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Predictors of Success in Controlled Drinking Outcomes

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Abstract

In order for clients to make informed decisions about what treatment goal is best for them and for clinicians to help clients bolster their chances of recovery, it is critical that we know for whom non-abstinence-based treatment goals are most effective. We collected data from 143 treatment-seeking adults with AUD. The final sample consisted of participants with baseline goals of controlled drinking and follow-up data ($N = 25$). Success for individuals with controlled drinking goals was not predicted by any of the variables studied in multiple regression analysis. Results of latent change score analyses indicate that participants of lower AUD severity at baseline experienced more change in control over drinking. Latent growth curve analyses showed a decrease in peak blood alcohol concentration between days 15 and 90, and this result was not moderated by gender. Future research should seek to replicate these results in a larger and more diverse sample.

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Introduction

Background

The Temprance Movement of the 19th and 20th centuries was a feminist and religiously-motivated movement that sought to empower women, curb domestic violence, and eradicate alcohol-related "immorality" through the prohibition of alcohol use (see Boness et al., 2022; Mann et al., 2000). Notably, the movement preceded national prohibition of alcohol from 1920 to 1933, the establishment of abstinence-required asylums for individuals with alcohol problems, and the founding of Alcoholics Anonymous (AA) (Hall, 2010; Witkiewitz et al., 2020). While prohibition reduced alcohol problems for some people, prohibition created an unregulated underground drug market and encouraged drinkers to switch to liquor, worsening the problems of alcohol use for many (Hall, 2010). However, in treatment contexts today (including the still-popular AA), abstinence is often regarded as the only acceptable goal for individuals with alcohol use disorder (Davis et al., 2017; Davis & Lauritsen, 2016; Davis & Rosenberg, 2013).

Despite this, a wealth of research supports non-abstinent recovery from alcohol use (also known as controlled drinking) as both achievable and sustainable (Hasin et al., 2017; Mann et al., 2017; Tucker & Witkiewitz, 2022; Witkiewitz et al., 2017, 2018, 2019, 2020, 2021; Witkiewitz & Tucker, 2020). In line with this, a new definition of AUD recovery—one that is inclusive of reductions in drinking short of total abstinence—was adopted by the National Institute on Alcohol Abuse and Alcoholism last year (Hagman et al., 2022). However, among treatment providers, non-abstinent drinking goals are more stigmatized than a goal of abstinence, particularly for individuals with

severe AUD and those from minoritized racial and ethnic backgrounds (Davis et al., 2017; Rosenberg et al., 2020; Xin et al., 2022).

AUD has a lifetime prevalence of 29.1% (Grant et al., 2015) and usually goes untreated. One of many contributing factors to this treatment gap is that many people with AUD do not want to be abstinent from alcohol (Grant et al., 2015; Probst et al., 2015). The costs of denying individuals with AUD treatment that is inclusive of non-abstinent goals are large: the Centers for Disease Control and Prevention has estimated the total economic cost of AUD to be $249 billion, and this number does not include the psychological, emotional, and social costs that individuals who suffer from AUD and their loved ones bear (Sacks et al., 2015).

To ease these burdens, the narrative that abstinent recovery is the only valid form of recovery must shift. Treatment providers continue to harbor misconceptions about the efficacy of controlled drinking, in particular among specific groups of people, and as the distributors of treatment services for AUD, providers control the narrative (Davis et al., 2017; Rosenberg et al., 2020; Xin et al., 2022). Research on the relative efficacy of controlled drinking for different groups may help to correct misconceptions about non-abstinent recovery, bolster treatment provider support for non-abstinent goals, improve treatment engagement among people with AUD, and ease the burdens of AUD.

Defining Success with Controlled Drinking

In prior literature, success with controlled drinking is often defined by a particular number of drinks per drinking day, drinks per week, peak blood alcohol level. In Stein & Witkiewitz (2019) and Witkiewitz et al. (2017), success with controlled drinking was defined by “1 or more days with < 4/5 drinks for women/men and no heavy drinking days

during a given week." Kuerbis et al. (2012) defined success with controlled drinking as "fewer than 6 drinks on any drinking day and no more than 96 standard drinks per month," while Graber & Miller (1988) defined success as to "not exceed 20 [standard] drinks per week, and peak blood alcohol levels did not exceed 80mg% more than once per month and never exceeded 150mg%." Contrastingly, Adamson and colleagues defined success by the number of alcohol-related consequences participants reported (Adamson et al., 2010). In the Adamson study, participants were categorized as "non-problem drinking" if they reported a score of zero on a questionnaire of alcohol-related consequences, regardless of the quantity or frequency of their alcohol consumption (Adamson et al., 2010).

To date, there is no gold standard for measuring success with controlled drinking. Most extant research has picked arbitrary cutoffs of alcohol consumption, and these cutoffs may not align with how participants themselves define success with controlled drinking (Enggasser et al., 2015; Graber & Miller, 1988; Stein & Witkiewitz, 2019; Witkiewitz et al., 2017). While Adamson et al. (2010) directly captured the construct of non-problem drinking, it is similarly plausible that not every participant with a controlled drinking goal defined success with controlled drinking by an absence of alcohol-related consequences. Importantly, no studies to-date have evaluated the validity of measures of success with controlled drinking. By imposing definitions of success with controlled drinking for participants, researchers may add bias and blunt important nuance. Therefore, there is a need for research evaluating success with controlled drinking that (1) employs self-report measures of perceived control over drinking and (2) does not define

success with controlled drinking by arbitrary consumption cutoffs or lack of any consequences, two outcomes that may be less relevant to individual goals.

Perceived Control over Drinking

Perceived behavioral control has been conceptualized as comprised of two dimensions—locus of control, an individual's perceived responsibility for their behavior, and self-efficacy, the belief in one's own ability to achieve their goals (Koski-Jännes, 1994). Individuals with an internal locus of control report perceived self-control over their behavior, while those with an external locus of control perceive their behavior as beyond their control (Koski-Jännes, 1994).

Research on perceived behavioral control as it relates to alcohol use identified impaired control and lower self-efficacy to be associated with alcohol-related consequences (Blume et al., 2003; Leeman et al., 2009, 2012; Nagoshi, 1999). While some research has found greater perceived control to be predictive of abstinence from alcohol (Kahler et al., 1995) and less frequent binge-drinking (Rhodes & Clinkinbeard, 2013), other research has found greater perceived control to be positively associated with drinking frequency (Wolfe & Higgins, 2008) and perceived control to be unrelated to alcohol consumption (Adalbjarnardottir & Rafnsson, 2001; Nagoshi, 1999). An internal locus of control is more common among successfully abstinent participants and an external locus of control is more common among unimproved participants (Koski-Jännes, 1994); however, whether locus of control is predictive of success with controlled drinking remains unknown. Likewise, other than one study that identified associations between trait self-control and drinking outcomes (Stein & Witkiewitz, 2019), there are

few studies that have examined how perceived behavioral control is related to one's ability to engage in controlled drinking.

Predictors of Success in Controlled Drinking

The dynamic model of returning to drinking posits that several tonic (i.e., chronic) and phasic (i.e., situational) risk and protective factors (including affect, coping behavior, withdrawal, craving, substance use behavior, and family history) interact to produce vulnerability to drinking, and both the proximity and timing of these factors are important (Witkiewitz & Marlatt, 2004). Pulling from this model, a variety of variables—both chronic and situational—may dynamically predict success in other drinking outcomes, namely, controlled drinking.

Indeed, research has found that trait self-control, fewer drinks per day at baseline, lower negative emotionality, fewer heavy drinkers in one's social network, and higher education are predictive of success in controlled drinking outcomes, while sex, race, marital status, and the presence of psychiatric comorbidities are not predictive of success in controlled drinking outcomes (Kuerbis et al., 2020; Stein & Witkiewitz, 2019; Witkiewitz et al., 2017). Results regarding AUD severity are mixed: while several studies have found lower AUD severity to be predictive of success in controlled drinking outcomes, other findings indicate that there is no relationship between AUD severity and success in controlled drinking (A. Kuerbis et al., 2012; Orford & Keddie, 1986; Rosenberg, 1993). Results on age are mixed, with one study (Kuerbis et al., 2020) identifying younger age as predictive of success with controlled drinking and another (Witkiewitz et al., 2017) finding older age to be predictive of consistent low-risk drinking

and younger age to be predictive of starting with abstinence and later engaging in low-risk drinking.

Of note, nearly all prior work (with the exception of Kuerbis et al., 2020) has examined predictors of controlled drinking in the context of abstinence-based treatments, and very little is known about predictors of controlled drinking in treatment that supports non-abstinent goals. Further, the group of potential predictors that have been tested excludes several variables that may be associated with controlled drinking based on related research.

Family History of AUD

Family history of AUD is associated with greater severity of AUD, a greater likelihood of returning to drinking, and greater mental health service use (Milne et al., 2009). As such, it is a variable central to the nature, course, and treatment pattern of AUD. Having a family history of AUD is associated with several cognitive predictors of returning to drinking post-treatment including higher impulsivity, lower planning capacity, and slower emotion regulation response time (Jakubczyk et al., 2018; Khemiri et al., 2022). Individuals with a family history of AUD also report less intense feelings of subjective intoxication, less of a behavioral response to alcohol, and greater increases in β-endorphins from alcohol use than those with no family history of AUD (Eng et al., 2005; Johnson et al., 2000; Schuckit, 1984; Schuckit et al., 2005). These findings indicate that family history of AUD may manifest emotionally, behaviorally, and biologically in ways that might make AUD more likely and predict difficulty in maintaining a controlled drinking outcome.

Further, a treatment efficacy study of naltrexone found that individuals with more relatives who have had AUD responded better to naltrexone than to placebo (Monterosso et al., 2001). In another efficacy trial of naltrexone, individuals with a high proportion of relatives who have had AUD (but not those with a low or zero proportion) reported decreased alcohol use during naltrexone treatment (Rohsenow et al., 2007). These findings illustrate that individuals with differing degrees of family history of AUD can respond to treatment differently and highlight the importance of testing family history of AUD as a predictor of success in controlled drinking.

Age of First Alcoholic Drink and Intoxication

Research supports both age of first alcoholic drink and age of first intoxication as significant predictors of AUD development, a chronic pattern of AUD, greater alcohol consumption per drinking occasion, more frequent alcohol use, and more alcohol-related consequences (Adam et al., 2011; Buchmann et al., 2009; DeWit et al., 2000; Henry et al., 2011; Hingson et al., 2006; Lipperman-Kreda & Grube, 2019; Soundararajan et al., 2017; Stamates et al., 2016; Staton et al., 2020; Vera et al., 2020; Warner & White, 2003). Earlier age of first alcoholic drink is also predictive of impairments in incentive salience, emotionality, and executive functioning as well as neurological changes that impair attention (DeMartini et al., 2021; Nguyen-Louie et al., 2018). In addition, earlier age of first intoxication is associated with a belief that one could safely and legally drink more alcohol before driving as well as a greater likelihood of driving under the influence of alcohol (Hingson et al., 2006).

While these relationships were previously theorized to be the result of confounding environmental factors such as familial influence, twin studies controlling for

environmental factors have determined the relationship between AUD and age of first alcoholic drink or intoxication to be not entirely explainable by environmental confounds (Davis et al., 2020; J. D. Grant et al., 2006).

Whether age of first alcoholic drink or age of first intoxication is a more relevant measurement remains undetermined. Preliminary research suggests that age of first intoxication might be more strongly predictive of later substance use disorders and mental health outcomes than age of first alcoholic drink (Newton-Howes et al., 2019). Additionally, age of first alcoholic drink has been found to lack test-retest reliability, and evidence of its effects on AUD is limited (Kuntsche et al., 2016; Maimaris & McCambridge, 2014). On the other hand, evidence of a relationship between age of first intoxication and AUD is also sparse, and to our knowledge, the test-retest reliability of this variable has not been evaluated. Preliminary results also indicate that there may be power in measuring both age of first alcoholic drink and age of first intoxication rather than one or the other—a quick progression from one's first alcoholic drink to one's first intoxication is associated with more frequent drinking of any kind as well as more frequent binge drinking and accounts for unique variance in drinking outcomes (Morean et al., 2012; Newton-Howes et al., 2019).

Self-Stigma

Existing research on the relationship between self-stigma and treatment outcomes for individuals with substance use disorders is limited, and literature specific to AUD is particularly lacking (Schomerus & Angermeyer, 2008). Among individuals with substance use disorders, one study found greater self-stigma is associated with longer residential treatment stays (Luoma et al., 2014), but a different study of individuals with

substance use disorder found greater self-stigma to be predictive of higher treatment motivation (Bozdağ & Çuhadar, 2022). These findings are complicated by a meta-analysis that cited greater self-stigma as a barrier to treatment for individuals with AUD and mixed results on stigma as a predictor of treatment utilization (Hammarlund et al., 2018). One meta-analysis concluded that the relationship between self-stigma and treatment outcomes for substance use disorder may be indirect (Crapanzano et al., 2019). Though existing literature is unclear with respect to directionality, it is suggestive of self-stigma as a variable with significance in AUD treatment. Future research on the role of self-stigma in AUD treatment is needed to clarify the relationship between self-stigma and AUD treatment outcomes.

Financial Stability

The relationship between financial disadvantage and barriers to AUD treatment is well-documented (see Collins, 2016). For example, low-income income individuals often face barriers to AUD treatment such as lack of treatment knowledge, lack of access to care, and stigma (McAuliffe & Dunn, 2004; Stringer & Baker, 2018; Wallhed Finn et al., 2014). In addition, for individuals without insurance, the cost of treatment for AUD can be prohibitive (Cohen et al., 2007; Wu et al., 2003). The barriers that people with financial disadvantage face in participating in AUD treatment are especially concerning considering that lower socioeconomic status (SES) is associated with both higher alcohol-related mortality (Nandi et al., 2014) and more alcohol-related consequences (Grittner et al., 2012). Further, these barriers are indicative of financial disadvantage as a potential risk factor for worse outcomes among individuals in treatment for AUD. Indeed,

lower SES is consistently predictive of worse drinking-related treatment outcomes (Adamson et al., 2009; Westermeyer, 1989).

The AARDoC Model

Finally, the Alcohol and Addiction Research Domain Criteria (AARDoC) is a recently proposed framework for understanding AUD etiology, maintenance, and recovery (Litten et al., 2015). The AARDoC is based primarily on the addiction cycle, a model that proposes alcohol use to occur in three stages (binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation) that map onto dysfunction in the reward, stress, and executive function networks of the brain (Koob & Volkow, 2016). The three primary domains of the AARDoC, derived from Koob & Volkow's addiction cycle framework, are incentive salience, negative emotionality, and impairments in executive functioning (Koob & Volkow, 2010; 2016; Witkiewitz et al., 2022). The AARDoC model posits that these domains map onto both underlying neurological dysfunction and AUD symptoms (Litten et al., 2015). Specifically, incentive salience is characterized by sensitivity and attraction to reward and involves neurobiological dysfunction in the basal ganglia (Koob & Volkow, 2016). Negative emotionality, defined by a propensity for anxiety, sadness, fear, and other negative moods, is associated with dysfunction in the extended amygdala and habenula (Koob & Volkow, 2016). Executive function involves cognitive processes that are necessary for cognitive control (e.g., working memory) and is characterized by dysfunction in the prefrontal cortex, insula, and allocortex (Koob & Volkow, 2016).

Greater incentive salience, negative emotionality, and executive functioning impairments have been validated as predictive of poorer abstinent and non-abstinent

AUD recovery outcomes (Witkiewitz et al., 2022). Neurological validation of these impairments has also been reported via functional and resting-state magnetic resonance imaging techniques (Al-Khalil et al., 2021; Drossel et al., 2022; Zilverstand et al., 2018). Through the integration of neuroscience and addiction treatment, the AARDoC model shows promise and practicality in improving client outcomes (Kwako et al., 2016; Verdejo-Garcia et al., 2019). However, the AARDoC constructs have only been examined as predictors of controlled drinking in the context of abstinence-based randomized control trials (Witkiewitz et al., 2022). Because participation in abstinence-based treatment for AUD does not offer support for controlled drinking goals, these results on the association between AARDoC domains and non-abstinent recovery outcomes might capture a characteristically different group of people than individuals who enter AUD treatment with a goal of maintaining controlled drinking. Moreover, randomized clinical trial participants represent a subgroup of individuals with AUD that might also be uncharacteristic of the broader population with AUD, and as such, naturalistic research of a treatment-seeking sample could offer more generalizable results.

The Present Study

The present study sought to validate the roles of demographic variables, self-stigma, behavioral measures, and the AARDoC model of addiction in predicting success with controlled drinking outcomes. We collected data from treatment-seeking adults in the United States with AUD ($N = 25$) who have non-abstinence-based treatment goals and who enrolled in telehealth treatment for AUD via the Ria treatment platform.

We predicted that demographic variables, stigma, financial stability, and the AARDoC model of addiction would be associated with success with controlled drinking

outcomes such that the following participant groups would show greater success in controlled drinking: those of younger age, those with less severe AUD, those with less of a family history of AUD, those with a greater gap in between first alcoholic drink and first intoxication, those with less self-stigma, those who are more financially stable, those who score lower on incentive salience and negative emotionality, and those who have less impaired executive function.

Method

Data Source

The Ria Treatment Program (henceforth "Ria") is a commercial telehealth platform established in 2017 that offers AUD treatment from certified addiction medicine specialists. The program offers treatment that is entirely virtual (telehealth) and tailored to the goals of patients (e.g., including goals of abstinence or non-abstinent drinking reductions). It offers prescriptions for medications for AUD, regular visits with addiction medicine specialists and recovery coaches, didactic learning materials, and daily breathalyzer readings of blood alcohol concentration to facilitate treatment progress monitoring. Currently, more than 20 patients per week initiate treatment with Ria. Hallgren and colleagues (2023) conducted a longitudinal observational study using data from 4121 patients in the Ria treatment program, finding that 65% of patients endorsed a non-abstinent drinking reduction goal. Approximately 50% of patients who initiated treatment with Ria were retained in treatment for at least 90 days (which is comparable to 90-day retention rates in other AUD settings) and those who were retained for over 90 days experienced significant reductions in blood alcohol concentration (BAC) over the

first 90 treatment days, reflecting significant reductions in drinking (Hallgren et al., 2023).

Participants

We recruited 132 participants with the inclusion criteria that they be a patient of Ria, receive both pharmaceutical and psychotherapeutic services from Ria (to collect a sample representative of Ria's clientele, as 98% of patients of Ria receive both pharmaceutical and psychotherapeutic services), be at least 18 years of age, have a controlled drinking goal, and opt-in to participating in our study. For recruitment, Ria built into their program a standard question during a multi-stage intake process that asked patients if they would like to participate in a research study conducted at The University of New Mexico. The statement indicated that participation in the study would be voluntary and that choosing to participate would not impact the treatment they received through Ria. The statement indicated that participants would be compensated for their time via gift cards. The statement also indicated that if they were interested in participating, the Ria treatment program would share anonymized patient information with researchers at The University of New Mexico.

Procedures

The following procedures received IRB approval at the University of New Mexico and were conducted via an internet survey.

Individuals who opted to participate in the study during Ria's intake process were provided with a link to an informational page and a consent form for participation in research. The consent form described how patients' data will be shared between Ria and the University of New Mexico and the risks and benefits of participation. Upon

consenting, the participants were then directed to a baseline survey. The informational page, consent form, and baseline survey (and, later, the follow-up survey) were linked via a patient identification number that was embedded in the link to the informational page. This identification number was unique to the participant and was shared between Ria and UNM. In doing so, Ria was able to provide researchers at the University of New Mexico with a dataset that included individual participants' demographics, intake information (i.e., drinking goal, medical and treatment history), diagnoses, medications, clinical encounters (i.e., clinical visits, coaching encounters), breath alcohol concentrations, screening measures, and dates of enrollment and engagement (i.e., coaching calls) in the Ria treatment program.

After signing the consent form, participants completed a series of self-report measures (41 items total) of demographic information, AARDoC domains, AUD symptoms, treatment goal, family history of AUD, age of first drink and intoxication, self-stigma, and financial stability. Upon completion of the survey, participants received a $20 e-gift card.

Three months after the baseline time point, participants completed a series of self-report measures (41 items total) of AARDoC domains, AUD symptoms, treatment goal, evaluation of treatment, self-stigma, financial stability, and past-week alcohol consumption. Upon completion of the survey, participants received a $20 e-gift card. Participants were prompted via automated email messages to complete the follow-up survey 90, 92, 94, and 96 days after they completed the baseline survey. Additionally, participants indicated their preferred method of contact (phone call, text message, or email) during the baseline survey and were contacted via their chosen method of contact

three times by members of the research study team. In total, participants were prompted up to seven times to complete the follow-up survey.

Measures

All measures were assessed at baseline and the 3-month follow-up unless otherwise noted.

Primary Outcome Measure

Success with controlled drinking. Success with controlled drinking was planned to be defined by participants' rating of perceived success from the Evaluation of Treatment questionnaire at the 3-month follow-up (COMBINE Study Research Group, 2003). The item asks “Overall, how would you describe how you have changed your drinking behavior since you began treatment?” with Likert-type response options ranging from “I’m much better” to “I’m much worse.” However, lack of variability in the final dataset prevented us from using this item. Specifically, no participants reported iatrogenic treatment effects, 8.0% of participants in the final sample reported no perceived change over treatment, 60% reported “a little” improvement, and 32% reported “much” improvement. Instead, success was measured using an item from the executive function subscale of the AARDoC measure at the 3-month follow-up (Witkiewitz et al., 2022). The item probes “How much control do you have over drinking?” with 5 Likert-type response options ranging from “I am rarely able to delay drinking even momentarily” to “I have complete control.” 12.0% of participants reported being in “complete control” over their drinking, 28% reported “usually” being able to control their drinking, 48% reported “difficulty” in controlling their drinking, and 8% reported that they “must drink and can only delay drinking with difficulty.” Of note, the amended measure of success

with controlled drinking aligns better with the primary objective of the present study, to measure predictors of success *with controlled drinking outcomes*. Participants who began treatment with a controlled drinking goal and later found success with a goal of abstinence would, under the original measure of success with controlled drinking, be classified as successful. However, these participants were not successful with controlled drinking outcomes. Using the amended measure of success with controlled drinking, this nuance is accounted for, and participants who initially report a controlled drinking goal and later find success with abstinence are not classified as having a successful controlled drinking outcome.

Predictor Measures

Demographics. Participants were asked to report on their racial makeup, ethnic background, date of birth, sex, gender, and whether they are transgender. Demographics were assessed only at baseline.

AARDoC domains. Fourteen items assessing addiction cycle domains (i.e., incentive salience, negative emotionality, and executive function), validated by Witkiewitz and colleagues (2022), were administered. An example item from this measure is "After taking one or two drinks, can you usually stop? (during the past 12 months)" with response options "Yes" and "No." Of note, one item in the executive function subscale, "How much control do you have over drinking?," was used as the outcome variable and excluded from the scoring of executive function. Items from each domain were mean-scored, and higher scores indicated greater incentive salience, heightened negative emotionality, and more impaired executive functioning. The incentive salience domain had an omega reliability coefficient of 0.74 at baseline (0.83 at

follow-up), negative emotionality had an omega reliability coefficient of 0.82 at baseline (0.84 at follow-up), and executive function had an omega reliability coefficient of 0.85 at baseline (0.80 at follow-up).

AUD symptoms. Symptoms of AUD were captured via the Alcohol Symptom Checklist, an 11-item questionnaire developed to assess the presence or absence of DSM-5 criteria for AUD over the past year (Hallgren et al., 2022). An example item from this measure is "In the past 12 months, when you drank, did you drink more or for longer than you planned to?" with response options "Yes" and "No." Items from this measure were sum-scored, and higher scores indicated more severe AUD. The omega reliability coefficient of this measure was 0.77 at baseline and 0.85 at follow-up.

Treatment goal. Treatment goal was assessed using a single item from the Treatment Experiences and Expectations measure (COMBINE Study Research Group, 2003). The item reads, "We would like to know what goal you have chosen for yourself about using alcohol at this time. Pick only one of the following goals." and response options include goals of controlled drinking, occasional use, short-term abstinence, long-term abstinence with room for slip ups, permanent and total abstinence, no clear goal, and an open-response field for individuals to describe an alternative, unlisted goal. Treatment goal was assessed only at baseline. Goals of controlled drinking and occasional use were scored as controlled drinking goals, and goals of short-term abstinence, long-term abstinence with room for slip ups, and permanent and total abstinence were scored as abstinent goals.

Family history of AUD. Family history was measured using the Family History scale, adapted to a single item format (Miller & Marlatt, 1984). This item asks "Have any

of your blood relatives had what you would call a significant drinking problem? (select all that apply)" with response options including parents, siblings, and children of the participant. Family history was assessed only at baseline. The number of family members with a significant drinking problem that a participant endorsed were summed in scoring this item.

Age lapse. Time between age of first drink and age of first intoxication was calculated by subtracting participants' age of first drink from their age of first intoxication. Higher scores indicate a longer lapse in time between age of first drink and age of first intoxication.

***Age of first drink*.** Participants were asked a single question regarding the age of their first alcoholic drink that is more than a sip. Specifically, we used the wording from Sartor et al. (2009), which asked "How old were you the first time you had more than just a sip of beer, wine, or spirits?" Age of first drink was assessed only at baseline.

Age of first intoxication. Participants were asked a single question regarding the age of their first intoxication. We used wording from Morean et al. (2014), which asked ""How old were you the first time you got drunk?" Age of first intoxication was assessed only at baseline.

Self-stigma. Stigma was assessed using a modified version of the Substance Abuse Self-Stigma Scale (Luoma et al., 2013). This version consists of three items. An example item from this measure is "I have the thought that I should be ashamed of myself." with Likert-type response options ranging from "Never or almost never" to "Very often." Items were mean-scored, and higher scores indicated greater self-stigma.

This measure had an omega reliability coefficient of 0.68 at baseline and 0.87 at follow-up.

Financial stability. Socioeconomic stability was captured via an abbreviated version of a social determinants of health assessment created by Kaiser Permanente (Gordon et al., 2023). This version consists of four items; one from each subscale (financial resource strain, food insecurity, transportation needs, and housing instability) (Gordon et al., 2023). An example item from this measure is "How hard is it for you to pay for the very basics like food, housing, medical care, and heating?" with Likert-type response options ranging from "Very hard" to "Not hard at all." Items were scored in a binary fashion; participants who endorsed any socioeconomic instability on this measure were coded as "1," and those who did not report any socioeconomic instability were coded as "0." This measure had an omega reliability coefficient of 0.76 at both baseline and follow-up.

Alcohol consumption. Participant alcohol consumption was measured in two ways: via a self-report, past-week version of the Timeline Follow-Back and via breath alcohol concentration (BAC) measurements collected by Ria.

Timeline Follow-Back. To measure alcohol consumption, we asked participants to record the number of standard drinks of alcohol they consumed on each day in the past week (Sobell & Sobell, 1992). This measure was scored by calculating participants' average drinks per drinking day. Those who were abstinent were coded as "0" drinks per drinking day.

BAC. Breathalyzer readings of participants' breath alcohol concentration were collected by Ria and shared with the UNM team.

Analyses

Data was analyzed using *R* version 2022.12.0+353 (R Core Team, 2022).

To validate the role of demographic variables, stigma, AUD severity, and the AARDoC model of addiction in predicting success with controlled drinking outcomes, we employed multiple linear regression analyses. Model covariates included sex, age, AUD severity at baseline, self-stigma at baseline, family history of a "significant drinking problem," number of years between one's first drink and first intoxication, incentive salience at baseline, negative emotionality at baseline, and executive function at baseline. Models controlled for baseline level of control over one's drinking and baseline alcohol consumption. Financial stability at baseline was a planned covariate but excluded in our analyses because a large proportion of the final sample (25%) were missing these data. An a priori power analysis was conducted using G*Power version 3.1.9.7 (Faul et al., 2007). With a significance criterion of $\alpha < .05$ and power = .80, the minimum sample size needed to detect a "small" effect (per Cohen's general guidelines) is $N = 34$ (Cohen, 1988). Given the large number of tests, we used a Holm correction to correct for multiple comparisons (Holm, 1979). In Holm corrections, p-values are ranked from smallest to largest and each alpha value (α) is adjusted based on its rank (Hochberg, 1988). Specifically, $\alpha = \alpha/(m - i + 1)$, where i is equal to the rank of the p-value and m is equal to the total number of hypotheses (Hochberg, 1988). As opposed to the Bonferroni correction, where , $\alpha = \alpha/m$, the Holm correction adjusts significance levels according to the rank of the p-value (Hochberg, 1988). A null hypothesis is rejected when the p-value is less than or equal to its adjusted significance level (Aickin & Gensler, 1996). This process begins with the smallest p-values and continues until the first encountered non-

rejection (Aickin & Gensler, 1996). As a result, Holm corrections are less conservative in adjusting for multiple comparisons and bolster statistical power beyond what Bonferroni corrections can offer (Aickin & Gensler, 1996).

For baseline predictors that demonstrated change over time in a paired sample t-test, we used latent change score models (LCSM) (via the *lavaan* package in *R*; Rosseel, 2012). LCSM models leverage latent variables to provide a more accurate representation of change over time than t-tests. The latent component allows us to differentiate true variation from measurement error, and this is important because raw change scores can have low reliability and validity (Cronbach & Furby, 1970; Lance et al., 2000). LCSM models were run using the following structure: paths from baseline to follow-up true scores, from baseline to follow-up measurement error, and from latent slopes to measurement error. Additionally, the following parameter constraints were applied: (1) a latent slope of 1.0, which assumes a constant rate of change, and (2) equivalent measurement error within each measure and across timepoints, which assumes that within-measure, measurement error remains constant over time. An a priori power analysis was conducted using the *semPower* package in *R* (Moshagen, 2023). With a significance criterion of $\alpha = .05$ and power = .80, the minimum sample size needed to detect a "small" effect (per Cohen's general guidelines) is $N = 1195$ (Cohen, 1988). Because our sample size is significantly smaller than this number, we interpret LCSM model results with caution.

In order to replicate the results of Hallgren et al. (2022) and model trajectories of peak BAC over the 3-month follow-up period, we computed piecewise latent growth curve models among participants with 90 days of BAC data (via the *lmerTest* package in

R) (Kuznetsova et al., 2017). These mixed effect models offer far greater statistical power compared to traditional methods of measuring change over time (Curran et al., 2010) and were fit following the methodology set forth in Hallgren et al., (2022). Specifically, changes over time were captured in two time periods: days 1-14 and days 15-90. In doing so, we sought to (1) replicate the results of Hallgren et al., (2022) in a sample of Ria patients that opted-in to participation in a research study and (2) be able to detect nonlinear change over time. Gender was evaluated as a moderator of peak BAC trajectory in a separate latent growth curve model.

Results

Descriptive Statistics

Of 1054 individuals contacted and potentially eligible to participate in the research, 143 participants completed the baseline survey, indicating a relatively low response rate (13.6%). Of those 143 participants who completed the baseline, 43.40% had a controlled drinking goal (n=62). As of the date of data extraction (3/29/2024), 118 participants of the original 143 were eligible for follow-up and 55 were retained (46.6%). Of those retained, 27 participants completed both baseline and follow-up surveys and had a baseline goal of controlled drinking. One participant completed the follow-up survey over 5 months after completing the baseline survey and had several outlier values. Given this participant was identified as an outlier, we removed their data from the sample. A second participant was removed from the sample due to missingness on the BAC predictor variable, resulting in a final sample of $N = 25$. Comprehensive information on the characteristics of the final sample can be found in **Table 1**. Of those who completed the follow-up survey, 6.62% of data among the final sample was missing. Missing data

analyses indicated that participants who were retained at the 3-month follow-up timepoint were not significantly different from those who were not retained on any baseline characteristic except family history of AUD. Specifically, participants who were retained at the 3-month follow-up reported more family members with a history of AUD compared to those who were not retained ($t = 2.28$, $p = 0.02$).

Participants were primarily white (84.0%), not Hispanic or Latine (88.0%), and 44.2 years old on average (SD = 11.0). Mean time between first drink and first intoxication was 2.6 years (SD = 8.4, range = 23-61), participants had an average of 1.5 family members with a history of AUD (SD = 1.3), and 36.0% of the sample endorsed financial instability at both baseline and follow-up timepoints. All participants in the sample identified as cisgender, and sex was skewed slightly female (56.0% female, 44.0% male).

Between baseline and follow-up timepoints, AUD severity ($t = -7.74$, $p < 0.01$), incentive salience ($t = -3.21$, $p < 0.01$), and executive functioning impairment decreased ($t = -4.32$, $p < 0.01$), and control over drinking increased ($t = 3.22$, $p < 0.01$). Between baseline and follow-up timepoints, there were no significant changes in self-stigma ($t = 0.99$, $p = 0.33$), financial stability ($t = 0$, $p = 1.00$), negative emotionality ($t = -1.47$, $p = 0.15$), or peak BAC ($t = -0.14$, $p = 0.90$). At follow-up, participants reported drinking 3.9 drinks per week on average (SD = 2.3). Four participants who had a controlled drinking goal at baseline reported an abstinent drinking goal at follow-up.

Bivariate Correlations

A correlation matrix of all variables included in the following models can be found in **Table 2**. Bivariate analyses revealed significant negative correlations between

age and financial stability ($r = -0.48$, $p = 0.02$), control over drinking at baseline and incentive salience ($r = -0.46$, $p = 0.02$), control over drinking at baseline and impaired executive functioning ($r = -0.58$, $p < 0.01$), and control over drinking at follow-up and AUD severity ($r = -0.44$, $p = 0.03$). Significant positive correlations between AUD severity and self-stigma ($r = 0.43$, $p = 0.03$), AUD severity and family history of a significant drinking problem ($r = 0.50$, $p = 0.01$), AUD severity and impaired executive functioning ($r = 0.48$, $p = 0.01$), incentive salience and impaired executive functioning ($r = 0.70$, $p < 0.01$), and control over drinking at baseline and at follow-up ($r = 0.49$, $p = 0.01$) were observed. All other correlations were nonsignificant ($p \geq 0.05$).

Multiple Regression Analyses

Model results can be found in **Table 3**. Regression analysis indicated that no variables were predictive of success with controlled drinking (all Holm-corrected p-values ≥ 0.05). The R-squared indicated 56.0% of the variability in success with controlled drinking was explained by the model, however only 12.0% of the variability in success with controlled drinking was explained using the adjusted R-squared value (which accounts for the number of degrees of freedom in the model). No variance inflation factor values were greater than 10, indicating multicollinearity was not present (Midi et al., 2010). While no predictors in the regression model and few bivariate correlations were significant, the directionality of several predictor-outcome relationships in the regression were consistent with bivariate correlations. The only exceptions were the correlation and regression results for self-stigma, impaired executive functioning, age lapse, and incentive salience, all of which were negatively correlated with the outcome variable in bivariate correlations but positively associated with the outcome variable in

the regression model. Given the effect was non-significantly different from 0 in either analysis, it is possible for the direction of the relationship to shift based on other variables included in the regression.

Latent Change Score Analyses

AUD severity. A bivariate latent change score model of AUD severity and control over drinking (see **Figure 1a**) revealed that lower baseline AUD severity was associated with an increase in the latent change score (i.e., more change) for control over drinking (estimate = -0.28, SE = 0.13, $p = 0.04$), but baseline control over drinking was not associated with the latent change score for AUD severity (estimate = 0.44, SE = 0.49, $p = 0.36$). Greater control over drinking at baseline was associated with a decrease in the latent change score (i.e., less change) for control over drinking (estimate = -0.66, SE = 0.13, $p < 0.01$), but greater baseline AUD severity was not associated with latent change score for AUD severity (estimate = -0.53, SE = 0.39, $p = 0.18$). In the context of control over drinking increasing across time across the total sample ($t = 3.22$, $p < 0.01$), these results indicate that participants of lower AUD severity at baseline experienced more of an increase in control over drinking during the 90-day follow-up period (see **Figure 1b**) and participants with greater control over drinking at baseline also experienced less increase in control over drinking over time (possibly representing a ceiling effect).

Incentive salience. A bivariate latent change score model of incentive salience and control over drinking (see **Figure 2**) revealed that greater baseline incentive salience was not associated latent change score for control over drinking (estimate = 0.04, SE = 0.14, $p = 0.78$), and baseline control over drinking was not associated with the latent change score for incentive salience (estimate = 0.01, SE = 0.19, $p = 0.98$). Greater control

over drinking at baseline was associated with a decrease in the latent change score for control over drinking (estimate = -0.56, SE = 0.14, $p < 0.01$), and greater baseline incentive salience was associated with a decrease in the latent change score for incentive salience (estimate = -0.57, SE = 0.14, $p < 0.01$). These results indicate that participants with greater control over drinking at baseline experienced less change in control over drinking during the 90-day follow-up period and those with greater incentive salience at baseline experienced less change in incentive salience.

Executive functioning. A bivariate latent change score model of executive functioning and control over drinking (see **Figure 3**) revealed that baseline executive functioning was not associated with the latent change score for control over drinking (estimate = 0.04, SE = 0.18, $p = 0.82$), and baseline control over drinking was not associated with the latent change score for executive functioning (estimate = -0.02, SE = 0.14, $p = 0.89$). However, greater control over drinking at baseline was associated with a decrease in the latent change score for control over drinking (estimate = -0.56, SE = 0.16, $p < 0.01$), and more impaired baseline executive functioning was associated with a decrease in the latent change score for executive functioning (estimate = -0.77, SE = 0.15, $p < 0.01$). These results indicate that participants with greater control over drinking at baseline experienced less change in control over drinking during the 90-day follow-up period and those with more impaired executive functioning experienced less change in executive functioning.

Since a paired t-test did not reveal significant changes in peak BAC from baseline to follow-up, piecewise latent growth curve analyses were run as a sensitivity analysis to

probe possible non-linear trajectories of peak BAC over the 3-month data collection period.

Latent Growth Curve Analyses

Table 4a and **Figure 4** detail 90-day latent growth curve changes in peak BAC for participants with at least 90 days of BAC data (n = 4) and results indicated that peak BAC did not change significantly over the first 14 days of treatment (p = 0.11); however, between days 15 and 90 of treatment, peak BAC linearly decreased (p = 0.02). Significant variance was found only in trajectories of peak BAC between days 15 and 90 of treatment (p = 0.02). **Table 4b** and **Figure 5** show 90-day latent growth curve changes in peak BAC by gender. When controlling for gender, peak BAC did not change significantly during treatment. In addition, trajectories of peak BAC did not significantly differ between men and women in the first 14 days of treatment (p = 0.683) or between days 15 and 90 of treatment (p = 0.415). Significant variance was found only in trajectories of peak BAC between days 15 and 90 of treatment (p = 0.01).

Smaller Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) values, which account for model complexity and measure model fit, were found in the gender-moderated latent growth curve analysis (AIC = -5626.2, BIC = -5557.3) compared to the latent growth curve analysis of the general sample (AIC = -7394.2, BIC = -7338.2). This indicates that the gender-moderated latent growth curve analysis was a better-fitting model than that of the general sample.

Discussion

Many people with AUD do not desire abstinence (Grant et al., 2015; Probst et al., 2015), and non-abstinent recovery is both attainable and sustainable (Hasin et al., 2017;

Mann et al., 2017; Tucker & Witkiewitz, 2022; Witkiewitz et al., 2017, 2018, 2019, 2020, 2021; Witkiewitz & Tucker, 2020). However, among trainees and clinicians, abstinence continues to often be considered the only acceptable goal for people with AUD (Davis et al., 2017; Davis & Lauritsen, 2016; Davis & Rosenberg, 2013). To help correct potential misconceptions about non-abstinent recovery (e.g., that this type of goal is not appropriate for people with severe AUD) and to empower individuals with AUD with knowledge relevant to their goal setting, the current project sought to examine predictors of success with controlled drinking in a sample of adults enrolled in a telehealth AUD treatment platform with baseline controlled drinking goals ($N = 25$).

The current project collected data from Ria, a telehealth treatment platform for AUD inclusive of controlled drinking goals, to explore predictors of success with controlled drinking outcomes. Despite best efforts, not enough participants were recruited to detect differences in missing data or to sufficiently power our models. As such, missing data analyses and model results must be interpreted with extreme caution. We conducted a multiple regression analysis and found that success in controlled drinking was not predicted by age, sex, AUD severity, self-stigma, family history of AUD, financial stability, time between age of first drink and first intoxication, incentive salience, negative emotionality, executive functioning, baseline alcohol consumption, or baseline control over drinking. To explore potential dynamic relationships between baseline predictors that showed change over time (i.e., AUD severity, incentive salience, and executive functioning) and control over drinking, we ran bivariate latent change score models. In line with our hypothesis that participants with less severe AUD would show greater success with controlled drinking, results indicated that participants of lower

AUD severity at baseline experienced greater change in control over drinking during the 90-day follow-up period. Additionally, participants with greater control over drinking, those with greater incentive salience, and those with more impaired executive functioning at baseline experienced less change in each construct over time. Finally, latent growth curve models tested possible non-linear trajectories of peak BAC and found a significant decrease in peak blood alcohol concentration between days 15 and 90 and no moderation of peak BAC trajectory by gender.

Missing data analyses indicated that participants retained at follow-up were only significantly different from those not retained on one variable, family history of AUD. People with a family history of AUD may be more familiar with alcohol-related consequences and, as a result, more motivated to stay in treatment. Alternatively, those with a family history of AUD may find inspiration in recovered family members that motivates them to stay in treatment and/or social support from family members in recovery that hold them accountable to staying in treatment.

Although no predictors in the regression model were significant, AUD severity, family history, and age lapse predictors were directionally aligned with our hypotheses (i.e., that those with less severe AUD, those with fewer family members with a significant drinking problem, and those with a greater gap in between first alcoholic drink and first intoxication would show greater success with controlled drinking). Conversely, while we predicted participants of younger age, those who were more financially stable, those with less self-stigma, those who scored lower on negative emotionality and incentive salience, and those with less impaired executive function to show greater success with controlled drinking, the directionality of the predictors in the regression

model (non-significantly) pointed to the opposite. It is plausible that the lack of statistical power in our sample may be the reason behind at least some of these differences in directionality. The measly R-squared value in our model suggests that variation in success with controlled drinking is not well-explained by our predictors and supports the notion that caution is warranted in drawing inferences from model results. A low R-squared value can be attributed to lack of statistical power, measurement error, and missing relevant predictors. Additionally, success with controlled drinking may be non-linearly related to our predictor variables.

The decision to measure success with controlled drinking via the extent to which participants felt control over their drinking instead of perceived success in treatment as originally planned was necessary due to lack of variability in the perceived success variable. **Table 5** presents a cross-tabulation of the planned versus employed outcome variable. While perceived success and control over drinking at follow-up were significantly correlated ($r = 0.53$, $p < 0.01$); a paired t-test indicated a significant mean difference such that participants reported greater control over drinking at follow-up compared to perceived success ($t = 6.37$, $p < 0.01$). These results indicate that perceived success and control over drinking are related but different constructs. While all participants in our sample had a controlled drinking goal, some participants may not have defined their success by degree of control over drinking. Indeed, in an open-response question asking how participants define success with their drinking goal, several participants did not define success by level of control over drinking, but rather, by improvements in quality of life, personal health, or social relationships. These participants may, thus, have reported lower perceived success and greater control over

drinking. While there is unique value to studying perceived success, had we employed a variable lacking variability as the outcome variable in the multiple regression, the fit of the model would have been compromised, and estimates may have been inflated. Regression analysis relies on an assumption that all variables in the model are normally distributed—to have used the perceived success variable as our outcome variable would have violated this assumption and rendered any inferences from our model results invalid. Of note, 16% of our sample changed to an abstinent treatment goal over the 90-day follow-up period. These participants may have reported perceived success; however, their success does not reflect a controlled drinking outcome. By operationalizing success in the present study as degree of control over drinking, our model accounted for the participants who, at follow-up, may have defined success by abstinence.

A bivariate correlation showed that higher AUD severity at baseline was associated with less control over drinking at follow-up. This reflects prior research that has found an external locus of control to be associated with poorer AUD treatment outcomes (Koski-Jännes, 1994). Moreover, less control over drinking at baseline was associated with both greater incentive salience and more impaired executive functioning, which indicates impairments across several cognitive domains, indicating that impaired control over drinking may be a symptom of both heightened incentive salience and impaired executive functioning among individuals with AUD. Participants with more severe AUD reported higher levels of self-stigma, a result consistent with extant literature that has found alcohol severity and self-stigma to be significantly, positively correlated (Dearing et al., 2005). Additionally, participants with more severe AUD reported, on average, more extensive family history of a significant drinking problem. Again, this

result is not surprising, as prior literature has consistently found family history of AUD to be positively associated with AUD severity (Gleeson et al., 2009; Milne et al., 2009; Palaniappan et al., 2016). The connection between family history of AUD and AUD severity may be genetic and environmental in nature. While research has yet to probe the context underlying this association, individuals with AUD and with a family history of AUD have decreased binding of the 5-Hydroxytryptophan ($5\text{-}HT_{2A}$) receptor in their prefrontal cortex compared to those without a family history of AUD (Underwood et al., 2008), a result indicative of a genetic predisposition for AUD. Further, Keenan and colleagues have found individuals with AUD who have had a first-degree relative with AUD to demonstrate greater neuropsychological impairment and theorize that people with family history of AUD may have a neurological predisposition to AUD (Keenan et al., 1997). Participants with more severe AUD also reported more impaired executive functioning; and those with more impaired executive functioning reported higher levels of incentive salience. These results align with research that has found a positive relationship between impaired executive functioning and AUD severity and between impaired executive functioning and heightened incentive salience (Day et al., 2015; Glass et al., 2009; Kwako et al., 2019). However, the directionality of these associations (i.e., to what extent impaired executive functioning and heightened incentive salience cause versus result from AUD) have yet to be elucidated. Finally, a bivariate correlation found older participants in the sample to be more financially stable, a result that is consistent with the logic that older adults have had more years to accumulate savings. In addition, older adults have had more time to acquire seniority in the workplace, and consequently, may possess greater earning power.

Paired t-tests indicated that AUD severity and incentive salience decreased over the 90-day follow-up period and control over drinking and executive functioning improved. Latent growth curve models also indicated a decrease in BAC over time, and a significant correlation was found between control over drinking at baseline and at follow-up. These findings point to the efficacy of Ria in treating individuals with AUD and controlled drinking goals. These findings support a wealth of existing evidence that non-abstinent recovery from AUD is both attainable and associated with significant improvements in functioning (Hasin et al., 2017; Mann et al., 2017; Tucker & Witkiewitz, 2022; Witkiewitz et al., 2017, 2018, 2019, 2020, 2021; Witkiewitz & Tucker, 2020).

Bivariate latent change score models of AUD severity, incentive salience, and executive functioning (baseline predictors that showed change over time) with control over drinking were run to further probe these relationships and account for measurement error. Bivariate latent change score models measure cross-domain couplings (the extent to which change in the latent change score of one variable is a function of the other variable at baseline) and autoregressive effects (the extent to which change in the latent change score of one variable is a function of that variable at baseline). The only significant cross-domain coupling found was that of AUD severity and control over drinking. That is, greater AUD severity at baseline was associated with a decrease in the latent change score for control over drinking during the 90-day follow-up period. While this model was underpowered, and therefore, inferences cannot be drawn, this finding aligns with our hypothesis that participants with less severe AUD would show greater success with controlled drinking and with prior research that has found high AUD

severity to be associated with less improvement during AUD treatment (e.g., Morley et al., 2006). Regarding autoregressive effects, greater control over drinking at baseline was associated with a decrease in the latent change score for control over drinking, and the same was true for incentive salience and executive functioning. With sufficient power, these results would suggest that participants with greater control over drinking at baseline experienced less change in control over drinking during the 90-day follow-up period, those with greater incentive salience at baseline experienced less change in incentive salience, and those with more impaired executive functioning experienced less change in executive functioning. While individuals with greater control over drinking at baseline would have less room to improve over treatment and those with higher AUD severity at baseline, greater incentive salience, and more impaired executive functioning could benefit more from treatment, because our sample was severely underpowered for bivariate latent change score models, we cannot draw these inferences.

To replicate the results of Hallgren et al. (2022), we ran piecewise latent growth curve models of peak BAC trajectories on participants with 90 days of BAC data. Given that success with controlled drinking may be non-linearly related to our predictor variables (as indicated by our low R-squared value), this approach allowed us to probe possible non-linear trajectories of peak BAC. Results demonstrated that peak BAC linearly decreased between days 15 and 90 of treatment; however, this result disappeared when controlling for gender. Importantly, although gender did not moderate trajectories of peak BAC, the fit statistics indicated that the gender-moderated latent growth curve analysis better fit the data, suggesting that gender is playing a role in the model. These findings are based on just 4 participants and therefore are not generalizable.

In addition to the omnipresent issue of statistical power, few participants had 90 days of BAC data, and the study sample lacked racial and socioeconomic diversity, further limiting the interpretability and generalizability of study findings. Initial response rate was also low despite that the baseline survey was present on the Ria dashboard (the homepage on the telehealth app) of each participant who had opted-in to the study until they completed the survey. Had we offered a greater financial incentive, our initial response rate might have been higher (Abdelazeem et al., 2022). While retention at follow-up appears low in relation to standards of in-person research (Abshire et al., 2017), internet-based surveys are well-documented as having significantly lower response and retention rates than in-person research, and the present study had a retention rate higher than comparable online trials (Anguera et al., 2016; Murray et al., 2009). Indeed, all participants were prompted between six and seven times (at least three of which via their indicated preferred method of contact) to complete the follow-up survey. That said, prior studies have found internet survey respondents to be more highly educated, healthier, and less likely to be Black or Hispanic compared to mail-in survey respondents (Anguera et al., 2016; Price et al., 2022). As such, inclusion of a mail-in survey option might have bolstered both sample diversity and response rates. Moreover, participation in the study was voluntary, and people who volunteered to participate may be characteristically different from the general population of individuals seeking treatment for AUD. Further, all study variables besides for BAC were collected only at baseline and/or at a 3-month follow-up; however, nonlinear, dynamic relationships between study variables have been theorized to exist (Witkiewitz & Marlatt, 2004).

Strengths of the present study include that our sample was relatively balanced with respect to sex, we tested a wide breadth of predictors evidenced to be associated with success in controlled drinking, and we explored predictors of success with controlled drinking in the context of a treatment platform supportive of controlled drinking goals. Future research ought to attempt replication of these results in a larger and more sociodemographically diverse sample, explore the extent to which results differ depending on how success with controlled drinking is operationalized, and investigate how people with lived experience of AUD define success with controlled drinking goals. Future work should also test the directionality of correlations between study variables and nonlinear relationships between study variables. Through this work, we may better understand the nature of controlled drinking goals, for whom they are most appropriate, and how and when variables such as self-stigma, family history, incentive salience, and executive functioning exert influence on treatment outcomes.

References

Abdelazeem, B., Abbas, K. S., Amin, M. A., El-Shahat, N. A., Malik, B., Kalantary, A., & Eltobgy, M. (2022). The effectiveness of incentives for research participation: A systematic review and meta-analysis of randomized controlled trials. *PLoS ONE*, *17*(4), e0267534. https://doi.org/10.1371/journal.pone.0267534

Abshire, M., Dinglas, V. D., Cajita, M. I. A., Eakin, M. N., Needham, D. M., & Himmelfarb, C. D. (2017). Participant retention practices in longitudinal clinical research studies with high retention rates. *BMC Medical Research Methodology*, *17*, 30. https://doi.org/10.1186/s12874-017-0310-z

Adalbjarnardottir, S., & Rafnsson, F. D. (2001). Perceived control in adolescent substance use: Concurrent and longitudinal analyses. *Psychology of Addictive Behaviors*, *15*(1), 25–32. https://doi.org/10.1037/0893-164X.15.1.25

Adam, A., Faouzi, M., Gaume, J., Gmel, G., Daeppen, J.-B., & Bertholet, N. (2011). Age of first alcohol intoxication: Association with risky drinking and other substance use at the age of 20. *Swiss Medical Weekly*, *141*, w13226. https://doi.org/10.4414/smw.2011.13226

Adamson, S. J., Heather, N., Morton, V., Raistrick, D., & on behalf of the UKATT Research Team. (2010). Initial preference for drinking goal in the treatment of alcohol problems: II. treatment outcomes. *Alcohol and Alcoholism*, *45*(2), 136–142. https://doi.org/10.1093/alcalc/agq005

Adamson, S. J., Sellman, J. D., & Frampton, C. M. A. (2009). Patient predictors of alcohol treatment outcome: A systematic review. *Journal of Substance Abuse Treatment*, *36*(1), 75–86. https://doi.org/10.1016/j.jsat.2008.05.007

Aickin, M., & Gensler, H. (1996). Adjusting for multiple testing when reporting research results: The Bonferroni vs Holm methods. *American Journal of Public Health*, *86*(5), 726–728. https://doi.org/10.2105/ajph.86.5.726

Al-Khalil, K., Vakamudi, K., Witkiewitz, K., & Claus, E. D. (2021). Neural correlates of alcohol use disorder severity among nontreatment-seeking heavy drinkers: An examination of the incentive salience and negative emotionality domains of the alcohol and addiction research domain criteria. *Alcoholism, Clinical and Experimental Research*, *45*(6), 1200–1214. https://doi.org/10.1111/acer.14614

Anguera, J. A., Jordan, J. T., Castaneda, D., Gazzaley, A., & Areán, P. A. (2016). Conducting a fully mobile and randomised clinical trial for depression: Access, engagement and expense. *BMJ Innovations*, *2*(1), 14–21. https://doi.org/10.1136/bmjinnov-2015-000098

Anhang Price, R., Quigley, D. D., Hargraves, J. L., Sorra, J., Becerra-Ornelas, A. U., Hays, R. D., Cleary, P. D., Brown, J., & Elliott, M. N. (2022). A systematic review of strategies to enhance response rates and representativeness of patient experience surveys. *Medical Care*, *60*(12), 910. https://doi.org/10.1097/MLR.0000000000001784

Berridge, K. C., & Robinson, T. E. (2016). Liking, wanting and the incentive-sensitization theory of addiction. *The American Psychologist*, *71*(8), 670–679. https://doi.org/10.1037/amp0000059

Blume, A. W., Schmaling, K. B., & Marlatt, A. G. (2003). Predictors of change in binge drinking over a 3-month period. *Addictive Behaviors*, *28*(5), 1007–1012. https://doi.org/10.1016/s0306-4603(01)00287-8

Boness, C. L., Votaw, V. R., Francis, M. W., Watts, A. L., Sperry, S. H., Kleva, C. S., Nellis, L., McDowell, Y., Douaihy, A. B., Sher, K. J., & Witkiewitz, K. (2022). Alcohol use disorder conceptualizations and diagnoses reflect their sociopolitical context. *Addiction Research & Theory*, 1–6. https://doi.org/10.1080/16066359.2022.2150935

Bozdağ, N., & Çuhadar, D. (2022). Internalized stigma, self-efficacy and treatment motivation in patients with substance use disorders. *Journal of Substance Use*, *27*(2), 174–180. https://doi.org/10.1080/14659891.2021.1916846

Buchmann, A. F., Schmid, B., Blomeyer, D., Becker, K., Treutlein, J., Zimmermann, U. S., Jennen-Steinmetz, C., Schmidt, M. H., Esser, G., Banaschewski, T., Rietschel, M., Schumann, G., & Laucht, M. (2009). Impact of age at first drink on vulnerability to alcohol-related problems: Testing the marker hypothesis in a prospective study of young adults. *Journal of Psychiatric Research*, *43*(15), 1205–1212. https://doi.org/10.1016/j.jpsychires.2009.02.006

Cohen, J. (1988). Statistical power analysis for the behavioral sciences. (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.

Cohen, E., Feinn, R., Arias, A., & Kranzler, H. R. (2007). Alcohol treatment utilization: Findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Drug and Alcohol Dependence*, *86*(2–3), 214–221. https://doi.org/10.1016/j.drugalcdep.2006.06.008

Collins, S. E. (2016). Associations between socioeconomic factors and alcohol outcomes. *Alcohol Research : Current Reviews*, *38*(1), 83–94.

COMBINE Study Research Group (2003). *The Combined Pharmacotherapies and*

Behavioral Interventions (COMBINE) study cookbook. National Institute on Alcohol Abuse and Alcoholism.

Crapanzano, K. A., Hammarlund, R., Ahmad, B., Hunsinger, N., & Kullar, R. (2019). The association between perceived stigma and substance use disorder treatment outcomes: A review. *Substance Abuse and Rehabilitation*, *10*, 1–12. https://doi.org/10.2147/SAR.S183252

Cronbach, L. J., & Furby, L. (1970). How we should measure "change": Or should we? *Psychological Bulletin*, *74*(1), 68–80. https://doi.org/10.1037/h0029382

Curran, P. J., Obeidat, K., & Losardo, D. (2010). Twelve frequently asked questions about growth curve modeling. *Journal of Cognition and Development : Official Journal of the Cognitive Development Society*, *11*(2), 121–136. https://doi.org/10.1080/15248371003699969

Davis, A. K., & Lauritsen, K. J. (2016). Acceptability of non-abstinence goals among students enrolled in addiction studies programs across the United States. *Substance Abuse*, *37*(1), 204–208. https://doi.org/10.1080/08897077.2015.1015702

Davis, A. K., & Rosenberg, H. (2013). Acceptance of non-abstinence goals by addiction professionals in the United States. *Psychology of Addictive Behaviors*, *27*, 1102–1109. https://doi.org/10.1037/a0030563

Davis, A. K., Rosenberg, H., & Rosansky, J. A. (2017a). American counselors' acceptance of non-abstinence outcome goals for clients diagnosed with co-occurring substance use and other psychiatric disorders. *Journal of Substance Abuse Treatment*, *82*, 29–33. https://doi.org/10.1016/j.jsat.2017.08.006

Davis, A. K., Rosenberg, H., & Rosansky, J. A. (2017b). American counselors' acceptance of non-abstinence outcome goals for clients diagnosed with co-occurring substance use and other psychiatric disorders. *Journal of Substance Abuse Treatment*, *82*, 29–33. https://doi.org/10.1016/j.jsat.2017.08.006

Davis, C. N., Slutske, W. S., Piasecki, T. M., Martin, N. G., & Lynskey, M. T. (2020). Comparing the potential causal influence of two indicators of early alcohol use on later alcohol use disorder symptoms. *Journal of Abnormal Psychology*, *129*(3), 256–265. https://doi.org/10.1037/abn0000474

Day, A., W. Kahler, C., C. Ahern, D., & S. Clark, U. (2015). Executive functioning in alcohol use studies: A brief review of findings and challenges in assessment. *Current Drug Abuse Reviews*, *8*(1), 26–40.

Dearing, R. L., Stuewig, J., & Tangney, J. P. (2005). On the importance of distinguishing shame from guilt: Relations to problematic alcohol and drug use. *Addictive Behaviors*, *30*(7), 1392–1404. https://doi.org/10.1016/j.addbeh.2005.02.002

DeMartini, K. S., Gueorguieva, R., Pearlson, G., Krishnan-Sarin, S., Anticevic, A., Ji, L. J., Krystal, J. H., & O'Malley, S. S. (2021). Mapping data-driven individualized neurobehavioral phenotypes in heavy alcohol drinkers. *Alcoholism, Clinical and Experimental Research*, *45*(4), 841–853. https://doi.org/10.1111/acer.14580

DeWit, D. J., Adlaf, E. M., Offord, D. R., & Ogborne, A. C. (2000). Age at first alcohol use: A risk factor for the development of alcohol disorders. *American Journal of Psychiatry*, *157*(5), 745–750. https://doi.org/10.1176/appi.ajp.157.5.745

Drossel, G., Brucar, L., Rawls, E., & Zilverstand, A. (2022). Subtypes in addiction and their neurobehavioral impairments in approach behavior, negative emotionality

and executive function. *Biological Psychiatry*, *91*(9, Supplement), S99–S100. https://doi.org/10.1016/j.biopsych.2022.02.265

Eng, M. Y., Schuckit, M. A., & Smith, T. L. (2005). The level of response to alcohol in daughters of alcoholics and controls. *Drug and Alcohol Dependence*, *79*(1), 83–93. https://doi.org/10.1016/j.drugalcdep.2005.01.002

Enggasser, J. L., Hermos, J. A., Rubin, A., Lachowicz, M., Rybin, D., Brief, D. J., Roy, M., Helmuth, E., Rosenbloom, D., & Keane, T. M. (2015). Drinking goal choice and outcomes in a web-based alcohol intervention: Results from VetChange. *Addictive Behaviors*, *42*, 63–68.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175–191. https://doi.org/10.3758/bf03193146

Glass, J. M., Buu, A., Adams, K. M., Nigg, J. T., Puttler, L. I., Jester, J. M., & Zucker, R. A. (2009). Effects of alcoholism severity and smoking on executive neurocognitive function. *Addiction*, *104*(1), 38–48. https://doi.org/10.1111/j.1360-0443.2008.02415.x

Gleeson, D., Jones, J. S., McFarlane, E., Francis, R., Gellion, C., Bradley, M. P., & Peck, R. J. (2009). Severity of alcohol dependence in decompensated alcoholic liver disease: Comparison with heavy drinkers without liver disease and relationship to family drinking history. *Alcohol and Alcoholism*, *44*(4), 392–397. https://doi.org/10.1093/alcalc/agp008

Gordon, N. P., Shanks, C. B., & Grant, R. W. (2023). Social risks, social needs, and attitudes toward social health screening 1 year into the COVID-19 pandemic: survey of adults in an integrated health care delivery system. *The Permanente Journal*, *27*(2), 61–74. https://doi.org/10.7812/TPP/22.142

Graber, R. A., & Miller, W. R. (1988). Abstinence or controlled drinking goals for problem drinkers: A randomized clinical trial. *Psychology of Addictive Behaviors*, *2*(1), 20–33. https://doi.org/10.1037/h0080474

Grant, B. F., Goldstein, R. B., Saha, T. D., Chou, S. P., Jung, J., Zhang, H., Pickering, R. P., Ruan, W. J., Smith, S. M., Huang, B., & Hasin, D. S. (2015). Epidemiology of DSM-5 Alcohol Use Disorder. *JAMA Psychiatry*, *72*(8), 757–766. https://doi.org/10.1001/jamapsychiatry.2015.0584

Grant, J. D., Scherrer, J. F., Lynskey, M. T., Lyons, M. J., Eisen, S. A., Tsuang, M. T., True, W. R., & Bucholz, K. K. (2006). Adolescent alcohol use is a risk factor for adult alcohol and drug dependence: Evidence from a twin design. *Psychological Medicine*, *36*(1), 109–118. https://doi.org/10.1017/S0033291705006045

Grittner, U., Kuntsche, S., Graham, K., & Bloomfield, K. (2012). Social inequalities and gender differences in the experience of alcohol-related problems. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *47*(5), 597–605. https://doi.org/10.1093/alcalc/ags040

Hagman, B. T., Falk, D., Litten, R., & Koob, G. F. (2022). Defining recovery from Alcohol Use Disorder: Development of an NIAAA research definition. *American Journal of Psychiatry*, *179*(11), 807–813. https://doi.org/10.1176/appi.ajp.21090963

Hall, W. (2010). What are the policy lessons of national alcohol prohibition in the United States, 1920–1933? *Addiction*, *105*(7), 1164–1173. https://doi.org/10.1111/j.1360-0443.2010.02926.x

Hallgren, K. A., Galloway, G. P., Witkiewitz, K., Linde, P., Nix, B., & Mendelson, J. E. (2023). Treatment retention and reductions in blood alcohol concentration (BAC) during the first 90 days of a telehealth program for alcohol use disorder. *The American Journal of Drug and Alcohol Abuse*, 1–11. https://doi.org/10.1080/00952990.2023.2175322

Hallgren, K. A., Matson, T. E., Oliver, M., Caldeiro, R. M., Kivlahan, D., & Bradley, K. A. (2022). Practical assessment of DSM-5 alcohol use disorder criteria in routine care: High test-retest reliability of an Alcohol Symptom Checklist. *Alcoholism, Clinical and Experimental Research*, *46*(3), 458–467. https://doi.org/10.1111/acer.14778

Hammarlund, R., Crapanzano, K., Luce, L., Mulligan, L., & Ward, K. (2018). Review of the effects of self-stigma and perceived social stigma on the treatment-seeking decisions of individuals with drug- and alcohol-use disorders. *Substance Abuse and Rehabilitation*, *9*, 115–136. https://doi.org/10.2147/SAR.S183256

Hasin, D. S., Wall, M., Witkiewitz, K., Kranzler, H. R., Falk, D., Litten, R., Mann, K., O'Malley, S. S., Scodes, J., Robinson, R. L., Anton, R., Fertig, J., Isenberg, K., McCann, D., Meulien, D., Meyer, R., O'Brien, C., Ryan, M., Silverman, B., … Zakine, B. (2017). Change in non-abstinent WHO drinking risk levels and alcohol dependence: A 3 year follow-up study in the US general population. *The Lancet Psychiatry*, *4*(6), 469–476. https://doi.org/10.1016/S2215-0366(17)30130-X

Henry, K. L., McDonald, J. N., Oetting, E. R., Walker, P. S., Walker, R. D., & Beauvais, F. (2011). Age of onset of first alcohol intoxication and subsequent alcohol use among urban american indian adolescents. *Psychology of Addictive Behaviors : Journal of the Society of Psychologists in Addictive Behaviors*, *25*(1), 48–56. https://doi.org/10.1037/a0021710

Hingson, R. W., Heeren, T., & Winter, M. R. (2006). Age at drinking onset and alcohol dependence: Age at onset, duration, and severity. *Archives of Pediatrics & Adolescent Medicine*, *160*(7), 739–746. https://doi.org/10.1001/archpedi.160.7.739

Hochberg, Y. (1988). A sharper Bonferroni procedure for multiple tests of significance. *Biometrika*, *75*(4), 800–802. https://doi.org/10.1093/biomet/75.4.800

Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics*, *6*(2), 65–70.

Jakubczyk, A., Trucco, E. M., Kopera, M., Kobyliński, P., Suszek, H., Fudalej, S., Brower, K. J., & Wojnar, M. (2018). The association between impulsivity, emotion regulation, and symptoms of alcohol use disorder. *Journal of Substance Abuse Treatment*, *91*, 49–56. https://doi.org/10.1016/j.jsat.2018.05.004

Johnson, B. A., Ait-Daoud, N., & Prihoda, T. J. (2000). Combining Ondansetron and Naltrexone effectively treats biologically predisposed alcoholics: from hypotheses to preliminary clinical evidence. *Alcohol: Clinical and Experimental Research*, *24*(5), 737–742. https://doi.org/10.1111/j.1530-0277.2000.tb02048.x

Kahler, C. W., Epstein, E. E., & McCrady, B. S. (1995). Loss of control and inability to abstain: The measurement of and the relationship between two constructs in male

alcoholics. *Addiction*, *90*(8), 1025–1036. https://doi.org/10.1046/j.1360-0443.1995.90810252.x

Keenan, E., O'Donnell, C., Sinanan, K., & O'Callaghan, E. (1997). Severity of alcohol dependence and its relationship to neurological soft signs, neuropsychological impairment and family history. *Acta Psychiatrica Scandinavica*, *95*(4), 272–276. https://doi.org/10.1111/j.1600-0447.1997.tb09631.x

Khemiri, L., Franck, J., & Jayaram-Lindström, N. (2022). Effect of alcohol use disorder family history on cognitive function. *Psychological Medicine*, *52*(4), 757–769. https://doi.org/10.1017/S003329172000238X

Koski-Jännes, A. (1994). Drinking-related locus of control as a predictor of drinking after treatment. *Addictive Behaviors*, *19*(5), 491–495. https://doi.org/10.1016/0306-4603(94)90004-3

Kuerbis, A., Morgenstern, J., & Hail, L. (2012). Predictors of moderated drinking in a primarily alcohol dependent sample of men who have sex with men. *Psychology of Addictive Behaviors : Journal of the Society of Psychologists in Addictive Behaviors*, *26*(3), 484–495. https://doi.org/10.1037/a0026713

Kuerbis, A. N., Shao, S., Padovano, H. T., Jadanova, A., Kumar, D. S., Vitale, R., Nitzburg, G., Vadhan, N. P., & Morgenstern, J. (2020). Context and craving among individuals with Alcohol Use Disorder attempting to moderate their drinking. *Experimental and Clinical Psychopharmacology*, 10.1037/pha0000349. https://doi.org/10.1037/pha0000349

Kuntsche, E., Rossow, I., Engels, R., & Kuntsche, S. (2016). Is “age at first drink” a useful concept in alcohol research and prevention? We doubt that. *Addiction (Abingdon, England)*, *111*(6), 957–965. https://doi.org/10.1111/add.12980

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest package: tests in linear mixed effects models. *Journal of Statistical Software*, *82*, 1–26. https://doi.org/10.18637/jss.v082.i13

Kwako, L. E., Momenan, R., Litten, R. Z., Koob, G. F., & Goldman, D. (2016). Addictions neuroclinical assessment: A neuroscience-based framework for addictive disorders. *Biological Psychiatry*, *80*(3), 179–189. https://doi.org/10.1016/j.biopsych.2015.10.024

Kwako, L. E., Schwandt, M. L., Ramchandani, V. A., Diazgranados, N., Koob, G. F., Volkow, N. D., Blanco, C., & Goldman, D. (2019). Neurofunctional domains derived from deep behavioral phenotyping in Alcohol Use Disorder. *American Journal of Psychiatry*, *176*(9), 744–753. https://doi.org/10.1176/appi.ajp.2018.18030357

Lance, C. E., Meade, A. W., & Williamson, G. M. (2000). We should measure change-and here’s how. In G. M. Williamson, D. R. Shaffer, & P. A. Parmelee (Eds.), *Physical Illness and Depression in Older Adults: A Handbook of Theory, Research, and Practice* (pp. 201–235). https://doi.org/10.1007/0-306-47178-7_10

Leeman, R. F., Patock-Peckham, J. A., & Potenza, M. N. (2012). Impaired control over alcohol use: An under-addressed risk factor for problem drinking in young adults? *Experimental and Clinical Psychopharmacology*, *20*(2), 92–106. https://doi.org/10.1037/a0026463

Leeman, R. F., Toll, B. A., Taylor, L. A., & Volpicelli, J. R. (2009). Alcohol-induced disinhibition expectancies and impaired control as prospective predictors of problem drinking in undergraduates. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors*, *23*(4), 553–563. https://doi.org/10.1037/a0017129

Lipperman-Kreda, S., & Grube, J. W. (2019). Associations of early age of first intoxication with past year drinking contexts and problems. *Substance Use & Misuse*, *54*(7), 1146–1153. https://doi.org/10.1080/10826084.2018.1563187

Litten, R. Z., Ryan, M. L., Falk, D. E., Reilly, M., Fertig, J. B., & Koob, G. F. (2015). Heterogeneity of Alcohol Use Disorder: Understanding mechanisms to advance personalized treatment. *Alcohol: Clinical and Experimental Research*, *39*(4), 579–584. https://doi.org/10.1111/acer.12669

Luoma, J. B., Kulesza, M., Hayes, S. C., Kohlenberg, B., & Larimer, M. (2014). Stigma predicts residential treatment length for substance use disorder. *The American Journal of Drug and Alcohol Abuse*, *40*(3), 206–212. https://doi.org/10.3109/00952990.2014.901337

Luoma, J. B., Nobles, R. H., Drake, C. E., Hayes, S. C., O'Hair, A., Fletcher, L., & Kohlenberg, B. S. (2013). Self-Stigma in substance abuse: Development of a new measure. *Journal of Psychopathology and Behavioral Assessment*, *35*(2), 223–234. https://doi.org/10.1007/s10862-012-9323-4

Maimaris, W., & McCambridge, J. (2014). Age of first drinking and adult alcohol problems: Systematic review of prospective cohort studies. *Journal of*

Epidemiology and Community Health, *68*(3), 268–274. https://doi.org/10.1136/jech-2013-203402

Mann, K., Aubin, H., Charlet, K., & Witkiewitz, K. (2017). Can reduced drinking be a viable goal for alcohol dependent patients? *World Psychiatry*, *16*(3), 325–326. https://doi.org/10.1002/wps.20476

Mann, K., Hermann, D., & Heinz, A. (2000). One hundred years of alcoholism: The twentieth century. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *35*(1), 10–15. https://doi.org/10.1093/alcalc/35.1.10

McAuliffe, W. E., & Dunn, R. (2004). Substance abuse treatment needs and access in the USA: Interstate variations. *Addiction (Abingdon, England)*, *99*(8), 999–1014. https://doi.org/10.1111/j.1360-0443.2004.00783.x

Midi, H., Sarkar, S. K., & Rana, S. (2010). Collinearity diagnostics of binary logistic regression model. *Journal of Interdisciplinary Mathematics, 13*(3), 253-267.

Miller, W.R. & Marlatt., G.A. (1984). *Manual for the Comprehensive Drinker Profile.* Odessa, FL; Psychological Assessment Resources.

Milne, B. J., Caspi, A., Harrington, H., Poulton, R., Rutter, M., & Moffitt, T. E. (2009). Predictive value of family history on severity of illness: The case for depression, anxiety, alcohol dependence, and drug dependence. *Archives of General Psychiatry*, *66*(7), 738–747. https://doi.org/10.1001/archgenpsychiatry.2009.55

Monterosso, J. R., Flannery, B. A., Pettinati, H. M., Oslin, D. W., Rukstalis, M., O'Brien, C. P., & Volpicelli, J. R. (2001). Predicting treatment response to Naltrexone: The influence of craving and family history. *The American Journal on Addictions*, *10*(3), 258–268. https://doi.org/10.1080/105504901750532148

Morean, M. E., Corbin, W. R., & Fromme, K. (2012). Age of first use and delay to first intoxication in relation to trajectories of heavy drinking and alcohol-related problems during emerging adulthood. *Alcoholism, Clinical and Experimental Research*, *36*(11), 1991–1999. https://doi.org/10.1111/j.1530-0277.2012.01812.x

Morean, M. E., Kong, G., Camenga, D. R., Cavallo, D. A., Connell, C., & Krishnan-Sarin, S. (2014). First drink to first drunk: Age of onset and delay to intoxication are associated with adolescent alcohol use and binge drinking. *Alcoholism, Clinical and Experimental Research*, *38*(10), 2615–2621. https://doi.org/10.1111/acer.12526

Morley, K. C., Teesson, M., Reid, S. C., Sannibale, C., Thomson, C., Phung, N., Weltman, M., Bell, J. R., Richardson, K., & Haber, P. S. (2006). Naltrexone versus acamprosate in the treatment of alcohol dependence: A multi-centre, randomized, double-blind, placebo-controlled trial. *Addiction*, *101*(10), 1451–1462. https://doi.org/10.1111/j.1360-0443.2006.01555.x

Moshagen, M. (2023). *Power Analysis for Structural Equation Models: semPower Manual*.

Murray, E., Khadjesari, Z., White, I. R., Kalaitzaki, E., Godfrey, C., McCambridge, J., Thompson, S. G., & Wallace, P. (2009). Methodological challenges in online trials. *Journal of Medical Internet Research*, *11*(2), e9. https://doi.org/10.2196/jmir.1052

Nagoshi, C. T. (1999). Perceived control of drinking and other predictors of alcohol use and problems in a college student sample. *Addiction Research*, *7*(4), 291–306. https://doi.org/10.3109/16066359909004388

Nandi, A., Glymour, M. M., & Subramanian, S. V. (2014). Association among socioeconomic status, health behaviors, and all-cause mortality in the united states. *Epidemiology*, *25*(2), 170–177.

Newton-Howes, G., Cook, S., Martin, G., Foulds, J. A., & Boden, J. M. (2019). Comparison of age of first drink and age of first intoxication as predictors of substance use and mental health problems in adulthood. *Drug and Alcohol Dependence*, *194*, 238–243. https://doi.org/10.1016/j.drugalcdep.2018.10.012

Nguyen-Louie, T. T., Simmons, A. N., Squeglia, L. M., Alejandra Infante, M., Schacht, J. P., & Tapert, S. F. (2018). Earlier alcohol use onset prospectively predicts changes in functional connectivity. *Psychopharmacology*, *235*(4), 1041–1054. https://doi.org/10.1007/s00213-017-4821-4

Orford, J., & Keddie, A. (1986). Abstinence or controlled drinking in clinical practice: A test of the dependence and persuasion hypotheses. *British Journal of Addiction*, *81*(4), 495–504. https://doi.org/10.1111/j.1360-0443.1986.tb00361.x

Palaniappan, H., James, A. G. W., & Annamalai, A. (2016). Correlation between severity of alcohol dependence with age of onset and family history among urban alcoholics: A cross-sectional study. *Journal of Evolution of Medical and Dental Sciences*, *5*(52), 3452–3458. https://doi.org/10.14260/jemds/2016/796

Probst, C., Manthey, J., Martinez, A., & Rehm, J. (2015). Alcohol Use Disorder severity and reported reasons not to seek treatment: A cross-sectional study in European primary care practices. *Substance Abuse Treatment, Prevention, and Policy*, *10*, 32. https://doi.org/10.1186/s13011-015-0028-z

R Core Team (2022). *R: A language and environment for statistical computing.* R

Foundation for Statistical Computing, Vienna, Austria. https://www.R-project.org/.

Rhodes, T., & Clinkinbeard, S. (2013). College students and binge drinking: Exploring the relationship between control and intention on behavior. *Applied Psychology in Criminal Justice*, *9*(1), 24–44.

Rohsenow, D. J., Miranda, R., McGeary, J. E., & Monti, P. M. (2007). Family history and antisocial traits moderate naltrexone's effects on heavy drinking in alcoholics. *Experimental and Clinical Psychopharmacology*, *15*(3), 272–281. https://doi.org/10.1037/1064-1297.15.3.272

Rosenberg, H. (1993). Prediction of controlled drinking by alcoholics and problem drinkers. *Psychological Bulletin*, *113*(1), 129–139. https://doi.org/10.1037/0033-2909.113.1.129

Rosenberg, H., Grant, J., & Davis, A. K. (2020). Acceptance of non-abstinence as an outcome goal for individuals diagnosed with Substance Use Disorders: A narrative review of published research. *Journal of Studies on Alcohol and Drugs*, *81*(4), 405–415. https://doi.org/10.15288/jsad.2020.81.405

Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, *48*, 1–36. https://doi.org/10.18637/jss.v048.i02

Sacks, J. J., Gonzales, K. R., Bouchery, E. E., Tomedi, L. E., & Brewer, R. D. (2015). 2010 National and state costs of excessive alcohol consumption. *American Journal of Preventive Medicine*, *49*(5), e73–e79. https://doi.org/10.1016/j.amepre.2015.05.031

Sartor, C. E., Lynskey, M. T., Bucholz, K. K., Madden, P. A. F., Martin, N. G., & Heath, A. C. (2009). Timing of first alcohol use and alcohol dependence: Evidence of common genetic influences. *Addiction (Abingdon, England)*, *104*(9), 1512–1518. https://doi.org/10.1111/j.1360-0443.2009.02648.x

Schomerus, G., & Angermeyer, M. C. (2008). Stigma and its impact on help-seeking for mental disorders: What do we know? *Epidemiologia E Psichiatria Sociale*, *17*(1), 31–37. https://doi.org/10.1017/s1121189x00002669

Schuckit, M. A. (1984). Subjective responses to alcohol in sons of alcoholics and control subjects. *Archives of General Psychiatry*, *41*(9), 879–884. https://doi.org/10.1001/archpsyc.1984.01790200061008

Schuckit, M. A., Smith, T. L., Kalmijn, J., & Danko, G. P. (2005). A cross-generational comparison of alcohol challenges at about age 20 in 40 father-offspring pairs. *Alcoholism, Clinical and Experimental Research*, *29*(11), 1921–1927. https://doi.org/10.1097/01.alc.0000187154.94681.65

Soundararajan, S., Narayanan, G., Agrawal, A., Prabhakaran, D., & Murthy, P. (2017). Relation between age at first alcohol drink & adult life drinking patterns in alcohol-dependent patients. *The Indian Journal of Medical Research*, *146*(5), 606–611. https://doi.org/10.4103/ijmr.IJMR_1363_15

Stamates, A. L., Lau-Barraco, C., & Linden-Carmichael, A. N. (2016). Alcohol Expectancies mediate the relationship between age of first intoxication and drinking outcomes in college binge drinkers. *Substance Use & Misuse*, *51*(5), 598–607. https://doi.org/10.3109/10826084.2015.1126745

Staton, C. A., Zhao, D., Ginalis, E. E., Hirshon, J. M., Sakita, F., Swahn, M. H., Mmbaga, B. T., & Nickenig Vissoci, J. R. (2020). Alcohol availability, cost, age of first drink, and its association with at-risk alcohol use in moshi, tanzania. *Alcoholism, Clinical and Experimental Research*, *44*(11), 2266–2274. https://doi.org/10.1111/acer.14457

Stein, E., & Witkiewitz, K. (2019). Trait self-control predicts drinking patterns during treatment for alcohol use disorder and recovery up to three years following treatment. *Addictive Behaviors*, *99*, 106083. https://doi.org/10.1016/j.addbeh.2019.106083

Stringer, K. L., & Baker, E. H. (2018). Stigma as a barrier to substance abuse treatment among those with unmet need: An analysis of parenthood and marital status. *Journal of Family Issues*, *39*(1), 3–27. https://doi.org/10.1177/0192513X15581659

Tucker, J. A., & Witkiewitz, K. (2022). *Dynamic pathways to recovery from alcohol use disorder: Meaning and methods*. Cambridge University Press.

Underwood, M. D., Mann, J. J., Huang, Y.-Y., & Arango, V. (2008). Family history of alcoholism is associated with lower 5-HT2A receptor binding in the prefrontal cortex. *Alcoholism: Clinical and Experimental Research*, *32*(4), 593–599. https://doi.org/10.1111/j.1530-0277.2007.00610.x

Vera, B. D. V., Pilatti, A., & Pautassi, R. M. (2020). ELSA cohort 2014: Association of age of first drink and progression from first drink to drunkenness on alcohol outcomes in Argentinean college freshmen. *The American Journal of Drug and Alcohol Abuse*, *46*(1), 58–67. https://doi.org/10.1080/00952990.2019.1608223

Verdejo-Garcia, A., Lorenzetti, V., Manning, V., Piercy, H., Bruno, R., Hester, R., Pennington, D., Tolomeo, S., Arunogiri, S., Bates, M. E., Bowden-Jones, H., Campanella, S., Daughters, S. B., Kouimtsidis, C., Lubman, D. I., Meyerhoff, D. J., Ralph, A., Rezapour, T., Tavakoli, H., … Ekhtiari, H. (2019). A roadmap for integrating neuroscience into addiction treatment: A consensus of the neuroscience interest group of the international society of addiction medicine. *Frontiers in Psychiatry*, *10*. https://www.frontiersin.org/articles/10.3389/fpsyt.2019.00877

Wallhed Finn, S., Bakshi, A.-S., & Andréasson, S. (2014). Alcohol consumption, dependence, and treatment barriers: Perceptions among nontreatment seekers with alcohol dependence. *Substance Use & Misuse*, *49*(6), 762–769. https://doi.org/10.3109/10826084.2014.891616

Warner, L. A., & White, H. R. (2003). Longitudinal effects of age at onset and first drinking situations on problem drinking. *Substance Use & Misuse*, *38*(14), 1983–2016. https://doi.org/10.1081/ja-120025123

Westermeyer, J. (1989). Nontreatment factors affecting treatment outcome in substance abuse. *The American Journal of Drug and Alcohol Abuse*, *15*(1), 13–29. https://doi.org/10.3109/00952998908993396

Witkiewitz, K., Falk, D. E., Litten, R. Z., Hasin, D. S., Kranzler, H. R., Mann, K. F., O'Malley, S. S., & Anton, R. F. (2019). Maintenance of World Health Organization risk drinking level reductions and posttreatment functioning following a large Alcohol Use Disorder clinical trial. *Alcohol: Clinical and Experimental Research*, *43*(5), 979–987. https://doi.org/10.1111/acer.14018

Witkiewitz, K., Kranzler, H. R., Hallgren, K. A., O'Malley, S. S., Falk, D. E., Litten, R. Z., Hasin, D. S., Mann, K. F., & Anton, R. F. (2018). Drinking risk level reductions associated with improvements in physical health and quality of life among individuals with Alcohol Use Disorder. *Alcoholism, Clinical and Experimental Research*, *42*(12), 2453–2465. https://doi.org/10.1111/acer.13897

Witkiewitz, K., & Marlatt, G. A. (2004). Relapse prevention for alcohol and drug problems: That was Zen, this is Tao. *The American Psychologist*, *59*(4), 224–235. https://doi.org/10.1037/0003-066X.59.4.224

Witkiewitz, K., Montes, K.S., Schwebel, F.J., & Tucker, J.A. (2020). What is recovery? *Alcohol Research: Current Reviews*, *40*(3), 01. https://doi.org/10.35946/arcr.v40.3.01

Witkiewitz, K., Pearson, M. R., Hallgren, K. A., Maisto, S. A., Roos, C. R., Kirouac, M., Wilson, A. D., Montes, K. S., & Heather, N. (2017). Who achieves low risk drinking during alcohol treatment? An analysis of patients in three alcohol clinical trials. *Addiction (Abingdon, England)*, *112*(12), 2112–2121. https://doi.org/10.1111/add.13870

Witkiewitz, K., Pearson, M. R., Wilson, A. D., Elena R. Stein, Votaw, V. R., Hallgren, K. A., Maisto, S. A., Swan, J. E., Schwebel, F. J., Aldridge, A., Zarkin, G. A., & Tucker, J. A. (2020). Can Alcohol Use Disorder recovery include some heavy drinking? A replication and extension up to 9 years following treatment. *Alcohol: Clinical and Experimental Research*, *44*(9), 1862–1874. https://doi.org/10.1111/acer.14413

Witkiewitz, K., Stein, E. R., Votaw, V. R., Hallgren, K. A., Gibson, B. C., Boness, C. L., Pearson, M. R., & Maisto, S. A. (2022). Constructs derived from the addiction cycle predict alcohol use disorder treatment outcomes and recovery 3 years following treatment. *Psychology of Addictive Behaviors*, No Pagination Specified-No Pagination Specified. https://doi.org/10.1037/adb0000871

Witkiewitz, K., & Tucker, J. A. (2020). Abstinence not required: Expanding the definition of recovery from Alcohol Use Disorder. *Alcoholism, Clinical and Experimental Research*, *44*(1), 36–40. https://doi.org/10.1111/acer.14235

Witkiewitz, K., Wilson, A. D., Roos, C. R., Swan, J. E., Votaw, V. R., Stein, E. R., Pearson, M. R., Edwards, K. A., Tonigan, J. S., Hallgren, K. A., Montes, K. S., Maisto, S. A., & Tucker, J. A. (2021). Can individuals with Alcohol Use Disorder sustain non-abstinent recovery? Non-abstinent outcomes 10 years after Alcohol Use Disorder treatment. *Journal of Addiction Medicine*, *15*(4), 303–310. https://doi.org/10.1097/ADM.0000000000000760

Wolfe, S. E., & Higgins, G. E. (2008). Self-control and perceived behavioral control: an examination of college student drinking. *Applied Psychology in Criminal Justice*, *4*(1), 108–134.

Wu, L.-T., Kouzis, A. C., & Schlenger, W. E. (2003). Substance use, dependence, and service utilization among the US uninsured nonelderly population. *American Journal of Public Health*, *93*(12), 2079–2085.

Xin, Y., España, M., & Davis, A. K. (2022). The acceptability of non-abstinent treatment goals among clinical social workers in the United States. *Journal of Social Work Practice*, *0*(0), 1–16. https://doi.org/10.1080/02650533.2022.2034768

Zilverstand, A., Huang, A. S., Alia-Klein, N., & Goldstein, R. Z. (2018). Neuroimaging impaired response inhibition and salience attribution in human drug addiction: A systematic review. *Neuron*, *98*(5), 886–903. https://doi.org/10.1016/j.neuron.2018.03.048

Appendix A: Figures

Figure 1a

Bivariate latent change score model of AUD severity and control over drinking

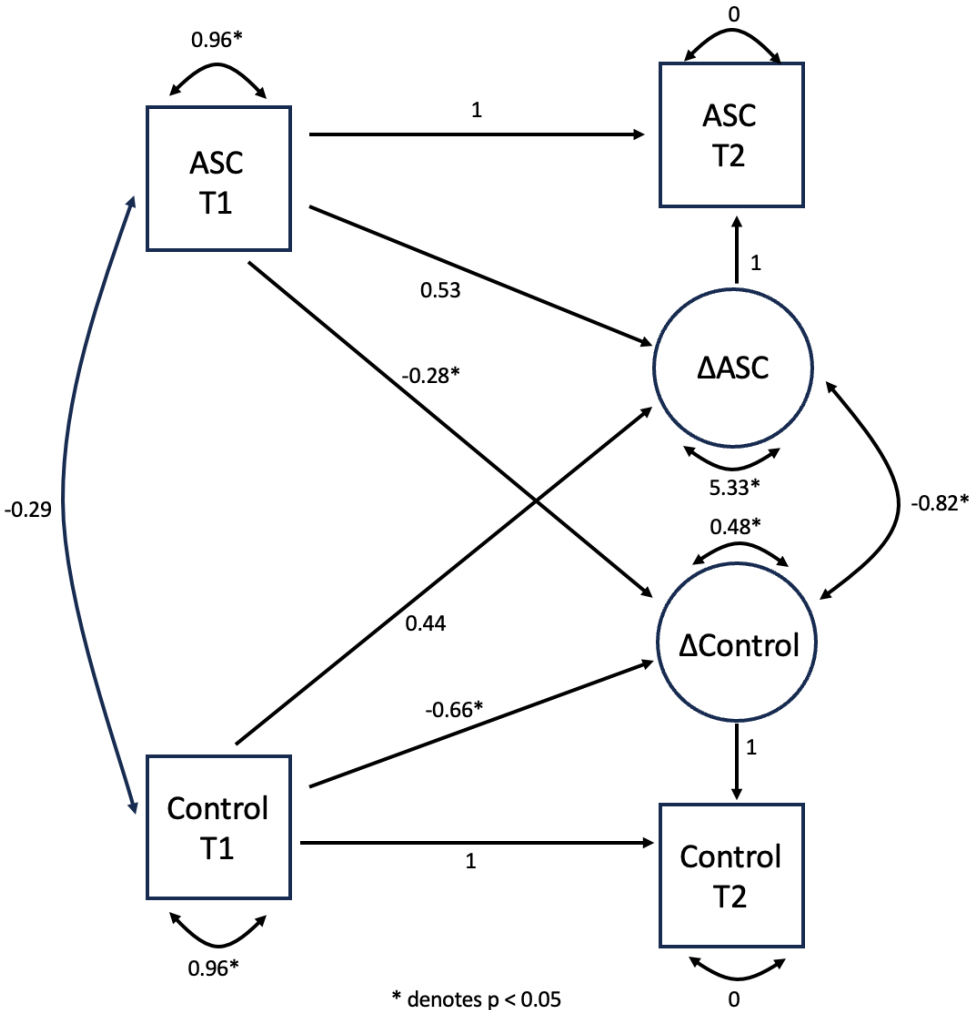


Figure 1b
Scatterplot of change in control over drinking from baseline to follow-up by AUD severity

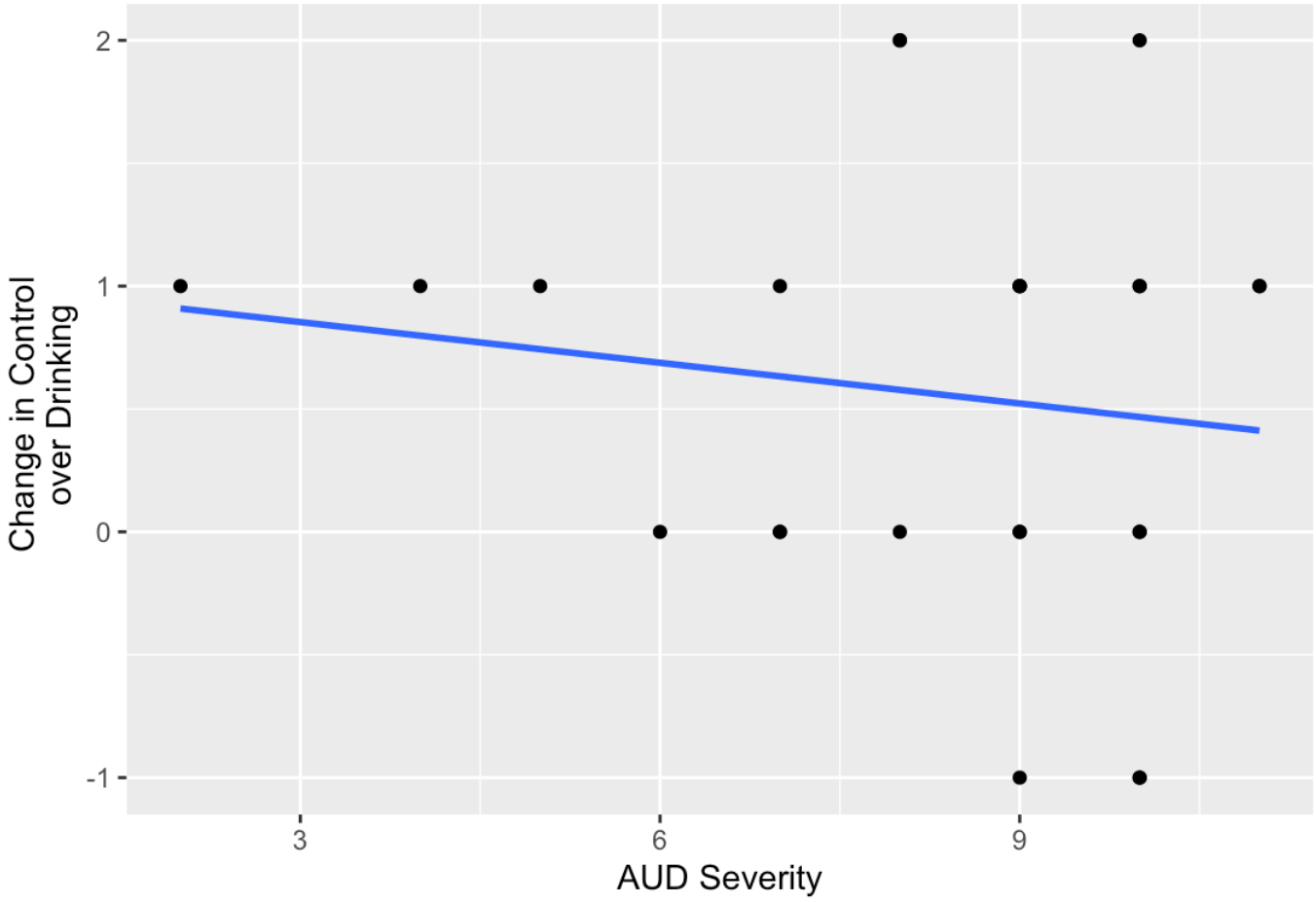


Figure 2
Bivariate latent change score model of incentive salience and control over drinking

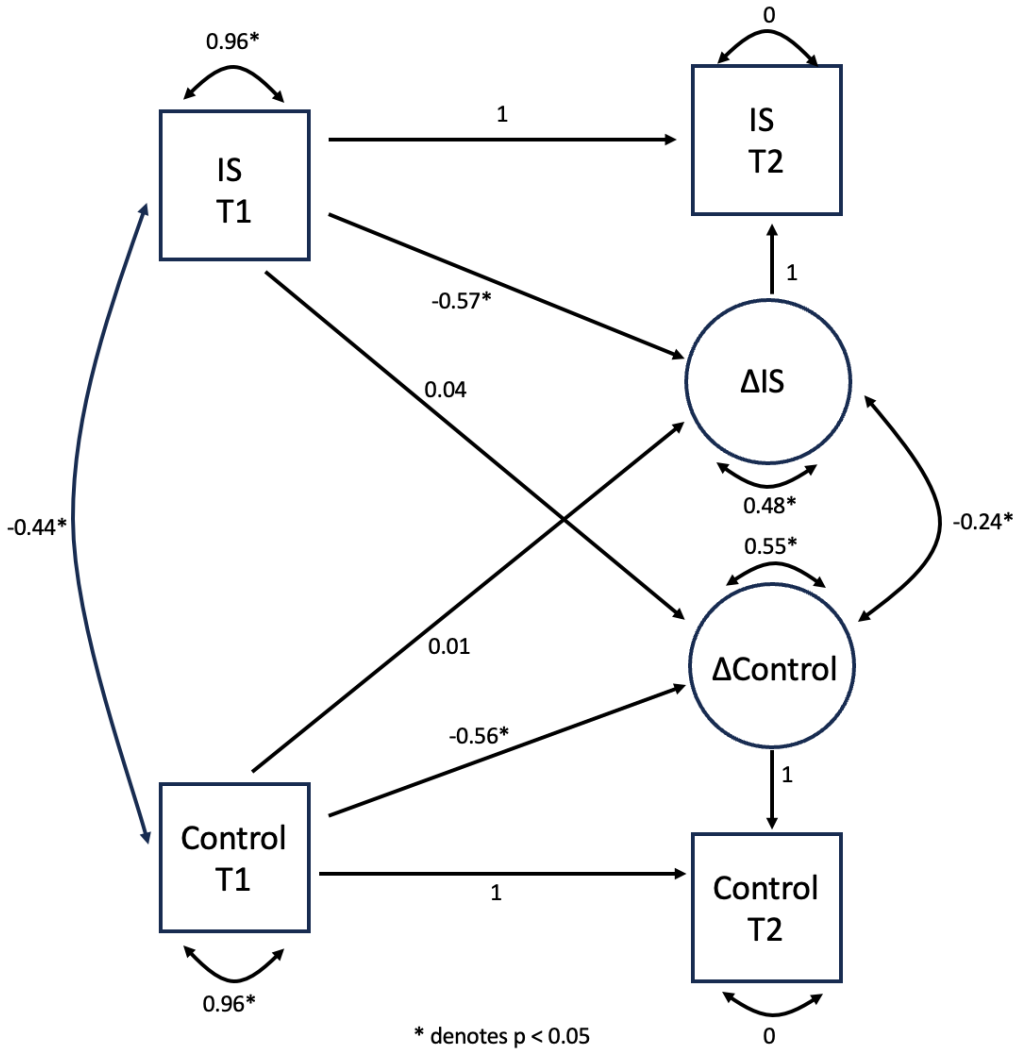


Figure 3
Bivariate latent change score model of executive functioning and control over drinking

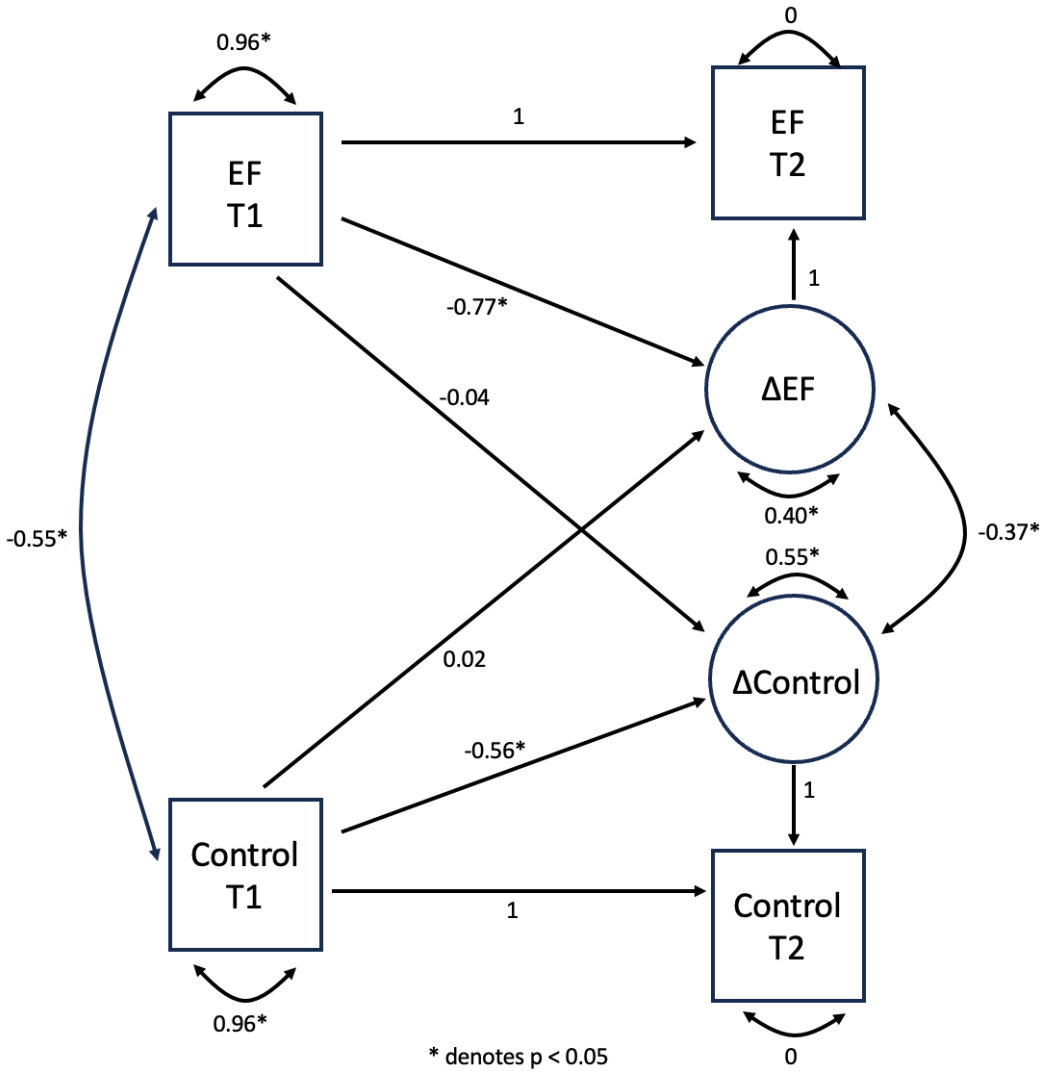


Figure 4
Trajectory of peak BAC among participants with 90-day treatment retention

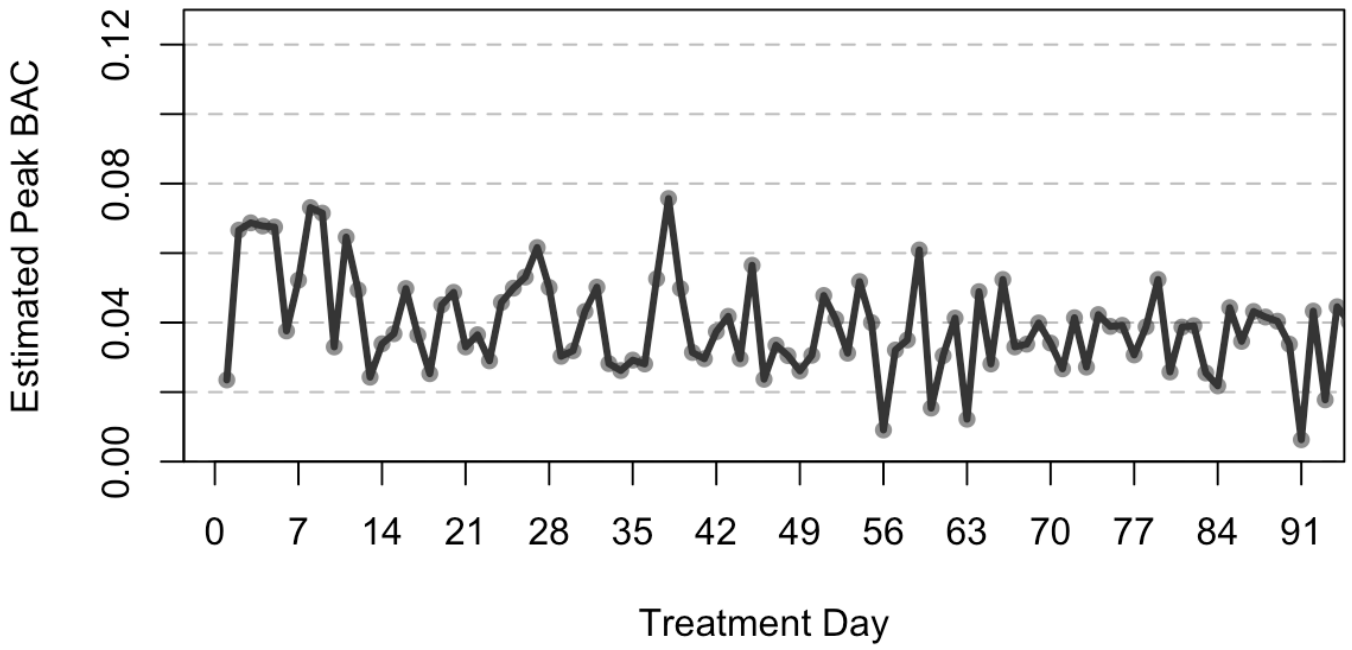
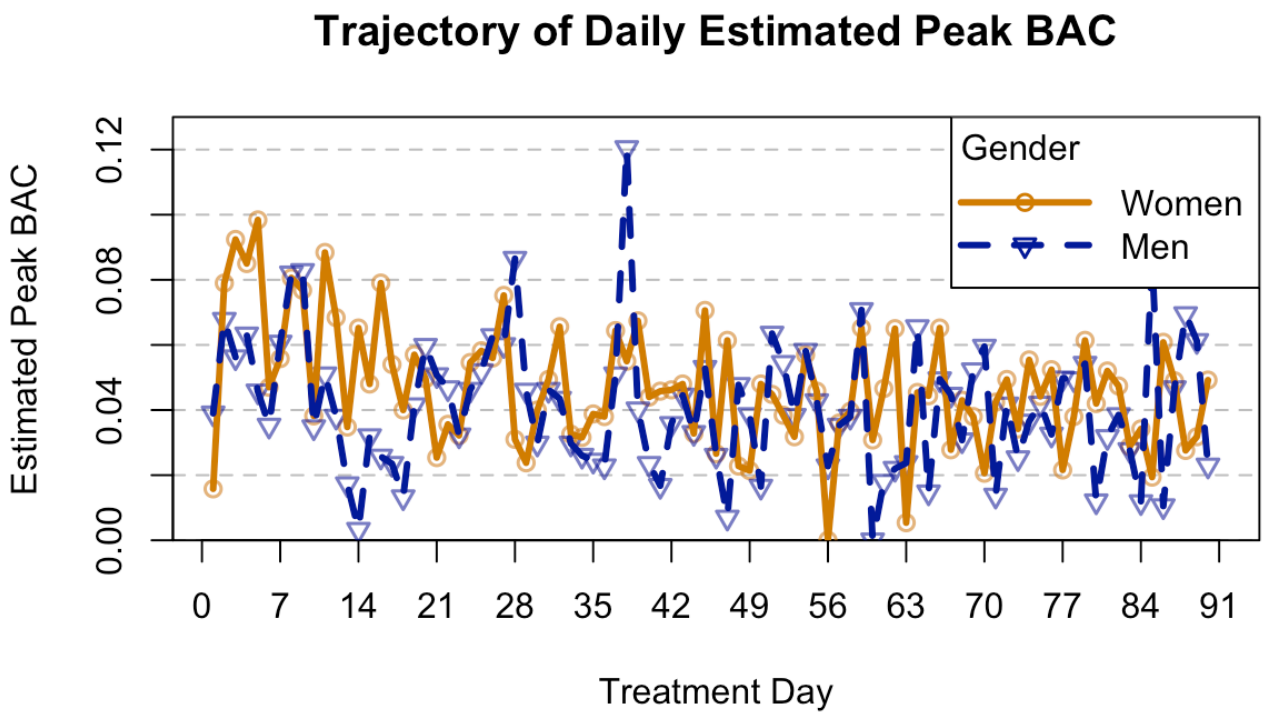


Figure 5
Trajectory of peak BAC by gender among participants with 90-day treatment retention



Appendix B: Tables

Table 1
Sample Characteristics

		N (%)	M (SD)	Paired t-test
Sample size		25 (100)	—	—
Race	White	21 (84.00)	—	
	American Indian or Alaskan Native	1 (4.00)	—	—
	Asian	0 (0.00)	—	—
	Native Hawaiian or Pacific Islander	0 (0.00)	—	—
	Black or African American	2 (8.00)	—	—
	Multi-racial	0 (0.00)	—	—
	Unknown	0 (0.00)	—	—
	Prefer not to say	1 (4.00)	—	
Ethnicity	Hispanic or Latino	1 (4.00)	—	—
	Not Hispanic or Latino	22 (88.00)	—	—
	Unknown	1 (4.00)	—	—
	Prefer not to say	1 (4.00)	—	—
Age		—	44.24 (11.00)	—
Sex	Male	11 (44.00)	—	—
	Female	14 (56.00)	—	—
AUD severity	Baseline	—	8.32 (2.21)	**t = -7.74, p < 0.01**
	Follow-up	—	4.40 (2.77)	
Family history		—	1.52 (1.26)	—
Age lapse		—	2.60 (8.40)	—
Self-stigma	Baseline	—	3.41 (0.89)	t = -0.99, p = 0.33
	Follow-up	—	3.27 (1.01)	

RESOLVE	Baseline	8 (33.33)		t = 0, p = 1.00
	Follow-up	8 (33.33)		
TLFB		—	3.74 (2.29)	—
Incentive Salience	Baseline	—	3.63 (0.72)	**t = -3.21, p < 0.01**
	Follow-up	—	3.14 (0.83)	
Negative Emotionality	Baseline	—	3.39 (0.84)	t = -1.47, p = 0.15
	Follow-up	—	3.12 (0.80)	
Executive Functioning	Baseline	—	2.99 (0.71)	**t = -4.32, p < 0.01**
	Follow-up	—	2.30 (0.69)	

Table 1 (cont.)

Peak BAC	Baseline	—	0.05 (0.07)	t = -0.14, p = 0.90
	Follow-up	—	0.04 (0.05)	
Follow-up goal	Abstinence	4 (16.00)		—
	Controlled drinking	21 (84.00)		—
Control over drinking	Baseline	—	3.56 (0.65)	**t = -3.22, p < 0.01**
	Follow-up*	—	3.64 (0.70)	

Note. ***** = Outcome variable

Table 2
Correlations of Model Variables

	Age	**AUD severity**	**Self-stigma**	**Family history**	**RESOLVE**	**Age lapse**	**IS**	**NE**	**EF**	**BAC**	**COD 1**	**COD 2**
Age	1											
AUD severity	-0.13	1										
Self-stigma	0.01	**0.43**	1									
Family history	-0.19	**0.50**	0.31	1								
RESOLVE	**-0.48**	0.08	0.19	0.36	1							
Age lapse	0.30	-0.11	0.03	0.01	-0.03	1						
Incentive salience (IS)	-0.01	0.38	0.15	0.21	-0.02	-0.30	1					
Negative emotionality (NE)	-0.12	0.16	0.39	0.23	0.00	-0.29	0.47	1				
Executive functioning (EF)	-0.10	**0.48**	0.22	0.31	0.22	-0.01	**0.70**	0.40	1			
BAC	-0.10	0.04	0.14	-0.03	0.03	-0.17	0.10	0.33	0.12	1		
Baseline control over drinking (COD1)	-0.14	-0.30	-0.33	0.12	0.04	-0.21	**-0.46**	-0.33	**-0.58**	-0.35	1	
Follow-up control over drinking (COD2)	0.04	**-0.44**	-0.17	-0.16	-0.06	-0.14	-0.19	0.04	-0.25	-0.31	**0.49**	1

Note. Correlations where $p < 0.05$ are bolded.

Table 3
Predictors of Follow-up Control over Drinking by Baseline Drinking Goals

	Standardized Coefficient	**Unstandardized Coefficient**	**Standard Error**	**95% Confidence Interval**	***p*-value (Holm corrected)**
Intercept	3.73	0.33	2.86	-5.89, 6.55	**< 0.01**
Male (ref.)	—	—	—	—	—
Female	-0.61	-0.60	0.44	-1.56, 0.34	> 0.99
Age	0.06	0.01	0.02	-0.04, 0.05	> 0.99
AUD severity	-0.30	-0.14	0.12	-0.39, 0.12	> 0.99
Self-stigma	0.18	0.20	0.26	-0.37, 0.05	> 0.99
Family history	-0.12	-0.10	0.21	-0.55, 0.36	> 0.99
RESOLVE	0.02	0.02	0.47	-1.00, 1.03	> 0.99
Age lapse	0.07	0.01	0.03	-0.06, 0.07	> 0.99
Incentive salience	0.07	0.09	0.42	-0.82, 1.01	> 0.99
Negative emotionality	0.28	0.34	0.30	-0.31, 1.00	> 0.99
Executive functioning	0.19	0.27	0.43	-0.67, 1.21	> 0.99
Baseline control over drinking	0.49	0.57	0.36	-0.21, 1.35	> 0.99
BAC	-0.26	-3.57	2.76	-9.58, 2.44	> 0.99

Table 4a
Latent growth curve changes in peak BAC over 90 days

Fixed Effects	Unstandardized Coefficient	Standard Error	p-value
Intercept (mean BAC at day 14)	0.044	0.014	**0.009**
Early treatment change (days 1-14)	-0.036	0.020	0.105
Later treatment change (days 15-90)	-0.005	0.002	**0.021**
Random Effects			
Intercept	0.002	—	—
Early treatment change	0.003	—	—
Later treatment change	0.000	—	—
Residual	0.001	—	—

Appendix K. Table 4b
Gender-moderated latent growth curve changes in peak BAC over 90 days

Fixed Effects	Unstandardized Coefficient	Standard Error	p-value
Intercept (mean BAC at day 14)	0.035	0.019	0.092
Early treatment change (days 1-14)	-0.028	0.029	0.367
Man (ref.)	—	—	—
Woman	0.017	0.027	0.529
Later treatment change (days 15-90)	-0.003	0.002	0.135
Early treatment change x Woman	-0.017	0.041	0.683

Later treatment change x Woman	-0.003	0.003	0.415
Random Effects			
Intercept	0.002	—	—
Early treatment change	0.003	—	—
Later treatment change	0.000	—	—
Residual	0.001	—	—

Table 5
Crosstabulation of planned outcome variable (perceived success) and employed outcome variable (control over drinking)

		Control over drinking				
		"I have complete control"	"I am usually able to exercise voluntary control over it"	"I can control it with difficulty"	"I must drink and can only delay drinking with difficulty"	Total
Perceived success	"I'm much better"	2	5	1	0	8
	"I'm a little better"	1	2	9	3	12
	"I haven't changed at all"	0	0	2	0	2
Total		3	7	12	3	25