treatment modality, ASE generally increases during treatment (Goldbeck, Myatt, & Aitchison, 1997; McKellar et al., 2008; Wong et al., 2004). There are several reasons why this might occur: individuals may learn specific skills in treatment to cope with difficult situations (similar to relapse prevention training); they may experience the benefits of sobriety and want to extend those benefits; or they may

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gain confidence in their ability to remain abstinent because they are able to experience a period of sustained abstinence.

When measured at varying time points, abstinence self-efficacy consistently predicts outcomes. Baseline ASE is as predictive of first smoking lapse as are daily reports of ASE (Shiffman et al., 2000). In a sample of adults with alcohol use disorders (AUDs), high self-efficacy at baseline predicted remission from AUDs at 3 years (Moos & Moos, 2006). A review of 63 alcohol treatment outcomes studies (51 unique) among adults at three months post-treatment and beyond found that the strongest predictors of treatment outcomes included baseline alcohol-related self-efficacy and psychopathology ratings, along with dependence severity, motivation to change, and treatment goal (Adamson, Sellman, & Frampton, 2009). Adamson and colleagues found that, among those studies that included alcohol-related self-efficacy as a predictor, 100% of the studies cited it as a significant predictor. Correspondingly, 83% of the studies found motivation to change and 57% found psychopathology to be significant predictors of outcomes.

ASE ratings at the end of an inpatient detoxification or treatment program predicted abstinence status three months post-treatment (Goldbeck et al., 1997). Goldbeck and colleagues circumvented the question of motivation by only including those who wanted to remain abstinent for at least three months post-treatment. In the current study, we included a measure of motivation to assess for covariance between motivation and ASE.

One hundred percent confidence in ability to remain abstinent at discharge (measured by the Situational Confidence Questionnaire; Annis & Graham, 1988) was the

strongest predictor of abstinence at one-year post-treatment in an adult male sample (Ilgen, McKellar, & Tiet, 2005). Among those in remission at three years, less self-efficacy at three years predicted relapse by the 16-year follow-up (Moos & Moos, 2006).

Abstinence self-efficacy is often measured in relation to specific tempting situations. In an outpatient sample of adolescents treated for substance use disorders, higher ASE in positive affect situations at treatment intake predicted less drug use during treatment (Burleson & Kaminer, 2005). Positive affect situations are those in which an individual uses drugs to enhance a positive mood, as compared to negative affect situations, where an individual uses to dull negative emotions.

When using ASE to predict treatment outcomes, it is important to take into account current and prior substance use. Gwaltney and colleagues (2009) reviewed the ASE and smoking cessation literature and stated that effect sizes will be overestimated if researchers do not control for smoking at the time of assessment. Without making this adjustment, ASE may largely reflect smoking behavior. Studies where current smoking was not a covariate yielded the highest effect sizes. While still robust, taking current smoking behavior into account yielded small to medium effect sizes when predicting smoking behavior from prior ASE (Gwaltney et al., 2009). The authors also recommended using post-treatment ratings of ASE to predict later use because they may be more realistic than overly confident in-treatment ratings. Additionally, because estimates of ASE may fluctuate, using them to predict proximal behavior is preferable to predicting distal behavior. Despite these cautions, ASE has been found to be one of the best predictors of long-term treatment outcomes (Adamson et al., 2009). For example, even after controlling for baseline drinking, Greenfield and colleagues (2000) found that

intake self-efficacy scores on the Situational Confidence Questionnaire (which measures confidence to cope with difficult drinking-related situations) predicted number of days until first drink among a group of men and women in inpatient alcohol use disorders treatment.

Most ASE studies have focused on alcohol or tobacco, but one study examined cocaine use. When controlling for pre-treatment cocaine use, baseline ASE predicted cocaine use outcomes at three-months, but not six-months, in a sample of adult residential treatment participants with cocaine dependence (Dolan, Martin, & Rohsenow, 2008). Interestingly, lower baseline ASE was associated with more depressive symptoms as measured by the Beck Depression Inventory (BDI). Depressed individuals may be at a disadvantage when trying to recover from substance use because they tend to have lower ASE, and lower ASE is associated with poorer outcomes. The relationship between depression, ASE, and outcomes has received little attention in the literature. In addition, no studies have specifically examined ASE and treatment outcomes in emerging adults. The current study aims to address both of these arenas.

Comorbidity and Substance Use Trajectory

Most emerging adults abandon or decrease heavy drinking and drug use as they enter their late twenties, but those with high levels of anxiety, hostility, and depression continue to drink heavily into adulthood (Costanzo et al., 2007). Because of their unique demographic characteristics and high risk for continued substance abuse, research with 18 to 24 year olds with substance dependence and high levels of psychological distress merits particular attention. The combination of Major Depressive Disorder (MDD) and substance use disorders puts emerging adults at risk for suicide attempts. For example, in

a sample of 1,709 adolescents, 19% of adolescents with MDD attempted suicide, compared to 35% of those with MDD and substance use disorders (Rohde, 2009). Major Depressive Disorder often prevents emerging adults from completing age-related developmental tasks, such as work, social development, and educational attainment (Zarate, 2009).

Comorbidity between depression and substance use disorders is common. In the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), 14.50% ($SE = 0.68$) of adults with a diagnosis of substance use disorder in the past year also met criteria for Major Depression in the past year (Grant et al., 2004). Among those with a diagnosis of Major Depression in the past year, 19.20% ($SE = 0.85$) also had a comorbid substance use disorder in the previous 12 months. The National Comorbidity Replication Study found that, among community-dwelling adults with a lifetime diagnosis of MDD, 24% also met DSM-IV criteria for substance use disorders at some point (Kessler et al., 2003).

For adolescents, the relationship between depression and substance use disorders may be stronger than among adults (Rohde, 2009). Comorbid diagnoses are particularly prevalent among those who seek treatment for substance use disorders, and among depressed treatment-seeking adolescents (Rohde, 2009). Among adults who had past-year substance use disorders, 5.81% of those with AUDs sought treatment, and 32.75% ($SE = 4.01$) of this smaller group had Major Depression in the past year. Among those with drug use disorders (of which 13.10% sought treatment), 44.26% also had comorbid Major Depression (Grant et al., 2004).

In teenaged samples, those with both an alcohol use disorder and a diagnosis of MDD at outpatient treatment intake relapsed more quickly than those without an MDD diagnosis (Cornelius et al., 2004). Moreover, any Axis I disorder combined with a substance use disorder has been associated with more substance use post-treatment in adolescents (Tomlinson, Brown, & Abrantes, 2004).

Although comorbidity increases the likelihood of relapse to substance use and the severity of that relapse for both adults and adolescents, this predictive relationship is weakened when a lifetime diagnosis of MDD is used as a predictor of relapse instead of a current MDD diagnosis or a general measure of depressive symptoms (Bradizza, Stasiewicz, & Paas, 2006; Ramo & Brown, 2008). This weaker association may occur because past depressive symptoms are less relevant to the current substance use disorder. Here, we focus only on studies that used a current measure of depression as a predictor of treatment outcomes.

In an adult sample with alcohol dependence, a diagnosis of current MDD (substance-induced or primary) on admission to hospital-based detoxification was significantly related to an earlier time to first drink and relapse following treatment (Greenfield et al., 1998). Dodge, Sindelar, & Sinha (2005) found that higher BDI scores among adults at outpatient treatment intake predicted less abstinence during the year following treatment. With each additional point scored on the BDI, the likelihood of abstinence at discharge decreased by 3.5% (Dodge et al., 2005). Age also predicted abstinence in this study. The younger the individual, the more likely he or she was to relapse. This adds import to the study of mechanisms of recovery in a younger population.

In a college sample, Geisner, Larimer, and Neighbors (2004) looked at the prevalence of psychological distress and alcohol use via the Brief Symptom Inventory. With low psychological distress, both men and women consumed a similar amount of alcohol. Those participants who reported more psychological distress consumed significantly more alcohol. The relation between psychological distress and alcohol consumption was significantly stronger among men. When psychological distress was high, men drank significantly more alcohol than women and experienced significantly more problems related to their alcohol use.

Using psychological distress as a continuous variable, Flynn and colleagues (2004) found that high Brief Symptom Inventory scores from the first month of adult outpatient or inpatient treatment for substance use predicted poorer two-year outcomes. When psychological distress at two years was taken into account, this relationship became non-significant for illicit drug use but not alcohol use. Among those who did not drink post-treatment, psychological distress as measured by the Brief Symptom Inventory decreased; it did not change for those who resumed drinking (Flynn et al., 2004). In general, high levels of psychological distress may be a marker of greater probability of return to substance use.

Situational Predictors of Relapse

Beyond influencing treatment outcomes, comorbidity and psychological distress also influence which situations pose the greatest risk for relapse. The relapse prevention model (Witkiewitz & Marlatt, 2004) emphasizes the importance of negative emotional states to relapse. Following treatment for substance use disorders, situational relapse precipitants differ according to age. Studies comparing relapse precursors have focused

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primarily on adolescents and adults (e.g., Ramo & Brown, 2008); information on

emerging adults is absent. In general, adolescents (12 to 18 year olds) report a first

relapse in social situations, while adults cite negative emotions or negative interpersonal

conflict as key relapse precipitants (Chung & Maisto, 2006). Comparing adults and teens

that relapsed in the 18 months following treatment, Ramo and Brown found similar but

more complex precipitant patterns in social situations. Most adults reported relapsing in

response to social situations in which they felt tempted to drink. For teenagers, the initial

relapse came in social situations when trying to enhance a positive emotional state.

Overall, teenagers were five times more likely than adults to relapse in response to a

positive emotional state (Ramo & Brown, 2008).

Ramo and Brown's findings (2008) are limited because the adult and adolescent

sample differed in key characteristics – the adult sample was primarily male and over half

of the teen sample met DSM-III-R criteria for a depressive disorder. Results from a teen

sample with high rates of comorbid diagnoses cannot be generalized to those without

because emotional state may play a role in primary relapse precipitants (Ramo & Brown,

2008). While differences in relapse precipitants have been noted in adolescents and

adults, the particular relapse precipitants for emerging adults are unclear, as are the

differences between those with and without psychological disorders or distress.

Self-Efficacy and Depression

Self-efficacy theory offers perspective on the role of cognition in depression,

particularly expectancies for control (Maddux, 1991). Within this framework, depression

occurs when individuals want something to happen, believe it is a common event, but

have low self-efficacy expectancies (Maddux, 1991). Low self-efficacy expectancies

influence depression in that individuals feel like they cannot: (1) perform effectively, (2) develop good relationships, or (3) control ruminations (Maddux & Meier, 1995). Cognitive behavioral therapy targets these low self-efficacy expectancies by addressing irrational thoughts and automatic negative thoughts (e.g., I'll never be sober). With low self-efficacy expectancies and low outcome expectancies, life events seem uncontrollable. This feeling of powerlessness leads to not emitting behavior, acting passively, and ending up in a sub-optimal environment. For example, individuals with depression and substance dependence may not choose new friends who are supportive of sobriety because of their depression, may not feel good about themselves, and are likely to have a hard time engaging in treatment and AA. In general, depression is associated with lower overall self-efficacy (Dolan et al., 2008). Depression is a product of, and manifest in, numerous situations and behaviors where low self-efficacy expectancies are present. In a study using Ecological Momentary Assessment to monitor levels of abstinence self-efficacy and negative affect during an attempt to quit smoking, negative affect was associated with a decrease in ASE (Gwaltney, Shiffman, & Sayette, 2005).

While ASE is lower among those with comorbid diagnoses, it still retains its predictive power for treatment outcomes. In a sample with co-occurring disorders (2/3 mood disorders), greater baseline ASE predicted less cocaine and alcohol use six months post-treatment entry (Warren, Stein, & Grella, 2007). Decreases in the severity of substance use disorders and MDD during the year following treatment predicted greater self-efficacy at one-year post-treatment (McKellar et al., 2008). Specific situations where individuals report low ASE (i.e., high risk situations) tend to be situations where they will relapse (Gwaltney et al., 2005). Adolescents who have low ASE and psychiatric

symptoms seem to be most vulnerable to relapse in negative situations following instances of conflict, life stress and negative emotional states (Ramo, Anderson, Tate, & Brown, 2005).

Understanding the relationship between ASE and depression can help to individualize treatment and improve treatment outcomes. Individuals with comorbid substance use disorders and other Axis I disorders are doubly disadvantaged: comorbidity predicts worse outcomes and is associated with lower self-efficacy, and lower ASE also predicts worse treatment outcomes (Burleson & Kaminer, 2005; Moss, Kirisci, & Mezzich, 1994).

Hypotheses

The current study examined levels of drug and alcohol abstinence self-efficacy among 18- to 24-year-olds during and after a month of inpatient treatment for substance use disorders. Specific comparisons focused on levels of psychological distress (as measured by the Brief Symptom Inventory 18) and depression status. This focus is relevant because psychological distress and depression are related to lower ASE (Dolan et al., 2008). Understanding how ASE shifts during and after treatment may inform specific interventions for those with comorbid disorders. Such an investigation is particularly relevant among 18- to 24-year-olds because they have high rates of comorbid Major Depressive Disorder and substance use disorders, but few investigations have concentrated on them.

Specific study hypotheses included: (1) Baseline depression status and psychological distress both would be associated with lower overall ASE at baseline and lower ASE in negative affect situations at baseline. (2) Depression and psychological

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distress would moderate changes in ASE during treatment: increases would be greater for those without depression or high baseline psychological distress. (3) Depression and distress would predict treatment outcomes, and levels of psychological distress at the end of treatment would better predict substance use at follow-ups than a diagnosis of depression at baseline. (4) For those who remained abstinent from the end of treatment to the 3-month follow-up, ASE would increase more than in those who relapsed. Among those who were abstinent, this increase would depend on the absence of depression or psychological distress, i.e. ASE would remain static for those with depression or high psychological distress, while increasing for those without depression or with lower levels of psychological distress.

Method

Participants

This was a secondary investigation using data from a larger study on young adult treatment outcomes and 12-Step group involvement. Study participants were recruited from the Hazelden Center for Youth and Families (CYF) between October 2006 and April 2008. For the larger study, inclusion criteria included: admittance to the CYF inpatient program, and being 18 years of age or older at the time of admittance. Exclusion criteria included overt cognitive difficulties or psychosis (if notable during the recruitment process by research assistants). No one was excluded from the study for these reasons because treatment at CYF requires a certain minimal level of functioning.

CYF is located in Plymouth, MN and is part of the Hazelden Foundation, a non-profit organization with substance dependence treatment facilities nationwide. CYF is described as a co-occurring disorders treatment facility with an emphasis on substance

dependence. Treatment is grounded in the 12-step based Minnesota model of treatment and supplemented with other models (e.g., cognitive behavioral therapy, motivational interviewing). Separated by gender, key components of the 28-day inpatient program include individual and group therapy, mental health assessment, individual assignments related to treatment goals and the 12 steps of AA, education through bibliotherapy and daily lectures, and recreational and spiritual care. Patients at CYF range in age from 14 to 25 years, with a mean age of 18 years.

Research assistants approached 367 individuals for study participation. Of these, 47 (12.8%) declined to participate, citing a lack of interest ($n = 15$), a desire to avoid follow-ups ($n = 8$), the study taking too much time ($n = 8$), preferring to focus on treatment ($n = 6$), not expecting to finish treatment ($n = 5$), and other miscellaneous reasons ($n = 5$; e.g., uncomfortable withdrawal symptoms, legal advice). A chi-squared test and an independent t-test were executed to test for significant differences in gender and age between those who consented versus those who declined to participate in the study. While there was no difference in gender between the two groups, there was a statistically significant difference in age, $t(360) = p < .05$; those who declined to participate were younger. The actual age difference between the two groups was slight, with a mean of 19.8 ($SD = 1.4$) among non-participants and a mean of 20.4 ($SD = 1.6$) among participants.

Out of the 320 individuals who agreed to participate in the study, 303 (95%) completed the baseline interview and are included in analyses. In an independent samples t-test, no significant differences in age, gender, or length of stay were found between those who completed the baseline interview and those who did not.

The sample of 303 participants included 79 females (26%) and 224 males (74%). This gender split reflects the male to female ratio at CYF. Participants were, on average, 20.4 years old ($SD = 1.6$), and spent 25 days at CYF ($SD = 6.47$). Ninety-five percent of the sample was Caucasian. Fifty participants had not completed high school (16.5%), 132 (43.6%) had a H.S. Diploma or GED, 115 had completed some college (38.0%), and five had a college or vocational degree (1.6%). On the Structured Clinical Interview for the DSM-IV (SCID-I/P; First, Spitzer, Gibbon, & Williams, 1995), 65.7% of participants met DSM-IV criteria for an Axis I diagnosis in addition to the substance use disorders diagnoses (excluding psychotic, somatoform and adjustment disorders). At baseline, most participants reported alcohol ($n = 81$; 26.7%) or marijuana ($n = 81$; 26.7%) as their substances of choice. Opiates ($n = 67$; 22.1%), cocaine ($n = 36$; 11.8%), and methamphetamines ($n = 15$; 5%) also were cited frequently as substances of choice.

In the 90 days prior to treatment intake, participants reported using substances an average of 68.51 days ($SD = 25.25$) and had a mean score of 64.99 ($SD = 24.77$) on the Inventory of Drug Use Consequences (InDUC-2R; Tonigan & Miller, 2002). Reported InDUC-2R scores ranged from 0 to 135, out of a possible total of 135. At intake, mean Alcohol and Drug Use Self-Efficacy scale (ADUSE; Brown, Seraganian, Tremblay, & Annis, 2002) total score was 2.0 ($SD = 2.9$) and mean Brief Symptom Inventory 18 Global Severity Index score (BSI 18; Derogatis, 2000) was 63.3 ($SD = 10.1$).

Because of its pertinence to the study hypotheses, *Table 1* displays demographic information separated by depression status at intake. Participants were considered "depressed" if they met DSM-IV criteria on the SCID-I/P for past-month Substance-Induced Mood Disorder with depressive features ($n = 51$) or past-month Major

Depressive Disorder ($n = 48$). Chi-square tests were conducted to test for differences in ethnicity, gender, drug of choice, and education between depressed and non-depressed participants. There were no significant differences in ethnicity or education. The gender test was significant, χ^2 (1, $N = 303$) = 5.219, $p < .05$, such that females made up a larger proportion of the depressed group than expected, and a larger proportion than in the non-depressed group. The results for drug of choice were also significant, χ^2 (7, $N = 303$) = 17.217, $p < .05$. Differences in expected drug of choice were found for opiates and marijuana: a higher proportion of the depressed group reported opiates as their drug of choice, while a higher proportion of the non-depressed group reported marijuana as their drug of choice. Independent samples t-tests were conducted to test for differences between depressed and non-depressed individuals in age, length of stay, pre-treatment use, and scores on the BSI 18, ADUSE, and InDUC-2R. Significant differences were found in pre-treatment use and on the BSI 18, ADUSE, and InDUC-2R (see *Table 1*).

Procedure

One of two research assistants (B.G. being one) approached potential participants for recruitment during their first three days of inpatient treatment. To achieve a balanced age sample, every other 18 to 20 year old was approached for study participation, while each individual 21 years and older was approached for study participation. Study participants gave informed consent and were scheduled for a baseline interview within their first seven days of treatment. An independent review board, Schulmann Associates IRB, approved the larger study.

Participants were given a packet of self-report forms to complete prior to the baseline interview. The baseline interview included the SCID-I/P (First, Spitzer, Gibbon,

& Williams, 2002) and was the longest session, ranging from 90 to 180 minutes (see Appendix for other study-relevant measures administered at baseline). The SCID-I/P was given in full, minus the psychotic, adjustment, and somatoform disorders modules. The bachelor's level research assistants who administered the SCID-I/P received extensive training in the instrument prior to conducting research interviews. Each SCID-I/P was audio-recorded; a research scientist listened to all tapes in the first month of recruitment and two tapes from each week thereafter to verify diagnostic agreement between herself and the research assistant. No reliability statistics were computed for the SCID-I/P.

Participants completed a mid-treatment research assessment session approximately 14 days into treatment and an end of treatment session prior to treatment discharge. The mid- and end of treatment sessions included self-report and interview components (see Appendix).

To assist in follow-up tracking, detailed information about the participants and their contacts was obtained at the baseline interview and verified in the end of treatment session prior to treatment departure. Research assistants obtained release of information forms for significant individuals in the participant's life, as well as for aftercare facilities such as halfway houses and continuing care facilities. These forms were for tracking purposes only, and they allowed research assistants to disclose that they were calling from Hazelden about a research study and inquire regarding the participant's whereabouts.

Follow-ups occurred at one-, three-, six-, and 12-months following treatment discharge. Only the one- and three-month follow-ups were analyzed in the current study. Each follow-up session included an interview and self-report component. The interview

component was completed by phone, or in-person at CYF or Fellowship Club, a Hazelden-owned halfway house. Interviews were conducted in person only if the participant lived within a 200-mile radius of Plymouth, MN, and was willing to come in for the session. If an interview was conducted in person and the individual reported no drug or alcohol use in the follow-up period, an Intercept Oral Fluid Drug Test was given to verify the "no-use" report. Those who reported alcohol or drug use in the period of interest did not receive the Intercept Oral Fluid Drug Test. Self-report measures were completed online or in a pen and paper version. Some participants preferred to complete the self-report measures verbally by phone; this was an acceptable option.

Following each completed research session (defined as completing both interview and self-report portions) study participants were mailed a check as compensation. The per-session compensation was scaled according to session duration (e.g., $30 for 90 minute baseline vs. $10 for 20 minute mid-treatment session) and length of time since treatment departure (e.g., $20 for 1-month follow-up, $50 for 12-month follow-up). If all research sessions were completed, participants received a total of $210. At the 1-month follow-up, 84.8% of participants ($n = 251$) completed the interview (e.g., Brief Symptom Inventory 18, Form 90, & Commitment to Sobriety Scale), 42.9% ($n = 127$) completed the self-report forms (e.g., Alcohol and Drug Use Self-Efficacy scale), and seven participants withdrew from the study. At 3-months, 84.8% ($n = 245$) completed the interview, 64.0% ($n = 185$) completed the self-report forms, and an additional seven participants withdrew. Reasons for study withdrawal were not recorded.

Measures

The Appendix details the frequency and timing of measure administration. The listed six and 12-month assessments were not included in the present analyses. Additional measures were given at all time points, but they were not listed because they are not pertinent to the current investigation.

Demographics. CYF treatment attendees provide information about their gender, age, ethnicity, and education as part of the routine treatment intake process. This information was taken from their treatment records for the purposes of this study.

Psychological distress. The Brief Symptom Inventory 18 (BSI 18; Derogatis, 2000) is a shortened version of the Brief Symptom Inventory (Derogatis, 1993), which in turn is based on the Symptom Checklist-90-R (Derogatis, 1994). In the current study, the BSI 18 was administered at each time point and provided a continuous measure of psychological distress. Eighteen questions reference the previous week and are rated on a zero (not at all) to four (extremely) Likert scale. The total score is the Global Severity Index (GSI); three sets of six questions are used to create Anxiety, Depression, and Somatization subscales. Male and female scores are normed separately and a T-score greater than 63 is considered the cut-off for psychological distress (Derogatis, 2000).

The BSI 18 has been normed in community and oncology samples and has acceptable test-retest reliability and internal consistency (Derogatis, 2000). In a sample of cancer patients, Zabora and colleagues (2001) found that the full BSI 18 had a Cronbach's alpha of .89, but a factor analysis revealed four subscales instead of the hypothesized three. The fourth factor combined panic and suicidal thoughts. A second study in a sample of Central American refugees and immigrants found that the items on

the BSI 18 were internally consistent (subscales: $\alpha = 0.81$, GSI: $\alpha = 0.91$; Asner-Self, Schreiber, & Marotta, 2006). Despite this internal consistency, the authors determined that the three dimensions of the BSI 18 are not robust – the factor analysis yielded only one factor. In light of these studies, the measure is best conceptualized as a good index of global psychological distress, but not a diagnostic tool. In addition, because it theoretically combines anxiety, somatization, and depression items, it is not synonymous with SCID-I/P depression diagnoses.

Depression status. The SCID-I/P is a semi-structured interview for the primary DSM-IV Axis I diagnoses (First et al., 2002). Diagnoses of past-month MDD and past-month Substance-Induced Mood Disorder with depressive features were combined for a dichotomous predictor variable of depression present/absent. Prior to combining the SCID-I/P depression categories, we tested for differences between the two depression groups using an independent samples t-test and a chi-square test. Persons in the two depression diagnosis groups did not differ in length of stay at CYF, age, gender, InDUC-2R or BSI 18 scores, but did differ in their Form 90 pre-treatment use scores, $t(97) = -2.87$, $p = .005$ (see *Table 2*). Despite this difference, the two categories were combined because we are primarily concerned with the two groups having equal levels of distress at intake, which was demonstrated by similar scores on the BSI 18.

Reliability for structured interviews can be assessed through joint or test-retest reliability. Joint reliability is inherently higher because a second rater listens to the first rater's interview, which provides some indication of what the first interviewer selected because of skip codes. Test-retest reliability involves independent interviews with the same client. For MDD, Kappas for joint reliability were excellent (0.80 to 0.93), while

Kappas for test-retest reliability were fair, ranging from 0.61 to 0.64 (First, Spitzer, Gibbon, & Williams, 1995; Segal, Kabacoff, Hersen, Van Hasselt, & Ryan, 1995; Skre, Onstad, Torgersen, & Kringlen, 1991; Zanarini & Frankenburg, 2001; Zanarini et al., 2000). Reliability in diagnosing Substance-Induced Mood Disorder with the SCID has not been reported in the literature.

Several studies have found lower reliability when using the SCID for comorbid diagnoses among those with substance use disorders. In an inpatient population with substance use disorders, Kranzler and colleagues (1996) found very good validity of the SCID in diagnosing substance use disorders, but only moderate to poor validity for diagnosing other disorders. The authors did find that SCID MDD diagnoses had concurrent validity, in that intake Addiction Severity Index scores distinguished between those with or without diagnoses of MDD. Ross and colleagues (1995) found that, while the SCID had good test-retest reliability for substance use disorders, the test-retest reliability of the SCID over one week was 0.32 for lifetime MDD, which is considered poor. However, only 16 of the 80 potential participants with substance use disorders in this sample had MDD.

Low reliability in diagnosing MDD among those with substance use disorders may result from a discrepancy in determining whether MDD is primary or secondary; substance use often mimics symptoms of other mental disorders (Grant et al., 2004). Because of this, the Brief Symptom Inventory 18 (BSI 18) offered an alternative measure of psychopathology in the current study.

Substance use frequency and general functioning. A modified version of the Form-90 (Project MATCH Research Group, 1993) was used to assess general functioning

and frequency of substance use. Study-relevant variables included percent days abstinent (PDA) from any substance (other than nicotine) at follow-ups, days using in the 90 days before intake, pre-treatment lifetime AA attendance, and participation in additional treatment for substance dependence issues in the post-treatment periods.

The Form-90 instruments were developed for Project MATCH to assess alcohol consumption using a combination of a consumption grid and timeline follow-back (Miller & Del Boca, 1994). This family of instruments has shown good test-retest reliability and validity across samples, including adolescents (Rice, 2007; Slesnick & Tonigan, 2004; Tonigan, Miller, & Brown, 1997). In a sub-group of participants in Project MATCH, Form 90 test-retest reliability for the same interviewer was excellent for recent alcohol and drug use and days in psychological treatment (ICCs = 0.89 - .99). For recent AA attendance, test-retest reliability was fair (ICC = 0.59). Among different interviewers at the same site, test-retest reliability was variable, ranging from 0.24 for tranquilizers to 1.0 for marijuana, hallucinogens, and sedatives (Tonigan et al., 1997). Alternate versions of the form have been developed (e.g. Form-90d for drug use). Form-90d demonstrated excellent reliability in measuring illicit drug use, residential living, and 12-step attendance in an adult outpatient sample (ICCs = 0.75 - 0.82; Westerberg, Tonigan, & Miller, 1998).

Substance use consequences. The InDUC-2R (Tonigan & Miller, 2002) was administered in self-report form to gauge consequences from alcohol and other drug use in the 90 days prior to treatment (baseline version) and in the period since last contact (follow-up versions). The InDUC-2R has 50 questions scored on a Likert scale from zero to three (0 = never, 1 = once or a few times, 2 = once or twice a week, 3 = daily or almost

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daily). Subscales of the InDUC-2R include physical, interpersonal, and intrapersonal consequences, impulse control, and social responsibility. Five of the 50 questions constitute a 'control' scale meant to catch careless or dishonest responding.

Tonigan and Miller (2002) evaluated the InDUC-2R in a clinical outpatient sample. Administered pre- and post-treatment, the InDUC-2R was sensitive to consequences after treatment and provided different information than post-treatment substance use measures. Blanchard, Morgenstern, Morgan, Labouvie, & Bux (2003) examined the psychometric properties of the InDUC-2R and found high internal consistency for the entire scale ($\alpha = 0.96$). The scale loaded strongly on one factor, suggesting that the InDUC-2R is best conceptualized as a single measure of consequences. Gillaspy and Campbell (2006) further investigated the psychometric properties of the InDUC-2R in an adult male sample at an inpatient VA substance use disorders treatment center. Participants completed the InDUC-2R at treatment entry and one month following treatment discharge. The authors found excellent internal consistency for the scale ($\alpha = 0.96$ at intake, $\alpha = 0.98$ at follow-up) and low convergent validity with numbers of drink per drinking day ($r = 0.17$). This suggests that the InDUC-2R measures a different aspect of treatment outcomes than alcohol and drug consumption. InDUC-2R scores changed between measurement points ($d = 1.39$), suggesting that the measure can sensitively detect changes due to treatment. Like Blanchard and colleagues, Gillaspy and Campbell found significant redundancy between subscales, pointing to a preference for the total score.

Based on these findings, the InDUC-2R was used in addition to the Form-90 to capture a different aspect of treatment outcomes. The overall score was used as a measure

of consequences, excluding the five "control" questions meant to detect careless responding.

Abstinence self-efficacy. The Alcohol and Drug Use Self-Efficacy (ADUSE) scale (Brown et al., 2002) is a modified version of the Alcohol Abstinence Self-Efficacy scale (AASE; DiClemente, Carbonari, Montgomery, & Hughes, 1994) that inquires about drug and alcohol use. Participants completed this measure at each assessment point.

Twenty duplicate questions ask first about temptation to drink or use in different situations and then about confidence to avoid using in those same situations, for a total of 40 questions rated on a Likert scale from 0 (not at all) to 4 (extremely) in our study. The usual scaling of this measure is from 1 to 5. The "confidence" questions form an index of abstinence self-efficacy. DiClemente and colleagues constructed the AASE from Marlatt and Gordon's relapse categories (1985). Subscales include Negative Affect, Social/Positive, Physical and Other Concerns, and Craving/Urges situations.

The psychometric properties of the ADUSE have not been evaluated; it has been used in only one published study (Brown et al., 2002). The closely related AASE demonstrated excellent reliability in a treatment-seeking outpatient sample with alcohol dependence ($\alpha = 0.81$-0.88 for subscales, $\alpha = 0.92$ for the total scale; DiClemente et al., 1994). DiClemente and colleagues found that the AASE was unrelated to motivation (as measured by the University of Rhode Island Change Assessment Scale), which provides evidence for the scale's discriminant validity.

Hiller, Broome, Knight, and Simpson (2000) adapted the AASE for drug use in a residential treatment sample on probation. Unlike Brown and colleagues (2002), they did not mention receiving permission from DiClemente to adapt the scale, did not use the

name ADUSE, and inquired only about drug use, instead of the combination of alcohol and drugs found on the ADUSE. Hiller and colleagues found the same four subscales as DiClemente et al. in a confirmatory factor analysis, as well as high construct validity for their measure of ASE.

We computed an internal consistency estimate of the ADUSE. The scale demonstrated excellent reliability at intake, with an overall alpha of 0.94 (20 items, $N = 295$). Subscales also demonstrated good reliability ($\propto = 0.86 – 0.91$). These values are similar to reliability estimates for the AASE (DiClemente et al., 1994). For the ADUSE, we used item means for missing values because individual items ranged widely in their correlation with the total ADUSE score.

Motivation. The Commitment to Sobriety Scale (CSS) was administered verbally at all time points except mid-treatment. John F. Kelly, PhD, a principal investigator on the larger study, developed the measure (J. F. Kelly, personal communication, June 3, 2009). It includes nine questions assessing desire for sobriety, rated on a Likert scale from one (strongly disagree) to six (strongly agree; $\alpha = 0.68$ at intake). We combined questions 2 ("I am totally committed to staying off of alcohol/drugs") and 3 ("I will do whatever it takes to recover from my addiction") to create a 'motivation' covariate.

Other items on the CSS provided discriminant and convergent validity for the construct of abstinence self-efficacy as measured by the ADUSE. The CSS has one question regarding importance of sobriety, which was uncorrelated with intake ADUSE total score, and one question regarding confidence to stay sober, which was significantly and positively related to ADUSE total, $r(280) = .342, p < .001$.

Power Analysis

Separate power analyses were conducted for each hypothesis using G*Power 3, allowing for a two-tailed alpha and a power of at least .80 (Faul, Erdfelder, Lang, & Buchner, 2007). For all hypotheses, our sample of 303 participants was more than adequate to detect an effect size of 0.15.

Statistical Analyses

Hypothesis 1. In this exploratory hypothesis, we examined the relationship between intake depression status, psychological distress, and abstinence self-efficacy. Prior to calculating a multiple correlation coefficient between these variables, we examined the intake distribution of the BSI 18, ADUSE, and Form-90 and checked for outliers. The relationship between depression status and psychological distress was determined in order to assess for multi-collinearity, and we tested for the potential covariates of pre-treatment use, baseline motivation, and lifetime AA attendance by looking at the relationship between these variables and baseline ADUSE scores. Finally, correlations were calculated between depression status from the SCID-I/P, ADUSE scales (Total, Negative Affect, Social/Positive, Physical and Other Concerns, and Craving and Urges), and BSI 18 scores (Global Severity Index, Anxiety, Depression, Somatization). These correlations were repeated while controlling for significant covariates.

Hypothesis 2. We examined whether mood disorders and psychological distress moderated changes in total abstinence self-efficacy (ASE) during treatment. Using the three in-treatment assessment points, random coefficient regression was used (RCR; Cohen, Cohen, West, & Aiken, 2003) to model within-subject linear changes in total

ASE over the course of treatment, and to assess for variability in those linear changes. By treating time as both a fixed and a random effect, RCR gives results regarding average change as well as the degree to which there is variability between participants in within-subject slopes for changes in a particular variable. Depression status at baseline (past-month Substance-Induced Mood Disorder with depressive features or past-month Major Depressive Disorder from the SCID-I/P; coded as 0 = no depression, 1 = depression), and its interaction with time were then added to the model to determine whether there was a main effect of depression on ASE, and whether depression moderated changes in ASE over time. A second series of models was estimated, this time with psychological distress (BSI 18 Global Severity Index) as a moderator. One model treated BSI 18 at baseline as a fixed effect and then a second model treated BSI as a time-varying covariate. Because motivation for treatment, pre-treatment substance use, and pre-treatment Alcoholics Anonymous attendance are often related to outcomes, we tested whether these variables were significantly related to end of treatment ASE scores. After running the above analyses, those variables that were significantly related to ASE were included as covariates in the analyses.

SAS Proc Mixed was used for these analyses. SAS Proc Mixed is preferred because it utilizes modern maximum-likelihood (ML) based approaches for missing data, allowing all possible data points to be utilized in the analyses. Proc Mixed utilizes an expectation-maximization algorithm for ML estimation of missing values, and a participant can be included in the analyses so long as they have two valid data points, even if all others are missing. The ML approach is considered state of the art for dealing with data that are missing at random (Schafer & Graham, 2002).

Hypothesis 3. In this hypothesis, we examined the effect of depression on treatment outcomes. First, we determined how well baseline depression status from the SCID-I/P predicted one- and three-month substance use outcomes using ordinary least squares regression for the continuous outcomes of InDUC-2R consequences and Form-90 percent days abstinent. Second, we determined how well end of treatment psychological distress (BSI 18 GSI) predicted one- and three-month substance use outcomes, again using ordinary least squares regression. Third, we looked at how well psychological distress at one-month post-treatment predicted three-month substance use outcomes. These separate analyses were meant to elucidate the role of proximal distress (e.g., 1-month BSI 18 scores) on treatment outcomes, as compared to more distal, ongoing distress (e.g., intake depression status).

We concluded by determining which measure of depression/distress best predicted one-month and three-month outcomes. We included depression status at baseline and BSI 18 GSI scores at end of treatment as predictors of one-month substance use outcomes, and depression status at baseline and BSI 18 GSI scores at end of treatment and one-month to predict three-month substance use outcomes. This analysis determined which variable accounted for more variance in outcomes.

Because the measures of depression and psychological distress were somewhat multicollinear, the final analysis should be interpreted with caution. The bivariate correlation between intake depression status and end of treatment BSI 18 GSI was significant, $r(261) = .158$, $p = .01$, as was the correlation coefficient between depression and 1-month BSI 18 GSI scores, $r(252) = .179$, $p = .004$, and the correlation between BSI 18 GSI scores at end of treatment and 1-month, $r(234) = .621$, $p < .001$.

Hypothesis 4. Participants were first separated into those who remained abstinent during the three-month follow-up, and those who did not. Abstinence was chosen as the independent variable because it is a primary goal of 12-step based treatment. We examined the effect of abstinence during the 3 months post-discharge on changes in ASE from end of treatment to 3-months post-discharge. The same random coefficient regression approach described under hypothesis 2 was utilized; time, abstinence status (coded 1 = abstinent and 0 = not abstinent) and the time X abstinence status interaction were added to the equation. A significant interaction would mean that the pattern of change in ASE was different among those who remained abstinent than those who did not.

We tested the influence of the potential covariates of pre-treatment substance use, pre-treatment AA attendance, and end of treatment motivation by examining their correlation with ASE at 3-months. Those variables that were significantly related to ASE at 3-months were included as covariates in the previously described analysis.

Additional models were estimated among only those who remained abstinent for the 3-month post-treatment period; the main effect and interactions for intake Depression status and BSI 18 GSI scores at end of treatment were added. As an example, for the model including Depression status, change in ASE over time was regressed on the fixed effect of time, Depression status at baseline, and their interaction. A significant time X depression interaction would mean that change in ASE over time among abstinent individuals was dependent upon Depression status at baseline. This same analysis was then repeated for BSI 18 GSI scores at end of treatment.

Results

Hypothesis 1

At intake, ADUSE scores were normally distributed, with a skew and kurtosis less than 1 for the overall score and the four subscales. The ADUSE total distribution was more normal than the distributions of the subscales. The 'Physical & Other Concerns' subscale had a negative skew, such that individuals generally felt more confident to resist using in these situations. The opposite was true for the 'Social/Positive' scale, which had a positive skew, and comprised situations where individuals felt less confident resisting substance use. In order to understand areas in which depressed individuals particularly may struggle when trying to remain sober, post-hoc independent samples t-tests were conducted to determine whether individuals with depression differed from individuals without depression at intake in mean scores on the ADUSE subscales. No significant differences were found in mean scores on the 'Physical and Other Concerns' or 'Social/Positive' scales. As hypothesized, depressed individuals had significantly lower scores on the Negative Affect subscale, $t(293) = 3.38$, $p = .001$, and on the Craving/Urges subscale, $t(293) = 2.48$, $p = .014$.

Intake BSI 18 GSI and subscales had a skew and kurtosis less than 2, indicating an adequate level of normality. In examining the histograms, BSI 18 GSI and the BSI 18 depression and anxiety subscales were negatively skewed, with individuals reporting high levels of psychological distress at intake. The BSI 18 somatization subscale was positively skewed, with most individuals endorsing few somatization symptoms.

At intake, both Form 90 days using alcohol or drugs and CSS motivation were negatively skewed. Most participants used substances on a high proportion of the 90 days

before treatment, and most participants reported a high level of motivation at intake. Kurtosis and skew values for these variables were less than 2.

At intake, total scores on the BSI 18 GSI were significantly related to depression status, $r(303) = .498$, $p < .0001$. Those who reported past-month depression were more likely to report high psychological distress on the BSI 18 GSI. BSI 18 subscales also were significantly and positively related to intake depression status.

Intake depression status was significantly and negatively related to overall score on the ADUSE, $r(295) = -.143$, $p = .014$, such that those with intake depression reported less ASE at intake. Intake depression status was negatively related to the ADUSE subscales, although the relationship was only significant for the Negative Affect and Craving/Urges subscales. *Table 3* provides the point-biserial correlations between intake depression status and ADUSE scales.

On the whole, BSI 18 scores at intake were significantly and negatively related to ADUSE scores. Correlations between BSI 18 and ADUSE scales related to negative affect were strongest. For example, the correlation between the BSI 18 depression subscale and the ADUSE Negative Affect subscale, $r(295) = -.263$, $p < .0001$, was larger than the correlation between BSI 18 depression and the ADUSE Physical and Other Concerns subscale, $r(295) = -.111$, $p = .058$. See *Table 3* for further specific correlations between the BSI 18 and ADUSE scales.

We tested for the potential covariates of motivation at intake, lifetime AA attendance, and substance use in the 90 days before treatment. While lifetime AA attendance was not related to ASE at end of treatment, $r(264) = -.011$, $p = .855$, CSS motivation at intake was significantly and positively related to end of treatment ASE,

$r(263) = .167, p = .007$. Pre-treatment use also was significantly related to end of treatment ASE, $r(258) = -.176, p < .0001$, such that individuals with higher levels of pre-treatment use had lower abstinence self-efficacy at end treatment.

Partial correlation coefficients were computed for ADUSE scores and depression/distress measures, holding constant pre-treatment substance use (see *Table 4*). All correlations decreased as compared to correlations that did not include pre-treatment use as a covariate (see *Table 3*). For example, the point-biserial correlation between intake depression and ADUSE total score became non-significant, $r_p(292) = -.113, p = .054$. Controlling for motivation did not affect the relationship between the measures of psychological distress and the ADUSE scale – if anything it slightly increased the strength of the correlations.

In the following analyses ADUSE total and BSI 18 GSI are used as independent variables. BSI 18 subscales were correlated similarly with ADUSE scores (see *Table 3*).

Hypothesis 2

Mean ADUSE scores were 1.96 ($SD = 0.87$) at intake, 2.39 ($SD = 0.85$) at mid-treatment, and 2.52 ($SD = 0.85$) at end of treatment. Mean scores on the BSI 18 Global Severity Index (GSI) during treatment were 63.32 ($SD = 10.07$) at intake, 55.64 ($SD = 8.71$) at mid-treatment, and 53.37 ($SD = 8.72$) at end of treatment. In the BSI 18 manual, scores above 63 are considered "positive cases" of psychological distress that warrant further evaluation (Derogatis, 2000).

Random coefficient regression of within-subject linear changes in ASE showed a significant effect of time ($\gamma = .28, SE = .03, p < .0001$), indicating that, on average, ASE increased significantly during treatment. Analysis of the variance components revealed

significant variability in both the ADUSE slope and intercept (see *Table 5*). The

significant variability in the intercept indicates that there were differences in ASE at

baseline between participants, while the significant variability in slope means that there

was variability in the linear change in ASE – not everyone changed in the same way. The

significant covariance between the slope and the intercept means that ASE and time were

related. As might be expected, individuals who came into treatment with higher ASE

experienced a smaller increase in ASE over time.

Next, depression status at intake and its interaction with time were added to the

model. The linear changes in ASE remained significant, and the effect of depression on

ASE also was significant ($\gamma = -.28$, *SE* $= .10$, $p = .005$). Those with baseline depression

reported less ASE at all three in-treatment assessments (see *Figure 1*). The interaction

between time and Depression was non-significant ($\gamma = .07$, *SE* $= .06$, $p = .27$), which

means that the degree of change in ASE over time did not vary as a function of

Depression status. Adding Depression status to the model accounted for 3.84% of the

variance in between-subject differences in ASE.

When BSI 18 GSI scores at intake and their interaction with time were added to

the model (in place of Depression status), the within-subject linear changes in ASE no

longer showed a significant effect of time ($\gamma = .20$, *SE* $= .19$, $p = .28$), suggesting that

intake BSI 18 GSI scores were more strongly related to change in ASE than the passing

of time. The effect of BSI 18 GSI scores on ASE was significant ($\gamma = -.02$, *SE* $= .005$, $p <$

.0001), such that individuals with higher scores on the BSI 18 at baseline had lower ASE

scores at intake, mid-treatment, and end of treatment. The BSI 18 X Time interaction was

non-significant ($\gamma = .001$, *SE* $= .003$, $p = .67$): knowing participants' BSI 18 GSI scores at

baseline did not help explain how their ASE scores changed during treatment. In both this analysis and the Depression status analysis, the variance components were essentially the same as in *Table 5*. Adding BSI 18 scores to the model accounted for 11.77% of the between-subject differences in ASE.

Because BSI 18 scores were obtained at each in-treatment interview, BSI 18 scores were treated as a time-varying covariate in a separate analysis. There was a significant effect of time on ASE ($\gamma = .17$, *SE* = .03, *p* < .0001), as well as a significant effect of time-varying BSI 18 scores on ASE ($\gamma = -.02$, *SE* = .003, *p* < .0001). While BSI 18 scores at each time point were correlated with ASE scores at that same time point, change in BSI 18 scores did not account for differences in the slopes of ASE scores over time. In other words, the trajectory of change in BSI 18 did not influence the trajectory of change in ASE. Analysis of the covariance estimates revealed some significant variability (see *Table 6*). The ASE intercept X slope covariance was still significant ($\gamma = .26$, *SE* = .10, *p* = .01), indicating that those with higher abstinence self-efficacy at baseline experienced smaller changes in ASE over time. No variability was found in the BSI 18 intercept (i.e., everyone had high BSI 18 scores at intake), but there was significant variability in the BSI 18 slope during treatment as well as between the intercept and the slope (see *Table 6*). Individuals who started with high BSI 18 GSI scores experienced more change in scores during treatment than those who started with low scores, potentially a result of regression to the mean.

Because intake motivation and pre-treatment use were significantly related to end of treatment ASE (see Hypothesis 1), they were individually added to the RCR analyses as covariates: first with BSI 18 as a fixed effect, then with BSI 18 as a time-varying

covariate. RCR showed a significant main effect of pre-treatment use on ASE (γ = -.005, SE = .002, p = .0006). However, adding pre-treatment use to this model did not alter the results found when BSI 18 and its interaction with time were added to the model. Similar results were found when pre-treatment use was added to the model with BSI 18 as a time-varying covariate.

When baseline motivation was added to the RCR model in place of pre-treatment use, it behaved similarly to pre-treatment use: motivation did not alter previous findings, but did have a significant main effect on ASE (γ = .07, SE = .02, p < .0001). This was also the case for the model with BSI 18 at baseline and its interaction with time added to the model, and when BSI 18 was treated as a time-varying covariate. In sum, with or without the addition of covariates, the findings regarding the relationship of depression and psychological distress on changes in self-efficacy during treatment were identical.

Hypothesis 3

At 1- and 3-months post-treatment, the distributions of InDUC-2R scores and Form-90 PDA were non-normal. Mean InDUC-2R scores were 10.85 at 1-month (SD = 18.58), with a skew of 2.00 and kurtosis of 3.12 (see *Figure 2*). At 3-months, mean InDUC-2R scores were 11.74 (SD = 24.07), with a skew of 2.63 and kurtosis of 6.52. This compares with a mean InDUC-2R score of 65.00 (SD = 24.77) at intake. The average Form-90 percent days abstinent was 93.95% at 1-month (SD = 17.55) and 93.11% at 3-months (SD = 17.18). Skew values for PDA were -3.82 and -3.55 at 1- and 3-months respectively, and kurtosis values were 14.99 and 13.46 at those same time points.

These non-normal values may be related to the disproportionate number of participants who reported receiving additional treatment for substance dependence after treatment departure (e.g., outpatient or inpatient treatment, detoxification services, or sober living environment). At the 1-month follow-up, 82.9% reported receiving continuing care; this group had a mean PDA of 97.47% (SD = 10.32) as compared to those who did not receive additional treatment and reported a mean PDA of 77.25% (SD = 31.01). At 3-months, 75.9% of participants said they had received additional treatment for substance dependence since the 1-month follow-up, and these treatment receivers had a mean PDA of 96.89% (SD = 11.15). Those who had not received additional treatment reported a mean PDA of 81.26% (SD = 25.58). The difference in pre-treatment use (a marker of dependence severity) between those who received additional services at 3-months and those who did not was non-significant, $t(243)$ = -.391, p = .696.

Using ordinary least squares regression, depression status at intake did not significantly predict outcomes at 1- or 3-months. Similar non-significant results were found with end of treatment BSI 18 GSI scores as a predictor, except that these scores significantly predicted 3-month InDUC-2R scores, $F(1, 154)$ = 6.90, p = .009, such that those with higher BSI 18 GSI scores at end of treatment reported more consequences from substance use at 3-months. BSI 18 GSI end of treatment scores accounted for 4.3% of the variance in 3-month InDUC-2R scores. *Figure 3* depicts the relationship between these two variables. BSI 18 GSI scores at 1-month did not significantly predict InDUC-2R or PDA at 3-months.

When end of treatment BSI 18 GSI scores and depression status were used as predictors for 1-month outcomes, the regression equation was non-significant for InDUC-

2R scores, $F(2, 176) = 2.68$, $p = .071$, as well as PDA, $F(2, 230) = 1.73$, $p = .180$.

Including depression status and end of treatment and 1-month BSI 18 GSI scores in a

regression model significantly predicted 3-month InDUC-2R scores, $F(3, 144) = 2.71$, p

$= .048$. The combination of predictors accounted for 5.3% of the variance in substance

use consequences at 3-months. Only BSI 18 GSI scores at end of treatment made a

significant contribution to this model, $t(146) = 2.164$, $p = .032$. These three predictors did

not significantly predict PDA at 3-months, $F(3, 202) = .913$, $p = .436$.

Hypothesis 4

Mean ADUSE scores were 2.52 ($SD = 0.85$) at end of treatment, 2.53 ($SD = 0.93$)

at 1-month post-treatment, and 2.61 ($SD = .91$) at 3-months post-treatment. As a whole,

scores on the ADUSE were static during the post-treatment period, increasing by .08

from end of treatment to the 3-month follow-up. Average BSI 18 GSI scores were also

fairly stable in this period: 53.37 ($SD = 8.72$) at end of treatment, 51.23 ($SD = 9.25$) at 1-

month, and 53.31 ($SD = 9.77$) at 3-months post-treatment.

RCR showed a trend towards increased ASE over time after treatment, but this

effect was not statistically significant ($\gamma = 0.06$, $SE = 0.03$, $p = .08$). Analyses of the

variance components revealed significant variability in the change in ASE over time (see

Table 7). Specifically, there was significant variation in both the intercept and slope,

meaning that participants had different levels of ASE at end of treatment and that the

trajectory of change in ASE from end of treatment to 3 months post-treatment also varied

between subjects. The covariance was not significant: ASE scores at end of treatment

were unrelated to the trajectory of change post-treatment.

When abstinence status at 3 months was added to the model, the effect of time on post-treatment ASE remained non-significant. Neither the effect of abstinence status on ASE ($\gamma = 0.15$, $SE = 0.11$, $p = .17$) nor the interaction between time and abstinence status was significant ($\gamma = 0.03$, $SE = 0.07$, $p = .68$). Knowing participants' abstinence status at 3 months post-discharge did not provide information about their level of ASE during that period, and changes in ASE post-treatment did not differ for those who were abstinent versus those who used substances in the 3-months after treatment departure (see *Figure 4*). The addition of abstinent status to the model accounted for 3.2% of the between-subject variance in ASE scores. We controlled for baseline use and end of treatment motivation because of their significant relationship with 3-month ASE, but doing so did not alter the reported results.

The next model only included those who remained abstinent from post-treatment to 3-months ($N = 156$). In this group, the effect of time was non-significant, $\gamma = 0.07$, $SE = 0.05$, $p = .15$, such that ASE did not change significantly post-treatment. There was significant variability in the variance estimates (see *Table 8*). The significant intercept indicated that there was variability in end of treatment ASE. The covariance between the slope and intercept was non-significant: knowing the level of end of treatment ASE did not provide information about the degree of change in ASE post-treatment. The significant slope meant that there was variability in the changes in ASE post-treatment.

When intake depression status and its interaction with time were added to the model, the effect of time remained non-significant, $\gamma = 0.02$, $SE = 0.05$, $p = .70$. Similarly, both the effect of depression status on post-treatment ASE ($\gamma = -0.13$, $SE = 0.14$, $p = .34$) and the interaction of depression status and time were non-significant ($\gamma =$

0.14, $SE = 0.10$, $p = .15$). ASE levels post-treatment and their changes over time did not

depend on intake depression. The addition of depression accounted for 3.8% of the

between-subject variance in ASE scores.

Next, BSI 18 GSI end of treatment scores were added to the abstinent-only model

in place of depression status. RCR revealed a marginally significant effect of time on

ASE, $\gamma = -0.62$, $SE = 0.31$, $p = .049$, such that ASE decreased somewhat from end of

treatment to the 3-month follow-up. The main effect of BSI 18 GSI scores was also

significant, $\gamma = -0.03$, $SE = 0.01$, $p < .0001$. Individuals with low scores on the BSI 18 at

end of treatment had low ASE scores at the three recorded time points. Covariance

parameter estimates in this model and the depression status model were similar to those

in the original model. BSI 18 GSI end of treatment scores accounted for 17.9% of the

between-subject variance in ASE scores at end of treatment, 1-month post-treatment, and

3-months post-treatment.

The interaction between BSI 18 GSI end of treatment scores and time was

significant, $\gamma = 0.01$, $SE = 0.006$, $p = .03$. Changes in ASE post-treatment for the

abstinent depended on end of treatment psychological distress. *Figure 5* depicts post-

treatment changes in ASE separated by median-split high versus low BSI 18 GSI scores

at end of treatment. To probe the interaction, we examined the effect of time at three

levels: mean BSI 18 GSI scores at end of treatment, one standard deviation above the

mean BSI 18 GSI, and one standard deviation below the mean (Cohen et al., 2003). The

effect of time was only significant one standard deviation above the mean, $\gamma = 0.13$, $SE =$

0.05, $p = .005$, such that those who reported the most psychological distress at end of

treatment also increased significantly in ASE post-treatment, while those with average or below average distress did not experience significant increases in ASE post-treatment.

Discussion

The current study aimed to improve our understanding of ASE in emerging adults by examining the relationship between ASE, depression and distress, and treatment outcomes, as well as by investigating the trajectory of ASE during and after treatment. In this sample, there was a clear relationship at intake between SCID-I/P diagnoses of substance-induced or primary depression and low abstinence self-efficacy, and self-reported psychological distress (BSI 18 scores) and low abstinence self-efficacy. The characteristic low self-efficacy of depressed individuals was manifest in the significantly lower confidence participants with depression felt in regards to their ability to resist substance use. Conversely, low self-efficacy may have lead to depression – our findings do not permit a directional conclusion. The association between ASE and depression corroborates previous findings with cocaine-dependent adults at treatment intake and provides information about correlates of abstinence self-efficacy in emerging adults (Dolan et al., 2008). Dolan and colleagues used the Beck Depression Inventory in their sample; extending this to alternate measures of depression in our sample strengthens confidence in this finding. As Bandura (2004) posits in his self-efficacy theory, emotional states seem to be important in determining levels of abstinence self-efficacy.

Similar to findings from Gwaltney and colleagues (2009), controlling for pre-treatment use attenuated the baseline relationship between abstinence self-efficacy and depression, but the correlations between ASE and depression/distress measures were mostly still significant. Although our analysis was slightly different from other studies

that controlled for substance use while predicting outcomes from ASE, these partial correlations do parallel previous findings indicating that abstinence self-efficacy is partially a function of behavior. Without controlling for concurrent or previous substance use, the relationship between abstinence self-efficacy and depression is arguably overestimated (Gwaltney et al., 2009). While substance use was associated with lower ASE, the opposite is also true – less use was associated with higher ASE. This coincides with Bandura's conceptualization of increasing self-efficacy as a function of mastery experiences (2004), as well as Marlatt and Gordon's cognitive-behavioral model of relapse in which effective coping responses in high-risk situations are followed by increased self-efficacy (1985). Future studies should investigate the time-sequence of effective coping or success in abstinence and increased self-efficacy to determine the degree to which each variable influences the other, and whether these influences are bi-directional.

Although pre-treatment motivation was related to baseline ASE, controlling for motivation did not reduce the relationship between ASE and depression or psychological distress. In our sample, motivation and depression both were related to ASE and to each other, but motivation did not account for a substantial proportion of the variance in the relationship between ASE and depression. DiClemente and colleagues (1994) found that motivation (as measured by the URICA) was mostly unrelated to the Alcohol and Abstinence Self-Efficacy scale. The slight discrepancy between these two findings may relate to using different measures of motivation or different age groups. Understanding the relationship between motivation and ASE is important for theoretical clarity: are ratings of motivation synonymous to ASE ratings? Future studies should validate the CSS

(our measure of motivation), or replicate our findings with a different measure of motivation or with a similar age group of emerging adults.

Situations in which participants reported low ASE inform our understanding of substance-related situations that are most difficult for emerging adults. Like adolescents, our sample of emerging adults reported low ASE in Social/Positive situations on the ADUSE (Chung & Maisto, 2006). In developing the AASE, DiClemente and colleagues (1994) found that age was significantly related to the Social/Positive subscale of the AASE, in that younger individuals were less confident in these situations. This is in line with our findings. Adolescents and emerging adults are strongly tied to their peers and have a difficult time resisting alcohol or drugs in social situations when positive emotions are expected to follow. Our sample reported high abstinence self-efficacy in Physical & Other Concerns situations, suggesting that such situations are uncommon for this group (e.g., injury or headache), or that they feel particularly well equipped to deal with them. This study offered some support for the former, in that most participants reported very low levels of somatization on the BSI 18, which would correspond with low experience with Physical & Other Concerns situations. Understanding high-risk situations for emerging adults can help clinicians who work with this population: In general, clinicians should focus on relapse prevention for positively valenced social situations instead of situations related to physical concerns. However, this is a hypothetical leap because we only measured situations in which emerging adults *thought* that they would relapse – not those in which they actually did. In addition, high-risk situations may vary from individual to individual. Clinicians should assess ASE in different situations and

complete a functional analysis for relapse situations to determine antecedents as future therapy targets.

As hypothesized, participants with depression and high psychological distress reported significantly lower ASE in Negative Affect situations on the ADUSE than those with low psychological distress and no depression. Depression and BSI 18 scores also were significantly related to the other ADUSE subscales, although negative correlations were strongest with the Negative Affect subscale. Compared to participants without intake depression, those with depression also had significantly less ASE in craving/urges situations on the ADUSE. Marlatt and Gordon (1985) highlighted the general importance of negative affect situations as high-risk situations for relapse, which may be particularly true for those with depression because they are in a constant state of negative affect. Clinicians should help depressed individuals consider ways to cope with negative affect situations and deal with craving and urges without resorting to alcohol or drugs.

Similar to previous findings, abstinence self-efficacy increased during treatment (Goldbeck et al., 1997; Ilgen et al., 2005; Wong et al., 2004). Because abstinence self-efficacy is one of the best predictors of outcomes, increases in abstinence self-efficacy during treatment bode well for treatment outcomes (Ilgen et al., 2005; Moos & Moos, 2006). Future research should investigate the mechanisms by which self-efficacy increases. Bandura (2004) has suggested that self-efficacy may increase through social modeling. This might happen at CYF when speakers from AA visit and share how they have remained sober. Bandura also cited social and verbal persuasion as an avenue for increased self-efficacy. Counselors and peers might convince an individual of his or her ability to maintain sobriety. It seems that increases in abstinence self-efficacy are a by-

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product of a month spent in a controlled environment, among peers, learning about the 12 steps. How does this happen? At this point, any consideration of mechanisms of action is pure speculation, but future research may uncover how ASE increases during treatment.

We also found that those with intake depression or higher psychological distress had consistently lower levels of ASE during treatment. Similar to Hypothesis 1, in which high intake ASE was negatively related to intake depression and BSI 18 GSI scores, low ASE and high distress were related throughout treatment. Although the difference in ASE between the depressed and non-depressed was statistically significant throughout treatment, the clinical significance of this difference is unknown. Future research should investigate whether this gap in ASE is clinically important, or accounts for the traditionally poorer outcomes of the depressed (Grant et al., 2004).

Contrary to our predictions, in-treatment abstinence self-efficacy increased similarly for those who were depressed and those who were not, and increased similarly regardless of baseline BSI 18 scores. Even though depressed individuals had consistently low levels of ASE, they still experienced similar increases in ASE during treatment. CYF does provide additional mental health treatment for the depressed (medications and individual therapy), which could have helped them to achieve similar increases in ASE. However, these findings suggest that ASE operates similarly in the depressed, especially when considering that ASE is equally predictive of outcomes for samples with and without comorbidity (Warren et al., 2007). It is a hopeful finding for clinicians because it suggests that they can help individuals with comorbid depression and substance dependence. Future research should work to increase levels of ASE in this depressed group.

Changes in ASE during treatment seemed to be more sensitive to levels of psychological distress as measured by the BSI 18 than to intake depression status. When modeled separately, BSI 18 scores at intake accounted for 12% of the between-subject differences in ASE during treatment, while intake depression status accounted for only 4% of the between-subject differences in ASE. In addition, when BSI 18 scores were added to the model of in-treatment ASE, the effect of time on ASE became non-significant. This finding suggests that changes in ASE over time had more to do with BSI 18 scores than time itself. Improvements in BSI 18 scores during treatment may be driving changes in ASE. When depression was added to the model, the effect of time was still significant. The BSI 18 may be a more sensitive and reliable measure of depression than the SCID-I/P for substance users, or it may be tapping different aspects of psychological distress that are more related to ASE than the DSM-IV criteria for depression. The BSI 18 is easy to administer, and could be a useful tool to monitor progress during treatment.

Overall, BSI 18 scores and intake depression status did not predict 1-month or 3-month substance use outcomes: thus hypothesis 3 was not supported. This finding contradicts a body of literature in which depression has been found to predict poorer treatment outcomes (Dodge et al., 2005; Greenfield et al., 1998). We could not evaluate whether intake depression status or BSI 18 score was a better predictor of outcomes for two reasons: (1) intake depression and BSI 18 scores were strongly related and hence multi-collinear, and (2) the variables did not predict outcomes individually, so it did not make sense to continue to the combined model. Further studies with longer follow-up periods need to be conducted among emerging adults to examine the predictive power of

depression on outcomes. Six and 12-month follow-ups for this study have yet to be analyzed, and may answer this question.

The fact that depression did not predict outcomes may mean that the two are unrelated, but in this case, the narrow range of outcomes seems to be a more logical explanation. Almost everyone was abstinent at the follow-up assessments (93% PDA on average). Furthermore, few negative consequences from substance use were reported. The exemplary outcomes of this sample may have more to do with their aftercare situation than the effects of treatment: at 1 month post-treatment, 83% of the sample reported receiving additional services for substance dependence, and this group had an average PDA of 97.5 versus those who did not receive continuing care (mean PDA of 77.3). These findings speak to the importance of extended treatment for emerging adults who have a goal of abstinence from substance use. However, those who received continuing care were not necessarily more dependent – there was no difference in pre-treatment substance use between the groups who received continuing care at 1-month and those who did not – but the difference in substance use outcomes was substantial.

The drop in reported consequences on the InDUC-2R from intake to post-treatment (from an average of 65.0 to 11.0) is a noteworthy feature in this sample that diverges from many adult samples that experience consequences long after substance use ceases. Intake InDUC-2R scores in this sample were comparable to an adult outpatient sample, but considerably less than an adult inpatient sample (Tonigan & Miller, 2002). At this point in their lives, emerging adults seem to experience fewer, or more fleeting consequences.

Examining post-treatment ASE, no significant changes were found from end of treatment to 3-months, and abstinence status at 3-months did not moderate ASE trajectories. This finding was peculiar and unexpected. In line with Bandura's theory of increasing self-efficacy through mastery and performance experiences (2004), it would seem that individuals who remain abstinent would experience increases in ASE because of the strong relationship between behavior and ASE. For those in continuing care, we would expect self-efficacy to continue to increase similarly to those in treatment. Brown and colleagues (2002) did find that increases in ASE during continuing care occurred only in a Relapse Prevention continuing care group, and did not occur for a 12-step oriented aftercare group. Because CYF is a 12-step oriented program, clinicians probably prescribe 12-step oriented aftercare. Similar to Brown et al., 12-step oriented aftercare may account for the static ASE we found post-treatment. The high percentage of abstinent emerging adults also may be relevant to the null findings because of the restricted range of outcomes. Also, follow-up rates were low, decreasing power and overall confidence in the generalizability of these findings. An independent samples t-test between those who completed the ADUSE at least one post-treatment follow-up and those who did not revealed no significant differences in end-treatment ADUSE scores or 3-month PDA rates. A post-hoc analysis could look specifically at those who received residential continuing care and determine whether self-efficacy continued to grow in this sub-group. If it did not, this would support the specific nature of growth of ASE in residential treatment. It is possible that ASE reaches a ceiling by end of treatment.

When looking only at the 141 participants who remained abstinent until the 3-month follow-up and had ADUSE data at two of the three included time points, there

were no significant changes in post-treatment ASE. Similar to the in-treatment findings, depression did not moderate changes in ASE post-treatment. Again, ASE seems to be operating similarly for the depressed and non-depressed, which provides added support for in-treatment findings.

BSI 18 end of treatment scores did predict ASE at the follow-ups. Participants who experienced high psychological distress at end of treatment had less ASE at end of treatment, 1-month, and 3-months post-treatment. The importance of BSI 18 at end of treatment over depression status at intake in predicting ASE during the follow-up period points to the importance of using more proximal variables (end of treatment versus intake measures) when predicting ASE. On the other hand, BSI 18 score may have better predicted ASE because they are a continuous measure of distress (versus the dichotomous SCID-I/P depression status).The interaction was significant: BSI 18 end of treatment scores moderated changes in ASE post-treatment among the abstinent, and accounted for 17.9% of the between-subject variance in ASE. When this interaction was deconstructed, changes in ASE post-treatment only occurred for those with end of treatment BSI 18 GSI scores that were one standard deviation above the mean. This specific effect for those with high psychological distress deserves further investigation: if this group sought additional treatment, perhaps they needed the extra boost of additional care to elevate their ASE. For those with high distress that discontinued formal care, perhaps the effect of AA meetings or confidence-building successes outside of a confined setting led to differential growth in ASE.

Our findings must be considered in light of several limitations. The sample was three-quarters male, but depression occurs more frequently among women in the general

U.S. population (Kessler et al., 2005). In our sample, those in the depressed group were significantly more likely to be female than those in the non-depressed group. Future research should examine depression and ASE among a larger sample of female treatment seekers. Because they sought inpatient treatment and used substances an average of 70 out of the 90 days before treatment, this group was more dependent than most emerging adults. Abstinence self-efficacy may operate differently in a less severe sample. The collapsing of two SCID diagnostic categories may have obscured or biased findings: those with current MDD reported less substance use at baseline than those with substance-induced depression. Also, the overrepresentation of heroin users in the depressed group and overrepresentation of marijuana users in the non-depressed group may have implications for generalizability. The sample was primarily Caucasian and findings may not generalize to ethnic minority samples. Follow-up rates were low at 1 month (42.9%) and 3 months (64.0%) for self-reported assessments, versus 84.8% for 1- and 3-month phone or in-person interviews, thus affecting the testability of hypotheses that focused on post-treatment outcomes. Future analyses should examine how well ASE predicts treatment outcomes, which was not done in the current study. Including the six and 12-month follow-ups in future work with this sample will provide a more complete picture of changes in ASE during and after treatment.

Despite these limitations, this study had several strengths. Large inpatient clinical samples are notoriously hard to reach, and this study included multiple assessment points with such a population. It focused specifically on the understudied category of emerging adults with depression. Emerging adults have high rates of substance use, and those who have depression tend to continue drinking into adulthood, which adds import to the study

of comorbidity in emerging adults (Costanzo et al., 2007). Our sample did not exclude any substance dependence categories and included poly-drug users, which increases the external validity of our findings because emerging adults are often poly-drug users (Chung & Maisto, 2006).

The goal of this study was to examine changes in abstinence self-efficacy over time and its relation to depression and psychological distress in a specific sample: emerging adults attending a 12-step focused 28-day inpatient treatment program. Most of our findings corresponded with previous findings. At intake, emerging adults without depression were more confident in their ability to resist substance use. The entire sample reported low ASE in positive affect social situations. In the depressed, the lowest levels of ASE were reported in negative affect situations. Abstinence self-efficacy increased during treatment for the entire sample regardless of level of depression or psychological distress. The benefits of long-term aftercare for this dependent sample of emerging adults were apparent in post-treatment abstinence rates.

In some ways, our findings diverged from expectations: depressed individuals were able to increase their self-efficacy during treatment, even though they had consistently lower levels of ASE as compared to non-depressed individuals. If these findings hold for longer-term 6 and 12-month follow-ups, clinicians may have some assurance that emerging adults with depression can experience increases in abstinence self-efficacy. Future analyses should consider depression diagnoses or BSI 18 scores as a predictor of longer-term outcomes because of the effects of early continuing care and restricted range of drinking outcomes in this sample. In addition, investigations should determine whether increases in ASE are specific to 12-step treatment, if changes in ASE

mediate better outcomes, and whether ASE reaches a peak after 28 days. In this way, we will better understand both the construct of ASE and the experiences of emerging adults with depression and substance dependence.

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Appendix. Study Measures.

Measure	Assessment Point							Form
	Intake	Mid-Tx	End-Tx	1 mo.	3 mos.	6 mos.	12 mos.	
Demographics	X							Pull from Records
Clinical Diagnoses and Symptoms								
Brief Symptom Inventory 18	X	X	X	X	X	X	X	I
Structured Clinical Interview for the DSM-IV-TR	X							I
Substance Use Frequency								
Form-90	X			X	X	X	X	I
Substance Use Consequences								
Inventory of Drug Use Consequences	X			X	X	X	X	S
Abstinence Self-Efficacy								
Alcohol and Drug Use Self-Efficacy Scale	X	X	X	X	X	X	X	S
Motivation								
Commitment to Sobriety	X		X	X	X	X	X	I

Note: "I" = interview; "S" = self-administered questionnaire

Table 1

Participant Characteristics at Intake as a Function of Past-Month Depression Status

	Depression ($N = 99$)	No Depression ($N = 204$)
	(*n, %*)	(*n, %*)
Gender		
Female	34 (34.3)	45 (22.1)
Male	65 (65.7)	159 (77.9)
Ethnicity		
Caucasian	95 (96.0)	192 (94.1)
African American	1 (1.0)	3 (1.5)
American Indian	2 (2.0)	3 (1.5)
Asian American	0 (0.0)	3 (1.5)
Other/Unknown	1 (1.0)	3 (1.5)
Education		
Did not complete H.S.	14 (14.1)	36 (17.6)
H.S. Diploma/GED	48 (48.5)	84 (41.2)
Some College	36 (36.4)	79 (38.7)
Assoc./Bachelor's	1 (1.0)	4 (2.0)
Unknown	0 (0.0)	1 (0.5)
Substance of Choice		
Alcohol	23 (23.2)	58 (28.4)
Opiates	32 (32.3)	35 (17.2)
Marijuana	17 (17.2)	64 (31.4)
Cocaine	12 (12.1)	24 (11.8)

	Mean (*SD)*	Mean (*SD)*
Other	8 (8.0)	12 (5.9)
Methamphetamine	5 (5.1)	10 (4.9)
Age (years)	20.5 (1.6)	20.3 (1.6)
Length of stay (days)	25.1 (6.7)	25.2 (6.4)
BSI 18 GSI	70.5 (6.9)	59.8 (9.5)**
ADUSE Total	1.8 (1.0)	2.1 (0.8)*
Pre-treatment use (out of 90 days)	73.9 (22.1)	65.9 (26.3)**
Consequences (InDUC-2R)	79.5 (20.0)	58.0 (24.0)**

Note. *p* < .05; **p* < .01; Bolded categories were significantly different for depressed versus non-depressed individuals in a chi-squared test; BSI 18 GSI = Brief Symptom Inventory 18 Global Severity Index; ADUSE = Alcohol and Drug Use Self-Efficacy scale; pre-treatment use is taken from the intake Form-90; InDUC-2R = Inventory of Drug Use Consequences; InDUC-2R Total reflects reported consequences related to drug use in the 90 days prior to intake.

Table 2

Demographic Information at Intake by Depression Category

	Substance-induced (*N* = 51)	MDD (*N* = 48)
	(*n, %*)	(*n, %*)
Gender		
Female	16 (31.4)	18 (37.5)
Male	35 (68.6)	30 (62.5)
	Mean (*SD)*	Mean (*SD)*
Age (years)	20.53 (1.50)	20.50 (1.70)
Length of stay (days)	24.75 (7.45)	25.40 (5.89)
BSI 18 GSI	70.04 (7.39)	71.00 (6.31)
ADUSE Total	1.82 (1.04)	1.75 (0.94)
Pre-treatment use (out of 90 days)	79.90 (16.98)	67.60 (25.10)*
Consequences (InDUC-2R)	84.00 (18.88)	74.28 (20.31)

Note. *$p < .01$; Substance-induced = Substance-Induced Mood Disorder, depressive type, past month from the SCID-I/P; MDD = past-month Major Depressive Disorder from the SCID-I/P; BSI 18 GSI = Brief Symptom Inventory 18 Global Severity Index; ADUSE = Alcohol and Drug Use Self-Efficacy scale; pre-treatment use is pulled from the intake Form-90; InDUC-2R = Inventory of Drug Use Consequences; InDUC-2R Total reflects reported consequences related to drug use in the 90 days prior to intake.

Table 3

Correlations between Abstinence Self-Efficacy and Depression Indicators at Intake

	Alcohol and Drug Use Self-Efficacy Scale scores ($N = 295$)				
	Total	NA	P	C/U	S/P
Past month					
Depression	-.143*	-.194**	-.052	-.144*	-.096
BSI 18					
Total	-.253***	-.286***	-.174**	-.252***	-.148*
Depression	-.222***	-.263***	-.111	-.230***	-.153**
Anxiety	-.238***	-.293***	-.148*	-.205***	-.163***
Somatization	-.157**	-.126*	-.193**	-.174**	-.039

Note. *$p < .05$; **$p < .01$; ***$p < .001$; ADUSE subscales include Negative Affect (NA),

Physical and Other Concerns (P), Craving/Urges (C/U), and Social/Positive (S/P).

Table 4

Partial Correlations between Abstinence Self-Efficacy and Depression Indicators

(Controlling for Substance Use)

	Alcohol and Drug Use Self-Efficacy Scale scores ($N = 292$)				
	Total	NA	P	C/U	S/P
Past month					
Depression	-.113	-.169**	-.019	-.117*	-.074
BSI 18					
Total	-.201**	-.244***	-.118*	-.205***	-.109
Depression	-.186**	-.233***	-.070	-.198**	-.126*
Anxiety	-.187**	-.252***	-.094	-.157**	-.125*
Somatization	-.099	-.074	-.140*	-.124*	-.005

Note. *$p < .05$; **$p < .01$; ***$p < .001$; ADUSE subscales include Negative Affect (NA),

Physical and Other Concerns (P), Craving/Urges (C/U), and Social/Positive (S/P).

Table 5

Covariance Estimates for Between-Subjects Effects of Time on In-Treatment ASE

Abstinence Self-Efficacy ($N = 303$)					
Variance Component	Parameter	Estimate	SE	Z Value	*p*
Intercept	τ_{11}	0.42	0.06	7.20	< .0001
Slope/Intercept Correlation	τ_{21}	-0.06	0.03	-2.16	.03
Slope Variability	τ_{22}	0.09	0.02	3.88	< .0001

Table 6

Covariance Estimates for Between-Subjects Effects of Time on In-Treatment ASE with

Time-Varying BSI 18

Abstinence Self-Efficacy ($N = 303$)					
Variance Component	Parameter	Estimate	SE	Z Value	p
Intercept	τ_{11}	1.27	0.94	1.35	.09
Slope/Intercept Correlation	τ_{21}	0.26	0.10	2.54	.01
Slope Variability	τ_{22}	0.03	0.02	1.37	.09
BSI 18 GSI					
Intercept	τ_{31}	-0.02	0.02	-1.35	.18
Slope/Intercept Correlation	τ_{32}	-0.004	0.002	-2.66	.008
Slope Variability	τ_{33}	0.0005	0.0003	1.71	.04

Table 7

Covariance Estimates for Between-Subjects Effects of Time on Post-Treatment ASE

Abstinence Self-Efficacy ($N = 303$)					
Variance Component	Parameter	Estimate	SE	Z Value	*p*
Intercept	τ_{11}	0.47	0.07	6.84	< .0001
Slope/Intercept Correlation	τ_{21}	-0.05	0.03	-1.49	.14
Slope Variability	τ_{22}	0.09	0.03	2.98	.002

Table 8

Between-Subjects Effects of Time on Post-Treatment ASE among the Abstinent

Abstinence Self-Efficacy ($N = 141$)					
Variance Component	Parameter	Estimate	SE	Z Value	p
Intercept	τ_{11}	0.39	0.08	4.71	< .0001
Slope/Intercept Correlation	τ_{21}	-0.04	0.04	-0.90	.37
Slope Variability	τ_{22}	0.12	0.04	2.77	< .0001
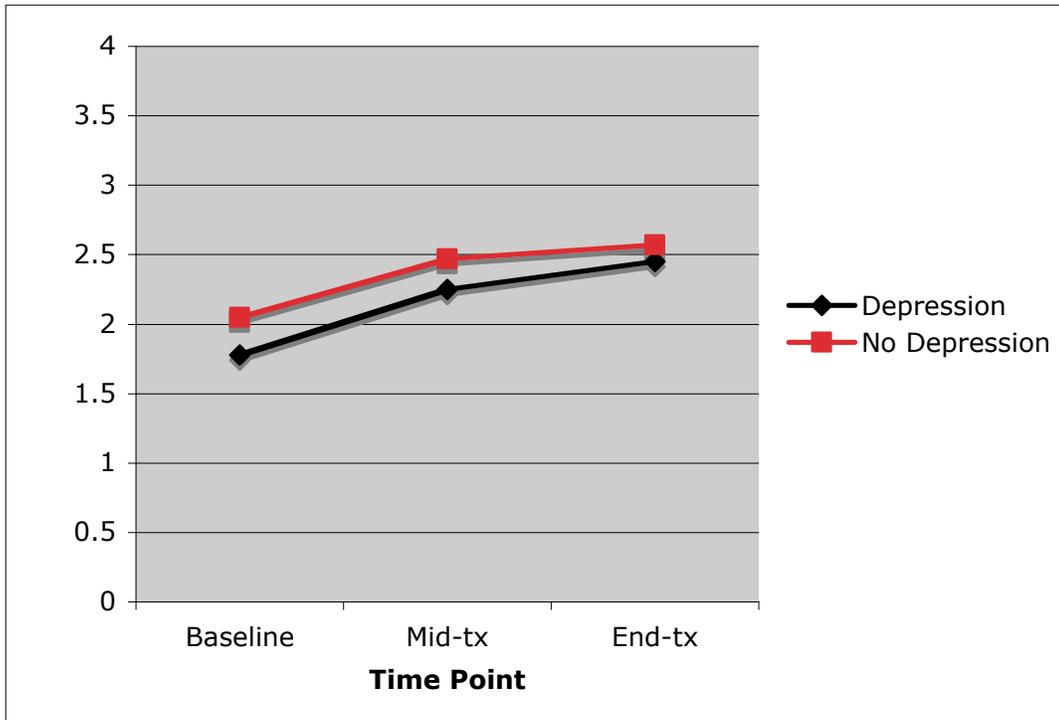
Figure 1. Changes in abstinence self-efficacy during treatment as a function of intake

depression. ADUSE = Alcohol and Drug Use Self-Efficacy scale total.
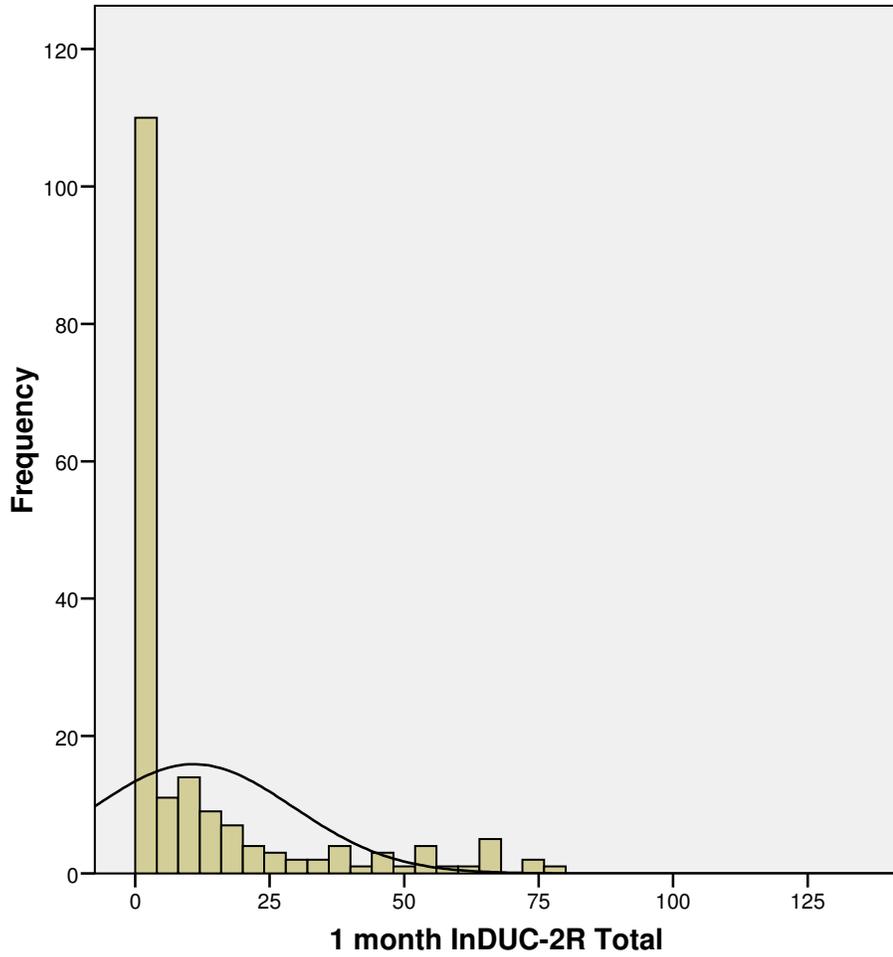
Figure 2. Distribution of substance use consequences at 1-month post-treatment. InDUC-

2R = Total score on Inventory of Drug Use Consequences in the period since discharge.

InDUC-2R scores can range from zero to 135. In this figure, $N = 185$, mean InDUC-2R =

10.85 ($SD = 18.58$).
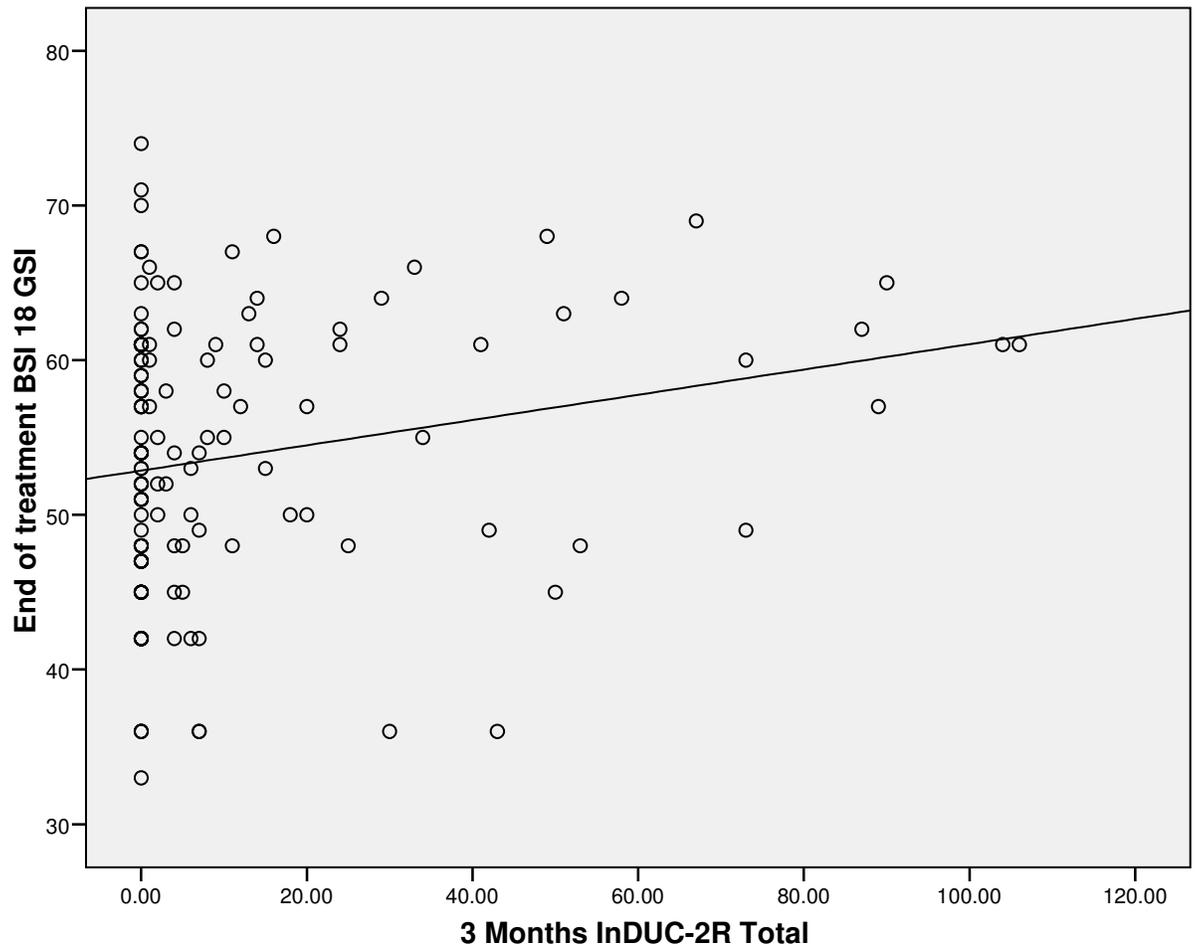
Figure 3. The relationship between end of treatment distress & 3-month substance use

consequences. BSI 18 GSI = Brief Symptom Inventory 18 Global Severity Index.

InDUC-2R = Inventory of Drug Use Consequences for period from 1 month post-

treatment to 3-months post-treatment.
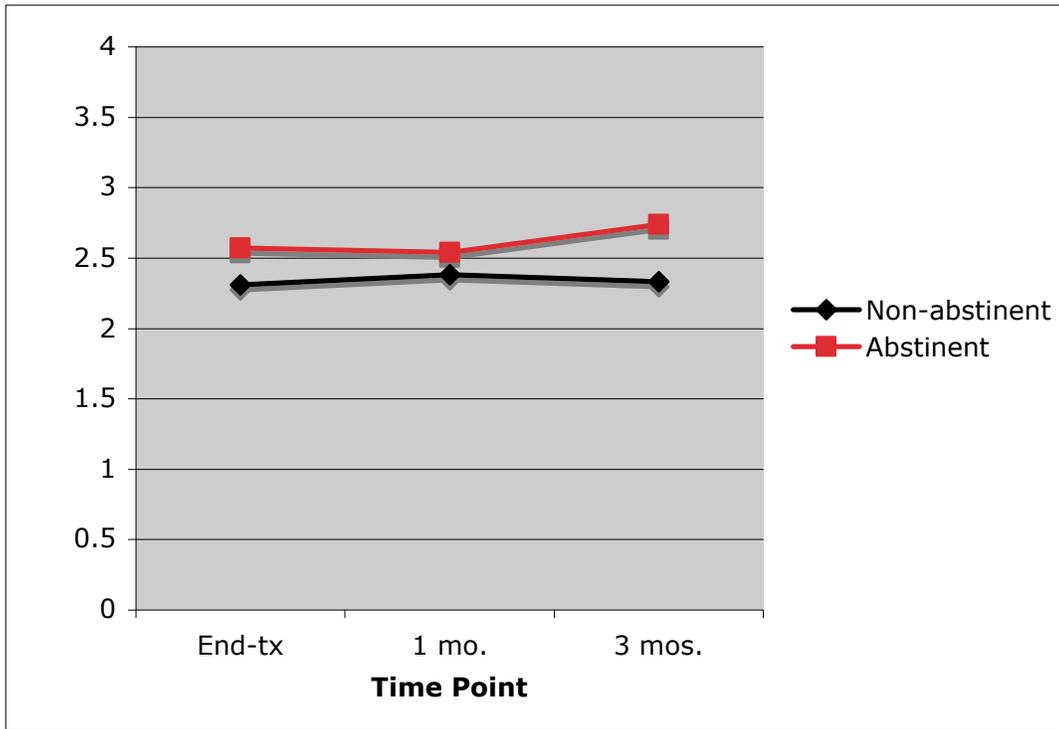
Figure 4. Changes in abstinence self-efficacy post-treatment as a function of 3-month abstinence status. ADUSE = Alcohol and Drug Use Self-Efficacy scale total score. Abstinence was defined as no substance use in the period from discharge until the 3-month interview.
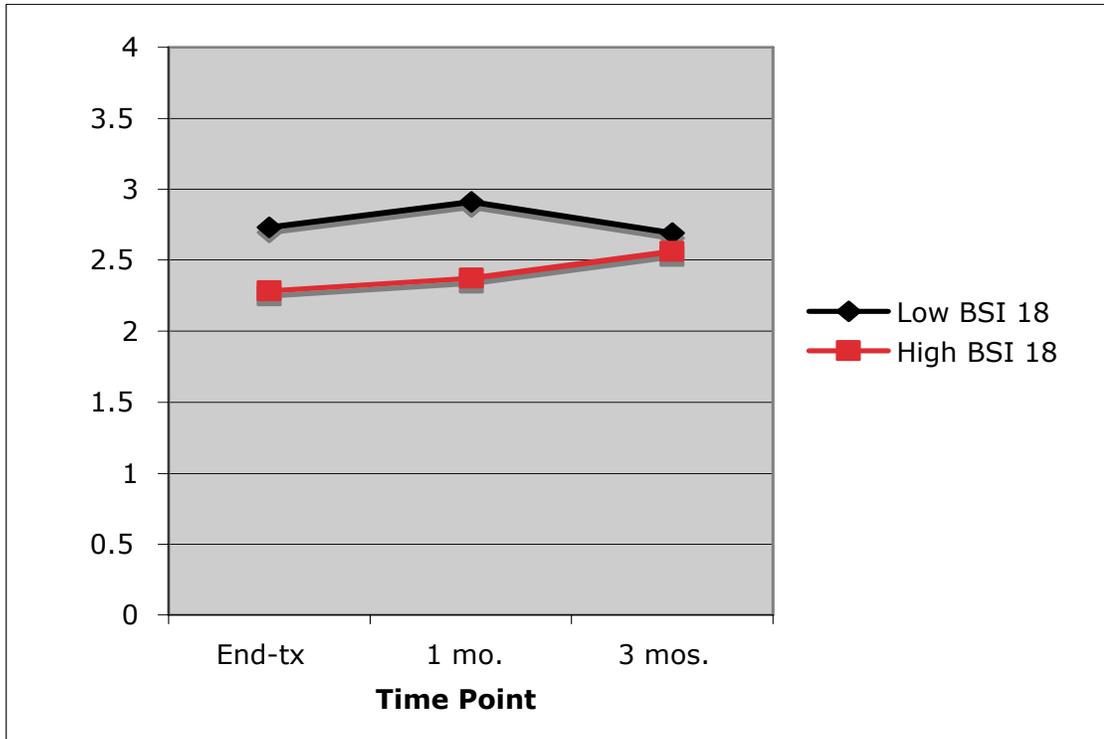
Figure 5. Changes in abstinence self-efficacy post-treatment as a function of end of treatment BSI 18 GSI scores. ADUSE = Alcohol and Drug Use Self-Efficacy scale total. BSI 18 GSI = Brief Symptom Inventory 18 Global Severity Index. Low and high BSI 18 GSI end of treatment scores were determined using a median split.