Labor Market Outcomes of Individuals in Recovery from Addiction to Alcohol

Arnie P. Aldridge

Department of Economics

University of North Carolina at Chapel Hill

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I. Introduction

By the 21st Century, 8.46% of adults in the United States met clinical criteria for alcohol abuse (4.65% or 9.7 million adults) or alcohol dependence (3.81% or 7.9 million adults) (Grant et al. 2004). Alcohol Use Disorders (AUDs), including abuse and dependence, impose significant costs on society, estimated at $184 billion in 1998 (Harwood 2000; Mokdad 2004). The majority of the cost burden (60%) is due to alcohol’s adverse impact on the labor market in the form of lost wages for those not employed and decreased productivity for those employed. It has been shown that alcohol abuse (MacDonald and Shields, 2004, Feng et al, 2001, and Mullahy and Sindelar, 1996) and dependence (Johannson et al, 2007) and are associated with unemployment and labor market detachment. AUDs are associated with lost productivity (Cook and Moore, 1999 and Mullahy and Sindelar, 1998) and lower earnings (Keng and Huffman, 2002, Jones and Richmond, 2006, Zarkin et al, 1998). Understanding the causal relationships between alcohol and labor market performance is valuable for constructing alcohol use, prevention, and treatment policies.

Literature on the connection between AUDs and labor market outcomes broadly fits two categories. The first looks at how outcomes differ between populations with and without AUDs. Public health
and economic research has sufficiently shown the deleterious effects of AUDs. Econometric studies in particular have been valuable in refining the estimates of these effects by addressing several common confounders: reverse causality and unobserved characteristics that simultaneously influence drinking and labor market success.\(^1\) The value of this literature is that it has identified AUDs as a measurable problem and has broadly described its costs to individuals and society.

The literature, however, has several limitations. Most studies are based on a snapshot of current substance use and labor market outcomes, ignoring how changes in both behaviors evolve over time and how the compositions of the AUD and non-AUD samples change.\(^2\) Economic decision making has a direct bearing on how the costs of substance abuse are determined, and understanding that behavior directly informs policy making. Finally, it does not incorporate the role of AUD treatment in changing the composition of the AUD and non-AUD populations that are compared in static cross-sections.

The second type of literature broadly focuses on the efficacy and effectiveness of prevention and treatment of AUDs (Room et al, 2007). These studies are generally based on clinical trials of interventions and specialty treatment and evaluations of programs or policies that directly provide treatment, reduce barriers to treatment, or create disincentives for alcohol consumption. Labor market outcomes are usually analyzed as secondary outcomes in these studies. These studies are limited in their understanding of how improvements in AUDs lead to labor market outcomes and how those labor market outcomes recursively influence AUDs (e.g., psychosocial benefits of employment as a protective factor or work stress). Often, they are simply limited by the period of time over which they follow participants and do not allow sufficient time for improvements in labor market outcomes. Moreover, studies of specialty

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\(^1\) These studies do not always compare two discrete populations, those with and those without AUDs, but estimate local average treatment effects and implicitly compare populations with marginally different AUDs.

\(^2\) Johannson (2006) shows for example that abstinent former dependent drinkers often have bad enough outcomes that comparisons between abstainers and moderate drinkers yields biased results.
treatment often ignore the extent to which participants seek additional or future treatment beyond the original study. Yet, additional or repeated treatment is considered appropriate and is often based on attained employment which provides insurance or financial accessibility (CSAT, 2004).

In the study I am proposing, I develop a model of employment, drinking, and treatment-seeking that is based on forward-looking behavior. Predictions from the model are estimated using longitudinal data on individuals from COMBINE, a National Institutes of Alcoholism and Alcohol Abuse and Drug Abuse (NIAAA) randomized control trial of two pharmacotherapies for dependence. The first aim of this study is to estimate the causal effects of AUD outcomes on employment over a three-year period following the COMBINE trial. The second aim is to estimate the effectiveness of the ongoing consumption of pharmacotherapies for managing AUDs.

The remainder of this manuscript contains a more in depth discussion on the background and literature of employment, alcohol and treatment modeling (Section II), a theoretical model (Section III), an empirical model (Section IV), a description of the sample used for estimation (Section V) and preliminary results (Section VI).

II. Background and Literature:

Alcohol Abuse and Alcohol Dependence

Understanding what is meant by different AUDs is necessary for interpreting the literature on alcohol use and related outcomes. Moreover, the specific measures of AUDs can have different theoretical relationships with outcomes being studied. The Diagnostic and Statistical Manual of Mental Disorders, currently in its fourth edition (DSM-IV), provides clinical criteria for diagnosing alcohol abuse and alcohol dependence. Although various levels and patterns of alcohol consumption can be detected through biological screening (e.g., urine tests detect increased liver enzymes), DSM-IV clinical
determinations are based on self-reported information. Indications of alcohol abuse are based on perceptions of drinking’s secondary effects: does the individual feel that alcohol caused problems at home, work, or school, led to dangerous behaviors, or led to criminal justice interactions. In addition, if the individual reports the inability to reduce consumption despite the perception of alcohol’s consequences he qualifies as abusing.

The DSM-IV defines dependence with a focus on consumption patterns, drinking’s primary consequences and the individual’s relationship with alcohol. A positive diagnosis of dependence is typically made when a clinician identifies three or more of the following:

1. Spent a great deal of time over a period of a month getting, using, or getting over the effects of alcohol;
2. Used alcohol more often than intended or was unable to keep set limits on alcohol use;
3. Needed to use alcohol more than before to get desired effects or noticed that same amount of alcohol use had less effect than before;
4. Inability to cut down or stop using alcohol every time tried or wanted to;
5. Continued to use alcohol even though it was causing problems with emotions, nerves, mental health, or physical problems;
6. Alcohol use reduced or eliminated involvement or participation in important activities; and has experienced two or more withdrawal symptoms during the same time period:
7. Reported experiencing two or more alcohol withdrawal symptoms at the same time that lasted longer than a day after alcohol use was cut back or stopped. Symptoms include (i) sweating or feeling that heart was beating fast, (ii) having hands tremble, (iii) having trouble sleeping, (iv)
vomiting or feeling nausea[ted], (v) seeing, hearing, or feeling things that were not really there, (vi) feeling like could not sit still, (vii) feeling anxious, and (viii) having seizures or fits. ³

It is important to recognize that abuse and dependence are psychological constructs that categorize a degree of severity in drinking behaviors, drinking consequences, and an individual’s relationship to drinking. AUDs represent a measurement problem described originally in psychological research in which a latent construct (e.g., dependence) is not observed but can be defined by how it manifests itself in behavior, consequences, and perceptions. AUDs are uniquely challenging to define because they are dynamic. Over time, individuals may cycle in and out of different levels of severity, even returning to abstinence. These observed cycles are not a reflection of the reliability of clinical testing but are in evidence when measured by self-reported consumption patterns, clinical interviews, and biological screening (McLellan 2007).

A special challenge for researchers is determining which measurement is useful for analysis. Consumption levels are correlated with the severity of a disorder as defined by the other criteria but their inconsistency has implications for some research questions. Clinical interviews have the advantage of evaluating an individual’s ongoing struggle with an AUD (e.g., strong cravings to drink or a fixation on alcohol) that may not be manifested through current consumption alone. For example, a currently abstinent individual may still have a latent disorder that is reducing his functioning or altering his preferences. In the first case, cross-sectional analyses of drinking and labor market outcomes would only represent the direct impact of drinking and the impact of disorders only for current drinkers. Altered preferences largely explain such phenomena as continued treatment seeking by abstainers as well as their avoidance of certain social environments (e.g., weddings with open bars). Longitudinal observa-

³ Text are from NSDUH’s version of the DSM-IV criteria.
tion of consumption resolves these challenges to some extent while also providing more specificity (i.e., timing, lagged consumption patterns) than discrete clinical diagnoses.

In addition to the DSM-IV diagnosis criteria and in response to the public health burden of moderately risky drinking, researchers have also developed screening instruments to detect both finer levels of less risky drinking while remaining sensitive enough to detect severe problems with minimal respondent information.\(^4\) Most screeners ask about an individual’s average alcohol consumption in standardized drinking units, usually the quantity of drinking in an episode and the frequency of episodes during a set time period. They also ask about perceptions of drinking and related consequences. Although consumption measures seem crude, they tend to be strong indicators of non-consumption criteria. In fact, there is an emphasis among public health and clinical researchers to move to a core quantity and frequency (Q-F) measure to more quickly screen individuals (Saitz, 2005; Gastfriend et al, 2007; McLellan, 2007). These instruments and Q-F measures are prevalent in many large observational studies and have been used to estimate the relationship of drinking with secondary outcomes. Again, the use of reported consumption is often more appropriate in models of alcohol’s causal impact, since the alternative constructed measures described above often include the measures of the dependent variables being analyzed (e.g., absences from work).

Following from these different measures of drinking disorders, terms like ‘risky’, ‘problem’, ‘harmful’ or ‘hazardous’ drinking are used in different studies and are sometimes used interchangeably with ‘abuse’. In the remainder of this literature review, I use the exact measures that the authors used and clarify their meanings when necessary. In the theoretical model described in Section III, AUD is a continuous variable representing the severity of an individual’s drinking disorder. In the empirical model

\(^4\) For example, the Alcohol Use Disorders Identification Test (AUDIT) is designed to detect a continuum of risky use levels and the four question CAGE (Cut-down, Annoyed, Guilty, Eye-opener) is designed to detect dependence.
in Section IV, I use a generic measure of drinking but am considering several alternative measures described in Section V.

*Employment and Drinking:*

Alcohol use disorders are associated with labor market outcomes along multiple causal pathways. Both acute alcohol abuse, such as binge drinking, and longer term dependence can reduce an individual’s work productivity through reductions in human capital, health, and motivation (Cook and Moore, 1999; Mullahy and Sindelar, 1998, Corrao et al, 2004). They likewise decrease the intensity of job searching through the same mechanisms. Even if real productivity decreases are not realized, such drinking behaviors can serve as a negative signal to current and prospective employers. These adverse effects accumulate over time with increasing productivity loss and a growing portfolio of negative signals that can include sporadic labor market attachment and a reputation of low productivity.

AUDs also alter observed labor market outcomes through the individual’s preferences. An abusive or dependent drinker may value leisure differently because of worse health or a complementarity of drinking and leisure. He may discount time differently or have a different attitude toward risk, relative to the general labor market population either due to pre-existing characteristics or due to neurological changes brought on by drinking [Dom et al, 2005; Moselly et al, 2001; Tavares et al, 2004]. Therefore, he may leave the labor market more often and for longer periods of time. He may choose to work part time which may have later consequences for his earnings profile and employment probabilities. Alternatively, the deleterious effects of AUDs on health may provide more incentive for an individual to remain with an employer who provides health insurance.

Identifying causal pathways is further complicated by the fact that an individual’s labor market experience also influences drinking behaviors. Employment produces an income effect on all consumption, potentially increasing drinking. There may likewise be an income and insurance effect on drinking
that operates through expectations about treatment for AUDs. Individuals anticipating sufficient income or insurance coverage for specialty treatment may increase their current drinking (ex ante moral hazard). Both employment and unemployment can induce stress that is associated with AUDs (Frone, 1999; Gallo et al, 2001). Employment may provide a social network that facilitates and encourages drinking. Finally, employment may provide protective factors that reduce the prevalence of AUDs. These include social norms that encourage safe drinking, wellness programs, and easier access to treatment through employer provided insurance and Employee Assistance Programs.

In this study, I focus on employment as the primary labor market outcome for several reasons. Employment is the broadest measure of labor market value and subsumes labor supply. For individuals currently in the labor market, real wages do not change much over a several-year time horizon. On the other hand, choosing to seek employment and finding employment are both outcomes with substantial variation over the study period.

Most of the estimated effects of AUDs on employment found in the economics literature rely on large, cross-sectional datasets. Specifically, these studies explain the different rates of employment between individuals with and without AUDs among an observed population. The fundamental econometric challenge in these studies is estimating the causal effects of AUDs in the face of a simultaneity problem or when unobserved heterogeneity is likely to explain both the AUD and labor market success. The standard approach in these studies is to use instrumental variables that predict an individual’s AUD but are theoretically and empirically uncorrelated with labor market outcomes other than through the AUD. With data from the National Health Interview Survey (NHIS), Mullahy and Sindelar (1996) used parental AUDs and beer and cigarette taxes as instruments of dependence, abuse and harmful drinking.

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5 A similar approach is seen in the literature on wages and labor supply as summarized in Jones (2006) and Johansson et al (2007).
MacDonald and Shields (2004) used non-acute illnesses that might limit drinking (e.g., asthma and diabetes) as instruments of dependence in the 2000 National Health Survey of England. Johansson et al (2007), using Finland’s Health 2000 survey, utilized parental characteristics, asthma and diabetes, religiosity, a person smoking behavior at the age of 18, and medical biomarkers as instruments of dependence. All of these studies found significant and large effects of abuse and dependence on employment. Several found a positive relationship between moderate levels of drinking and employment.

One study (Feng et al, 2001) used a repeated cross-sectional dataset to estimate the effect of problem drinking on employment. Problem drinking was defined by combinations of lifetime DSM-IV criteria and drinking behaviors during the previous 12 months. Employment was defined as any employment during the same 12 months. With data from the Epidemiological Catchment Areas of six southern US states, they estimated bivariate probit models of the contemporary effect of problem drinking on employment. When using county alcohol sales policies as instruments this study found no negative consequences of problem drinking on employment and argued that the effects of problem drinking on employment may occur over a long period of time.

While these studies have been useful in demonstrating reasonable estimates of the employment consequences of having an AUD, they exhibit a common limitation in cross-sectional studies. They provide only the estimated effect of recently having an AUD on current employment, when both the AUD and employment outcomes are the results of a long series of prior decisions. Abuse and addiction have complex dynamic paths over time within individuals. This limitation presents several challenges to interpreting results and to making alcohol policy. It is uncertain what proportion of the population ‘without a recent AUD’ has experienced one in the past. Without knowing how long the negative employment consequences of an AUD last, we cannot know to what degree we may be underestimating the effect of having an AUD. Moreover, without understanding the causal mechanisms, we do not know
whether we should expect prevention or treatment policies to have any short or long term labor market benefits. The only panel study of the AUD-employment relationship highlights this problem by offering the explanation that there may be a delay in employment consequences of problem drinking.

_Treatment for AUDs_

Although abstinence has traditionally been a goal of specialty treatment, researchers on treatment effectiveness have placed new emphasis on reductions in harmful drinking episodes, recognizing that a steady state of moderate drinking is can be the goal of individuals seeking treatment (McLellan 2004). Overall, specialty treatment for alcohol is effective with some studies finding more than half of recipients remaining abstinent by the end of the observation period (CSAT 2004; Room et al, 2005; Project Match Research Group, 1997). Although many studies use length of time to relapse as an outcome, relapsing to problem drinking does not mean that the recovery process has ended and returning to treatment is not a bad outcome. Initial abstinence is a good predictor of long term healthy behaviors (Maisto et al, 2006; McKay and Weiss, 2001) including the maintenance of safe or controlled levels of drinking after treatment (McLellan 2004; Gastfriend et al, 2007).

For an individual with a more severe AUD, ‘recovery’ is often defined by more than an episode of abstinence or controlled drinking. As noted earlier, consumption is useful for measuring outcomes over the limited periods of observation that studies face. However, clinicians, patients and researchers recognize that recovery is not simply an end state in which a ‘disease’ has been ‘cured’\(^6\). Rather, language such as ‘in recovery’ is more commonly used to refer to ongoing success with an acknowledgement of the potential for relapse. Moreover, successful recovery is better conceived of as a steady state in which not only consumption is controlled but the latent factors that motivate problematic consumption are also alleviated or managed. These factors include antecedent individual characteristics such as genetics and socioeconomic environment that led to the initial AUD. Manifestations of these are risk- or

\(^6\) Note for instance the “Disease” model of addiction that is implicitly used by Alcoholics Anonymous.
sensation-seeking personalities, depression, anxiety and other psychiatric disorders, social acceptability of excessive drinking, social norms regarding leisure activities, and limited opportunities for healthy or fulfilling activities are all risk factors for AUDs that may remain in place even after initial treatment has led to abstinence or controlled consumption. Dynamic factors brought on by past consumption also challenge the recovery process. These include changes in brain structure that alter decision-making faculties and alter preferences for alcohol and other goods and activities; development of mental illness; habits; and socioeconomic circumstances such as reduced human capital or a primary social group that is centered on alcohol. The broader goal of treatment is therefore to facilitate a steady state of recovery by managing these factors in addition to managing consumption. In recent years policy makers have begun to define substance abuse treatment in general more broadly as an integrated, holistic form of care that attempts to provide physical and mental health treatment, engage the individual’s family and community for social support, and even provide ancillary services such as travel to treatment and job training. For example, borrowing a model from psychiatric treatment, the federal Substance Abuse and Mental Health Services Administration (SAMHSA) has begun to promote Recovery Oriented Systems of Care (ROSC) as a model for managing substance abuse in the US.

Traditional Specialty Treatment

Conventional forms of specialty treatment vary by the severity of the AUD and most types of treatment may be considered part of a continuum of care that ideally helps an individual improve from his current AUD to steady state recovery. The intensity of treatment in the continuum is intended to match the severity of the AUD and decrease as an individual improves. The intensity is loosely correlated with consumption level, due in part to the biological nature of severe physical addiction. The most intensive care associated with AUDs a period of detoxification in which a patient is sequestered, monitored and medicated

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7 Many individuals also engage in informal self-help programs such as Alcoholics Anonymous as a complement or alternative to formal specialty treatment.
for safety and management of withdrawal symptoms. Inpatient is traditionally 28 days and nights of treatment in a facility that offers a range of services, including pharmacotherapy and counseling.

Along the continuum of care, residential treatment, day treatment and outpatient therapy follow inpatient treatment with group and individual sessions occurring 1 to 7 times per week. Inpatient and outpatient counseling usually rely on motivational enhancement therapy, 12-step facilitation treatment, cognitive behavioral treatment and behavioral family counseling, and contingency management and community reinforcement approaches or some combination of these. Each of these approaches share similar elements. They encourage goal setting and the development of a sense of self-efficacy through ‘practicing’ sobriety. They also encourage proactive restructuring an individual’s lifestyle. These include changes to work and social environments as well as developing alternative leisure activities. An individual is also encouraged to simultaneously treat mental or physical illness. Each approach seeks to change motivations by changing perceived social norms and reiterating the consequences of consumption, promoting positive social reinforcement and accountability (either from the family, a mentor or the clinician) and highlighting the positive value gained from alternative activities. They teach mechanisms for coping with stress and temptation, which include pre-commitment strategies (e.g., requesting hotel rooms without mini-bars) and contemporaneous coping strategies (e.g., cognitive tools for overcoming periods of temptation) (Moos 2007; Project Match Research Group, 1997).

**Pharmacotherapy for AUDs**

Pharmacotherapies are often prescribed in conjunction with inpatient treatment and outpatient counseling. Moreover, some of these medications are increasingly prescribed by primary care physicians for individuals with varying AUD severity and who may not otherwise be engaged in specialty substance abuse treatment. The medications commonly associated with AUDs typically fall into three categories: medications that alleviate withdrawal symptoms, medications enhance overall mental health,
and medications that support recovery by directly influencing an individual’s preferences for drinking (Williams, 2005). The last group is the focus of this study. Medications for withdrawal are prescribed for a short period of time to reduce the mental and physical effects of sharp reductions in alcohol consumption. The broadest class used is benzodiazepines which have anxiolytic and anticonvulsant properties. It should be noted that the availability of medically facilitated detoxification and medications to make withdrawal less unpleasant can have a perverse effect on long run recovery as it reduces one the disincentives to relapse and escalation of consumption. Moderate and severe mental health (MH) problems are commonly co-occurring with AUDs, with anxiety and moderate depression having the highest prevalence at all degrees of AUD severity. Regardless of the causal relationship between AUDs and MH, treating MH is expected to facilitate recovery indirectly by improving the individual’s overall wellbeing, his ability to cope, and by reducing the ‘pain’ of poor MH that leads to self-medication with alcohol. In other words, the intent is for these medications to be pharmacological analogues to many of the proximal outcomes of the counseling therapies described above. Selective Serotonin Re-uptake Inhibitors (SSRIs) and benzodiazepines (for longer run anxiety rather than detoxification) are among the most commonly prescribed medications to individuals with AUDs or who are in recovery (Berglund et al, 2006; Grant et al, 2004; Sher 2004; Watkins et al, 2006). Their use is complicated by contraindications with drinking and, in the case of benzodiazepines, the specific concern of exposing individuals to new addictive substance. There is ample evidence that individuals seek these medications regardless of any intent to alter their alcohol consumption and that primary care physicians prescribe them without knowledge of an existing AUD.

Medications in the final category are intended to support recovery directly and are usually prescribed specifically for the AUD. They theoretically aid recovery by reducing cravings, preventing compulsive relapse, or causing nausea or discomfort from drinking. There are currently three US FDA-approved medi-
cations for relapse prevention during recovery from alcohol dependence: Disulfiram (Antabuse), Naltrexone and Acamprosate. As post-withdrawal pharmacotherapies, they function in a similar fashion as some of the counseling strategies. Disulfiram, which causes nausea and discomfort if alcohol is also consumed, is a pre-commitment device. The two drugs studied in the COMBINE trial, Naltrexone and Acamprosate, both normalize brain functioning by affecting neurotransmitters that may be unregulated due to chronic alcohol consumption. They both reduce alcohol cravings. Naltrexone’s mechanism of action is dopaminergic, improving impulse control, reducing the intensity of cravings and reducing the pleasure of alcohol. Naltrexone has been found to reduce the amount of alcohol consumed in a single setting with patients noting a less desire to drink to excess. Acamprosate’s mechanism of action is not yet understood although it operates through the glutamate and gamma-aminobutyric acid (GABA) system. Acamprosate does not alter the effects of consumed alcohol. Acamprosate is the newest of the three drugs and was approved by the FDA in 2005. Several additional medications with similar pharmacology are either currently being studied for efficacy in managing drinking or are known to be prescribed off-label. These include quetiapine, topiramate, gabapentin, levetiracetam, baclofen, tiapride, bromocriptine and aripiprazole. Because certain benzodiazepines are ?-aminobutyric acid (GABA)ergic there is ongoing interest in their use as a longer run pharmacotherapy for alcohol dependence despite the challenges described above (Bankole, 2005). Finally, serotonergic medications continue to be studied explicitly for treating alcoholism. There is some evidence that SSRIs are effective for controlling alcohol consumption especially for late-onset dependents. However, there is conflicting evidence as to whether the reduced preference for alcohol observed is due to a general effect on consumption and satiety with respect to food and liquids or a selective effect on alcohol. Moreover, there is little evidence that SSRIs are more beneficial for individuals with co-morbid depression than placebo. Ondansetron is a serotonin antagonist (rather than an SSRI) with growing evidence of efficacy for drinking outcomes and also reported reductions in the cravings for alcohol and enjoyment of drinking.
Naltrexone, Acamprosate and this latter group of medications can have a proximal effect on alcohol consumption—both the decision to engage in drinking and the intensity of drinking. There is no set recommendation for how long Naltrexone and Acamprosate should be prescribed. The COMBINE trial dispensed medications for 4 months, while some clinicians have recommended 6-12 months (Fatemi and Clayton, 2008). Disulfiram is usually prescribed for shorter periods. For all of them, there is an understanding that additional prescriptions may be necessary as boosters, similar to traditional counseling. The longer run influence on recovery is expected to operate indirectly. Short run reductions in consumption allow the brain and physiological adaptations of addiction to heal and normalize. While on the medications, lifestyle changes and habit formation may occur more easily and individuals may develop coping strategies. Their influence can function in a way dissimilar to counseling therapy alone. While on the medications, an individual may be able to manage his drinking while not altering his lifestyle, an often infeasible challenge. He can thus be reconditioned to not drink in response to the cues and routines of daily life.

*Economic Models of Treatment for AUDs*

Any attempt to model an individual’s drinking, and treatment decisions must recognize that they are not made in ignorance of future consequences. An individual knows that abusive drinking can lead to near term and long term productivity loss, labor market challenges, poorer health and, most importantly, to severe dependence, a proclivity for continuing abuse or withdrawal effects. The latter consequence, that individuals know that drinking today influences the value of drinking later, is a key component of Becker and Murphy (1988)’s rational addiction framework for modeling substance use. This framework is a useful starting place for analyzing drinking choices jointly with other economic choices. Drinking decisions today may be influenced by expectations about productivity losses, employment probabilities and health. Moreover, individuals may recognize that consuming specialty treatment for AUDs can be an effective tool for moderating their drinking and its ultimate consequences.
Theorists have expanded the original rational addiction model in an attempt to make it better explain observed substance use and, to a limited extent, treatment seeking behaviors. The original theory lacked explicit motivation for treatment seeking: individuals chose substance use behaviors that provided them with maximum lifetime utility. Orphanides and Zervos (1996) provided a rationale for a posterior demand for treatment, after an individual discovered if they were an addictive personality type. Several observed phenomena were still lacking theoretical justification, including relapse to AUDs, ongoing treatment seeking even after achieving abstinence, the tendency for some individuals’ convergence to moderate drinking patterns rather than abstinence. Bernheim and Rangel (2004) incorporate the neuroscience on substance use behavior into a traditional rational addiction framework. A key component is that individuals can find themselves seemingly randomly in a ‘hot’ mental state in which their instantaneous marginal utility of a substance leads to behavioral ‘mistakes’ and a reduction in total lifetime utility. The ‘hot’ states are brought on by environmental cues that trigger brain mechanisms that are manifested as a compulsive desire to consume the substance. In the Bernheim and Rangel model, individuals in recovery manage this challenge in part by choosing safe environments in which the flow of cues is reduced. The most extreme example of this behavior is checking into a residential treatment facility. A second role of treatment that their model recognizes is learning to deal with cues, a common objective of most counseling therapy. Although they do not discuss pharmacotherapy, it can be justified in a similar way as counseling.

In summary, the theoretical literature does not explicitly model the demand for treatment. Even with improved justifications for treatment, competing models of addiction are underidentified by available data. However, the economic models are consistent in their implication that under certain assumptions individuals may seek treatment. Moreover, the economics literature is consistent with several stylized facts and with findings in the clinical literature: individuals vary in their substance use patterns.
The behaviors of some individuals are static over long periods of time, while others are dynamic, cycling through dependence, moderation, and abstinence. Individuals seek treatment all along the continuum between dependence and abstinence. Importantly, the rational addiction framework does not a priori assume that any particular substance use pattern is sub-optimal for the individual. Even individuals who reveal their preferences by seeking treatment are not necessarily falling short of their best lifetime utility scenario.

The policy relevance of studying treatment is threefold. First, there are externalities from AUDs, including decreased employment and lost productivity, accidents, public health care costs, and crime. Second, the existing treatment system is largely a public system. Given that some individuals are willing to seek treatment, it is worthwhile to study the relative effectiveness of different portfolios of treatment to inform policies that promote treatment. Finally, as synopsized by Bernheim and Rangel (2004) individuals may suffer from unanticipated compulsion to consume sub-optimally (internalities). Studying the effectiveness of alternative treatments is worthwhile for improving their welfare.

III. Theoretical Motivation

In this section I present a dynamic, theoretical model of the behavior of individuals who were at one time dependent on alcohol and who have previously sought formal specialty treatment. The model focuses on their employment, drinking, and ongoing AUD treatment decisions. In each period \( t \), an individual derives utility from general leisure, \( l_t \), alcohol consumption, \( a_t \), and consumption, \( c_t \). He begins each period with a set of state variables accounting for his previous decisions and experiences: work history prior to the current period, \( Q_t \); addictive stock, \( A_t \); and treatment choices over time that form a stock of capacity to moderate drinking, \( M_t \), which operates through a reduction in the marginal utility of alcohol. \( \alpha(A_t, M_t) \) represents the component of the marginal utility of drinking \( (a_t) \) that is
conditional on the addictive stock \((A_t)\), a standard feature in rational addiction models, and on previous treatment \((M_t)\).

The individual receives negative utility from experiencing any level of AUD, \(\lambda(A_t)\), which is a function of his addictive stock \((A_t)\). The AUD effect can be conceptualized as the subjective disutility of having a high addictive stock, negative psychic consequences of abuse, or guilt and frustration over drinking behaviors. Addictive stock itself is not a negative per se. The negatives associated with a high addictive stock are captured entirely by the AUD construct. For simplicity, the per-period utility function is constructed as a loss function to incorporate \(\lambda(A_t)\) and I begin by focusing on its implications for consumption of alcohol. For notational ease in the utility function I am excluding \(Q_t\) which is not an explicit factor in his utility, \(X_t\) a vector of individual characteristics and \(Z_t\), a vector of local environmental variables.

\[
U(a_t, l_t, c_t; A_t, M_t, \alpha_t, \lambda_t) = \mu(a_t, \alpha_t(A_t, M_t), l_t, c_t) + \lambda(A_t) \tag{1}
\]

\(U(a_t, l_t, c_t; A_t, M_t, \alpha_t, \lambda_t)\) is an increasing function of current drinking, leisure and other consumption and exhibits non-decreasing concavity for each. An individual can only drink a certain total amount in a given period: \(a \in (0, a^\text{max})\);

\[
\frac{\delta \mu(a_t, \alpha(A_t, M_t), l_t, c_t)}{\delta a_t} \geq 0; \quad \frac{\delta^2 \mu(a_t, \alpha(A_t, M_t), l_t, c_t)}{(\delta a_t)^2} \leq 0; \quad \forall l_t, c_t, A_t, M_t
\]

An individual’s marginal utility of alcohol consumption increases with the size of \(\alpha(A_t, M_t)\):

\[
\frac{\delta^2 \mu(a_t, \alpha(A_t, M_t), l_t, c_t)}{\delta \alpha_t \delta a_t} \geq 0 \forall a_t \tag{2}
\]

The general optimization problem is defined as

\[
\max \{a_t, m_t, l_t, c_t\} \sum_{t=1}^{T} \beta^t \left[ \mu(a_t, \alpha(A_t, M_t), l_t, c_t) + \lambda(A_t) \right] \tag{3}
\]
subject to two laws of motion and a budget constraint that does not include borrowing:

\[ A_t = A(a_{t-1}, A_{t-1}, \delta^A) \]  
(4)

\[ M_t = M(m_{t-1}, M_{t-1}, \delta^M) \]  
(5)

\[ p_{a,t} * a_t + p_{m,t} * m_t + c_t = w_t((O - l_t) + N_t) \]  
(6)

\[ w_t = w(Q_t, X_t, Z_t) \]  
(7)

\( \beta \) is a constant discount factor, \( p_{a,t} \) and \( p_{m,t} \) are the prices of alcohol and treatment, respectively, \( w_t \) is the individual’s wage if working, and \( N_t \) is any non-labor income.

Both the stock of addiction and the stock of treatment are positive functions of past drinking and treatment seeking and they depreciate at respective rates, \( \delta^A \) and \( \delta^M \). Consistent with the standard rational addiction framework, current drinking increases the marginal utility of future drinking as an increase in stock the following period. The stock of drinking \( (A_t) \) increases the marginal utility of drinking via \( \alpha(A_t, M_t) \):

\[ \frac{\delta \alpha(A, M)}{\delta A} > 0, \forall A, M \]  
(8)

Past treatment \( (M_t) \) also influences the marginal utility of drinking via \( a_t \):

\[ \frac{\delta \alpha(A, M)}{\delta M} < 0, \forall M, A \]  
(9)

\( \lambda(A_t) \) is the negative utility due to the current stock of drinking consumption \( (A_t) \). \( A_t \) is a function of recent drinking and the previous stock. The negative effect of the stock of drinking on utility is

\[ \frac{\delta \lambda(A)}{\delta A} > 0, \forall A \]  
(10)
In this simplified model, $t_i$ is not a function of current or past treatment ($M_t, m_t$). Treatment will be found effective only if its lagged clinical effect on drinking is greater than any short run increases in drinking due to treatment’s palliative effects.

The drinking component of the model is consistent with Becker and Murphy’s (BM) rational addiction theory of substance use in that preferences are conditional on an individual’s previous alcohol consumption. The model allows the individual to influence the value of future drinking through current drinking and current consumption of treatment. Specifically, current treatment reduces the marginal utility of future drinking, conditional on the addictive stock. Thus, the individual uses current treatment to manage the tradeoff between the future utility from drinking and the future disutility of the AUD. In the BM model, the motivation to reduce consumption is based on the secondary consequences of chronically high levels of drinking such as health, productivity, crime, or changes in the price of alcohol. By emphasizing the per se disutility of having an AUD, the model diverges from BM conceptually but is not inconsistent in its general predictions. Labor market is the only consequence that I explicitly include in the model and is described below.

Clinical treatment, self-help, and pharmacotherapy are special cases of the general effort spent to reduce drinking introduced by BM. In this model I ignore any non-formal treatment for three reasons. First, I do not observe any informal efforts. Second, all of the study subjects have engaged in formal treatment at some point in their lives. Almost all formal treatment has some component of therapy that teaches individuals behaviors and habits to help them control their drinking and prevent relapse. All treatment requires a

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8 A case might be made for treatment ameliorating the current psychic disutility of the past drinking (e.g., a reduction in guilt, medication to help with detoxification). Any direct effect of current treatment on $t_i$ might further improve the case for rationally demanded treatment but would unnecessarily complicate the theoretical model and might have an ambiguous effect on long run drinking. Such treatment may improve the short run welfare of the drinker, but could be contrary to the goal of ‘curing’ addiction since it is lowering the ‘cost’ or disutility of problem drinking. Consumption of treatment with the intention of directly reducing current $t_i$ is of little interest to public health advocates and third-party payers. Moreover, the goal of modeling treatment in this study is to estimate its impact on longer run (typically greater than a few months) AUDs. In the empirical specification defined in Section ZZ, the longer run net effect of treatment on AUDs will be estimated, implicitly testing which of treatment’s dual effects is greater.
component of personal effort and encourages ongoing personal effort. Therefore, it becomes difficult to disentangle pure personal effort from any ongoing treatment effect. Finally, because consumption of formal treatment, and especially pharmacotherapy, can be more easily encouraged by policy-makers, estimates of its effect are of greater interest.

The BM rational addiction framework does not by itself predict observed many drinking and treatment seeking behaviors. The individuals I am studying begin with a high stock of drinking (\( A_0 > A^{\text{High}} \)) that is problematic enough that they have sought treatment to improve their remaining lifetime utility. Having a high but undesirable \( A_0 \) within RA has been explained by Orphanides and Zervos (OZ) (1995). An individual is uncertain about what type of addictive personality he has, i.e., his preferences \( \mu(\alpha, a(A, M_i)) \), until after he drinks. If treatment was effective and assuming he now perfectly knows his preferences, then he would never choose to return to a bad state of \( A > A_0 = A^{\text{High}} \). In this study’s data, all individuals are at a similar point in which they have recognized that they have a drinking problem and wish to improve their remaining lifetime utility by seeking treatment. In an OZ model with stable preferences there are three possible types of steady states for individuals with a high \( A_0 \) who have discovered their addiction type and who have sought treatment. 1) Individuals may continue drinking at a level consistent with \( A_0 \) because treatment did not actually work. For others treatment works and, knowing their preferences and the long run consequences, they either (2) never drink again or (3) they achieve a steady state of ‘moderate’ drinking.\(^{10}\) The BM model does allow for changes in stable consumption patterns in response to new information about real or expected consequences: e.g., price differences for the addictive or other consumption goods, physical health outcomes, or criminal justice considerations.

\(^9\) Arguably, all personal effort after treatment is more productive because of the ‘technology’ acquired in treatment.\(^{10}\) Zinberg (1984) referred to controlled and sustained use of potentially addictive drugs as ‘chipping’ away. The BM RA model is consistent with chipping at controlled levels of consumption and allowed for preferences for which optimal \( a_i < a_{i+1}, \forall i > t_{M^{\text{High}}} \) so that a consumer, starting with a reduced preference for an addictive good, would use it at a diminishing rate until consumption becomes zero.
Among the individuals in this study, two series of choice combinations are observed after baseline treatment that appear to be inconsistent with rational convergence to the three types of steady-states implied by the BM and OZ models. These combinations are not necessarily observed as steady-state choice combinations but are sometimes consistently observed over the three years they are in the study.

1. Individuals relapse to a period of problem drinking after one or more periods of abstinence or moderate drinking.
2. Individuals remain abstinent for multiple periods and still seek treatment.
3. Individuals relapse to abusive drinking and then abstain or drink moderately thereafter with or without seeking additional treatment.

The OZ models can be made to appear consistent with these observations by ‘stretching’ the length of its periods. We could imagine a life-cycle with only 3 periods: an initial period with the possibility of becoming addicted, a second period of ‘one-time treatment’ conditional on addiction, and a third period representing the remainder of life spent in a single steady state. After that treatment period, the individual emerges as a dependent drinker, moderate drinker or an abstainer. Exogenous influences like current or expected price changes might make relapse to heavy drinking an optimal choice for an individual. However, these relapses happen frequently within an individual in recovery (e.g., every several months) and to a large majority of individuals who complete specialty treatment. These two facts imply that outside influences are not sufficient for explaining much of the observed relapses at least over short periods of time. Overall, making the BM and OZ framework fit these observations does not help to inform a better empirical model of short run treatment decisions that may be influenced by policy.

The second period of treatment described in the preceding example encapsulates a large number of drinking and treatment decisions that culminate in a final outcome. Consumption of treatment is rarely a single treatment event but is a set of ongoing decisions that can last for variable lengths of time depending
on the individual. In this simple OZ model, those ongoing treatment decisions all fit into a single long period. This is part of an overall observation that individuals often need to seek treatment many times over during their recovery before they achieve a safe level of drinking. The purpose of this study is to explore the shorter run treatment decisions and treatment effects that are policy relevant and better reflect the use of treatment to recover. In other words, this study has the opportunity to open up the black box of the hypothetical ‘second period’ in which treatment is consumed.

The first two observations noted above are a series of joint $a_t$-$m_t$ decisions that are not explicitly predicted by the original BM-RA model\textsuperscript{11}. One strategy that I have incorporated into my model is specifying treatment as a stock that can decay or be added to rather than a state that is switched on one time. This idea of treatment stock is an intuitive fit with treatment’s mechanism of action. Treatment’s effect on the motivation or capacity to consistently overcome compulsions can diminish over time. Endogenously altered preferences regress to their earlier state: motivation wanes, cognitive behavioral techniques erode and coping mechanisms are used less frequently or are forgotten.

\textsuperscript{11} Using two separate stocks of addiction Becker and Murphy do offer an explanation of ‘binging’ behavior among individuals who are neither consistently abstinent nor addicted. However, they use the term ‘binging’ to describe periods of consumption that lead back to an addictive state at which point consumption ceases and withdrawal occurs. Their key assumption is that withdrawal is low-cost or low-pain for these individuals. Therefore, the individuals can reap all of the benefits of drinking up to the level of addiction and then abruptly withdraw from drinking to avoid the negative utility of the addiction. The key deficiency of this model is that it does not fully explain ‘short’, seemingly random periods of abusive drinking. The argument could be made that individuals learn how often or intensively they can binge without returning to addiction. However, this idea is not well supported empirically for individuals in recovery who typically regard any drinking (and certainly heavy drinking) as a ‘relapse’ that jeopardizes their expected remaining life cycle utility. The idea that individuals and clinicians refer to relapse as ‘mistakes’ was the main impetus for the development of alternative models of addiction, specifically, projection bias (cite), hyperbolic discounting (cite) and state dependent utility (Laibson, 2001). A second important limitation is that the model only assumes disutility from addictive stock that crosses a certain threshold and does not acknowledge any disutility from short run heavy use such as the individual’s sense of not being in control, guilt or concern about still having a ‘disease’. Finally, if there are individuals for whom this version of the BM model is valid, i.e., a stable pattern of on and off binging, then public policies supporting curative treatment are only relevant inasmuch as their abusive drinking is correlated with externalities such as crime, automobile accidents, or publicly funded health care utilization. Conversely, publicly supported withdrawal treatment simply enables the pattern. On a technical note, BM make very strict functional form assumptions in order to analyze the comparative statics of this model which might make its implications inconsistent with the more general form of the RA model. In my study, patients have revealed a strong preference for recovery to safe drinking levels which makes this version of the RA model less appropriate.
Similarly, the stock can be restored through booster sessions of counseling, self-help therapy or medication therapy\textsuperscript{12}. Inasmuch as treatment decays faster than the addictive stock, we may observe individuals seeking treatment while abstinent or while maintaining moderate levels of drinking. This approach can also justify relapse to problem drinking if treatment is allowed to decay faster than the addictive stock. Note that for this to happen, the individual needs to be uncertain about the relative decay rates of the two stocks or about the joint A-M threshold at which near term problem drinking became optimal.

Although this differential decay rates approach offers a reasonable model that might predict observations 1 and 2, it is still lacking in several ways. Mainly, after many periods of abstinence, $A$ should have decayed to a steady lower bound. If an individual achieves this steady state through not drinking then he would never relapse thereafter. Second, it is not representative of clinical and neurological underpinnings of addiction and relapse, a key finding of which is the difference between two types of motivations to continue consuming an addictive substance: normal cravings and compulsion (CITE). Normal cravings are well modeled by traditional RA’s addictive stock construct which reflects the intuitive concepts of tolerance, withdrawal and physical addiction. Compulsion is a separate brain function from normal cravings. Addictive stock in this sense represents changes to the brain in which someone is conditioned to respond compulsively to cues.

The third observation is the least well explained by the pure RA model. Stable and known preferences should preclude random episodes of abusive drinking. To account for these observations, I incorporate random shocks to preferences in the spirit\textsuperscript{13} of Laibson (2001) and Bernheim and Rangel (2004). Building on the neuroscience of addiction, these models allow an individual to enter into a ‘hot’ mode in

\textsuperscript{12} Again, the long run impact of medication is not just a pill taken to keep from drinking but an opportunity to desensitize oneself to relapse impulses and develop habits of thoughts and behaviors that support stable and safe substance use. A common part of traditional treatment is learning to enjoy alternative sober activities (Cite). The long run impact of pharmacotherapies has the similar outcome of facilitating sober living experiences, in particular by reducing the urge to drink in usual settings. Recovery is the habit of living sober experiences with less and less temptation to drink.

\textsuperscript{13} Bernheim and Rangel explicitly represent cue–induced hot modes as cognitive process failures, referring to these episodes as leading to behavioral ‘mistakes’ that are at odds with rational life cycle utility maximization. Laibson simply models an individual as having two different preference states.
which he feels a compulsion to use an addictive substance. In their models, the probability of realizing a ‘hot’ preference state is dependent on the addictive stock. For some individuals, the probability of entering the hot state approaches zero slowly. In this way, individuals who have abstained for long periods of time might still find themselves severely ‘tempted’ to relapse. BR's model allows an individual to choose his environment (to alter the probability of entering a hot state) by reducing their exposure to cues that induce the ‘hot’ state. Moreover, when a cue-induced relapse is anticipated individuals can pre-commit to a protective environment such as inpatient treatment. The BR model also implicitly justifies demand for treatment that is effective in the long run, including CBT, ME, and pharmacotherapy. These treatments not only train the individual to avoid cues but help him manage his reaction to cues and thus reduce the probability of entering into a hot mode.

The cue-induced relapse model is important for the face validity of this model and for framing the estimated treatment effects. However, it does not significantly alter the theoretical model; I simply include a random shock to preferences whose distribution is a positive function of $A_t$, and cues, $r_t$, and a negative function of $M_t$.

$$U(a_t, l_t, c_t; A_t, M_t, \alpha_t, \varepsilon_t^\alpha, r_t, \lambda_t) = \mu(a_t, \alpha(A_t, M_t, \varepsilon_t^\alpha(A_t, M_t, r_t)), l_t, c_t) + \lambda(A_t)$$  \hspace{1cm} (11)$$

where

$$\frac{\delta \alpha(A, M, \varepsilon^\alpha, r)}{\delta \varepsilon^\alpha} < 0, \forall A, M, \varepsilon, r$$  \hspace{1cm} (12)$$

In the BR model, the distribution of $r$ is endogenously determined as an ongoing choice of environment. I do not fully employ this part of their model as I do not observe the cues experienced. Nor do I observe the brain structure (represented as function of addictive stock) that is exposed to those cues. I do
however observe one key environment choice: employment. Individuals who are forward-looking may, at
the margin, choose employment status as a protective factor in their lives. Therefore, the distribution of the
arrival of cues during a period is a function of their employment status:

Fortunately, this does not alter empirically what I am able to estimate. The directions of the effects
of $A$ and $M$ on the random shock that would lead to relapse are the same as for their deterministic effects.
The theoretical functional form of their effectiveness is influenced by the random components $\epsilon_\alpha$ and $r_t$, so I
will be limited to estimating the composite effect of these separate mechanisms.

The sequence of the individual’s decisions each period is the following:

1. The individual begins each period with complete information about past behaviors $(Q_t, A_t, M_t)$,
current preferences, current prices for alcohol, treatment and consumption goods. He observes
his current wage offer (including no offer) which is also a function of the state variables. Finally,
he has expectations about the distributions of $\epsilon_\alpha$ and $r_t$.

2. Given this information he chooses his employment status which does not change during the rest
of the period. He makes this choice in light of prices for alcoholic beverages, treatment and gen-
eral consumption, his preferences for leisure and these consumption choices. He also knows
how his employment choice will alter those preferences deterministically and has expectations it
will influence those preferences.

3. Conditional on employment status and his observed wage, he simultaneously chooses how much
to drink during the period and how much AUD treatment to consume. These choices are made
continuously throughout the period.
4. The individual knows that the decisions made during the current period affect future preferences for alcohol and treatment, current and future productivity, the probabilities of future wage offers, the distribution of future wage offers, and future health.

The employment decision is the first phase of decision-making in each time period. At the beginning of the period, he makes an employment decision that will remain in effect during the remainder of the period. If employed he receives a wage, $w_t$. I model his utility as a function leisure, $l_t$, conditional on his state variables $Q_t, A_t, M_t$, and $\varepsilon_t$, a random utility shock known by him at the beginning of the period:

$$U_t^e = U^e(l_t \mid Q_t, A_t, M_t, \alpha_t, \lambda_t, \varepsilon_t)$$  \hspace{1cm} (13)

s.t.

$$w_t = w(Q_t, X_t, Z_t)$$  \hspace{1cm} (14)

The decision to be employed or not depends on the tradeoffs between current leisure loss, current consumption which will be modeled explicitly as a second phase of within-period decision-making, and the expected net present value of his future utility conditional on employment status today.

The employment decision remains fixed throughout the remainder of the period. The primary rationale for this is that employment decisions are made over longer time horizons while drinking and treatment seeking and adherence decisions are made more frequently. Altering employment status exacts higher transactions costs than altering drinking and most treatment decisions. Employment’s main effect on drinking and treatment decisions occurs through increased income. Forward-looking individuals may make long term employment choices in anticipation of expected consumption. Secondary effects of employment on drinking, such as stress or social activities, are justified by the same rationale.
One concern with this construction is that drinking may result in termination from employment or a quit. Even though such an event occurs within a period, if the drinking has a lasting effect on employment then it will be captured by the resulting employment status in the next time period.\(^{14}\)

I model consumption choices conditional on employment status and the state variables. The individual chooses alcohol, treatment for alcohol dependence and general consumption to maximize expected lifetime utility. Treatment sought during this period enters the utility function only indirectly through the budget constraint. I define this per period consumption utility function as

\[
U_{t}^{a,m} = U^{a,m}(a_{t}, c_{t} \big| Q_{t}, A_{t}, M_{t}, \alpha_{t}, \lambda_{t}, \varepsilon_{t}^{a}, r_{t}, l_{t}, w_{t}, \varepsilon_{t}^{a,m})
\]

s.t.

\[
A_{t} = A(a_{t-1}, A_{t-1}, \delta^{A})
\]

\[
M_{t} = M(m_{t-1}, M_{t-1}, \delta^{M})
\]

\[
p_{a,t}a_{t} + p_{m,t}m_{t} + c_{t} = w_{t}(O - l_{t}) + N_{t}
\]

where \(\varepsilon_{t}^{a,m}\) is a vector of unobserved random shocks to utility.

IV. Empirical Model

I define \(Q_{t}\), employment history, as a vector of several different relevant measures: number of periods since being employed which will be 0 if employed in the previous period, total lifetime employment (experience), number of periods employed during most recent employment (tenure). I do not

\(^{14}\)Terminations and quitting due to alcohol can happen with little apparent impact on employment probability. In the longer run, a labor market history like that can reduce both the probability of employment and wage, but this study does not attempt to directly identify this effect.
observe the actual addictive stock, \( A_t \). Therefore, I define \( A_t \) to be a vector of different drinking measures that capture different consumption patterns up through the current period. These include time proportion of recent time engaged in problem drinking defined as either heavy drinking days or weeks of intensive drinking defined as greater than fourteen drinks in a seven day period. These implicitly represent drinking in the most recent period. Alternative measures that I will explore will include time since last drink (time abstinent), percent of days drinking of heavy drinking in the previous period, and percentage of heavy drinking weeks in the previous period. Although \( M_t \) theoretically represents a stock of treatment in the theoretical model number of days taking medications to avoid drinking, and the evolving ‘stock’ of treatment will be constructed as time since a given intensity of treatment. All measures are standardized by the time during which they were counted (e.g., number of days of heavy drinking, number of days of pharmacotherapy). These variables are formally defined in Table 2 and are discussed in greater detail in Section V.

The vector \( Z_t = \{Z_t^e, Z_t^a, m\} \) contains local (metropolitan statistical area, MSA) unemployment rates, wages for major industry types and prices for beer, liquor, doctor visits, gas and other consumption good prices by quarter. Also included are local specialty substance use treatment system variables by year. They are per capita counts of available outpatient and inpatient treatment (i.e., total sessions or total beds), differentiated by whether the provider accepts different types of insurance or charges according to a sliding scale. I include annual national average prices for pharmacotherapies. Finally, I include self-reported travel times to treatment or other medical facilities. The vector \( X_t \) contains individual characteristics of gender, age, race, education, marital status, and non-labor income.

The employment outcome in each period is defined as four mutually exclusive categories:
These categories were defined based on observed data described in Section V and are intended to capture the variation in meaningful outcomes. The probability of \( e_t \) being realized is expressed as

\[
\Pr( E_t = e ) = \frac{\exp( \delta_0^e + \delta_1^e Q_t + \delta_2^e A_t + \delta_3^e M_t + \delta_4^e A_t * M_t + \delta_5^e Z_t + \delta_6^e X_t )}{\sum_{e=0}^{3} \exp( \delta_0^e + \delta_1^e Q_t + \delta_2^e A_t + \delta_3^e M_t + \delta_4^e A_t * M_t + \delta_5^e Z_t + \delta_6^e X_t )}
\]  

The employment outcome depends on work history and exogenous labor market conditions, \( Z_t \). It also depends on preferences for consumption and the prices of consumption. Past drinking and past treatment are key pieces of information on his preferences for drinking and the need for ongoing treatment. Moreover, accumulated and recent drinking might also have had a negative impact on the current employment outcome that is independent of observed labor market history. For example, a decrease in unobserved employer perception of his productivity might lead to a layoff.

Consider a simple dichotomous outcome \( a_t \) that is 1 if the individual engages in abusive drinking, defined as either one or more days of heavy drinking or at least one heavy drinking week.

\[
\Pr( a_t = a ) = \frac{\exp( \delta_0^a + \delta_1^a Q_t + \delta_2^a A_t + \delta_3^a M_t + \delta_4^a A_t * M_t + \delta_5^a Z_t^{a,m} + \delta_6^a X_t + \delta_7^a E_t + \delta_8^a w_t )}{\sum_{a'=0}^{1} \exp( \delta_0^a + \delta_1^a Q_t + \delta_2^a A_t + \delta_3^a M_t + \delta_4^a A_t * M_t + \delta_5^a Z_t^{a,m} + \delta_6^a X_t + \delta_7^a E_t + \delta_8^a w_t )}
\]  

In accordance with the theoretical framework, conditional on employment status, \( E_t \), current drinking is a function of past drinking \( (A_t) \) a proxy for addictive stock, previous treatment \( (M_t) \), which has ongoing behavioral effects independent of the stock of drinking, and the interaction of past drinking and past
treatment \( (A_t^* M_t) \) which should capture the effect on current drinking of past treatment on the marginal effect of past drinking. In addition, drinking is dependent on prices and the current wage if employed. In addition, employment has an effect on drinking independently of the wage. Employment’s effect subsumes several possible effects that cannot be separately observed or identified. Employment might be a protective factor as a relatively safe, cue-free environment. It might shift preferences for drinking by increasing self-esteem and reducing general anxiety about livelihood. Alternatively, employment could increase preferences for drinking to relieve stress. The vector \( Z_i^{a,m} \) captures the local market prices and other characteristics but excludes the labor market conditions (unemployment rate and wages). Conditional on being employment, these factors theoretically would not influence the drinking and treatment decisions.

Let \( m_t \) be defined as 2 mutually exclusive categories:

\[
m_t = \begin{cases} 
0 & \text{if no outpatient or pharmacotherapy is consumed} \\
1 & \text{if any outpatient or pharmacotherapy is consumed}
\end{cases}
\]  

Then the probability of choosing treatment combination \( m \) is

\[
\Pr( M_t = 1) = \frac{\exp( \delta_0^m + \delta_1^m Q_t + \delta_2^m A_t + \delta_3^m M_t + \delta_4^m A_t^* M_t + \delta_5^m Z_t^{a,m} + \delta_6^m X_t + \delta_7^m E_t + \delta_8^m w_t) }{\sum_{m=0}^{1} \exp( \delta_0^{m'} + \delta_1^{m'} Q_t + \delta_2^{m'} A_t + \delta_3^{m'} M_t + \delta_4^{m'} A_t^* M_t + \delta_5^{m'} Z_t^{a,m} + \delta_6^{m'} X_t + \delta_7^{m'} E_t + \delta_8^{m'} w_t) }
\]

During the period, the individual chooses to consume treatment to alter the long run preferences for drinking. Wage income affects demand for any of the treatment options. Current employment, denoted as \( E_i \), influences the treatment choice along multiple pathways. While I do not observe insurance status, employment may be associated with insurance and increase the demand for treatment. Alternatively, working reduces the amount of time available for treatment.

\textit{Estimation}
I estimate the three main equations jointly using the discrete factor method (DFM), a flexible random effects estimation technique (Heckman and Singer, 1984; Mroz and Guilkey, 1992; and Mroz, 1994) in which individual time varying and time invariant heterogeneity components of each equation’s error term are model across all equations. Specifically,

$$\begin{align*}
\varepsilon_{1,t}^e &= \mu_1^e + \nu_{1t}^e + e_{1t}^e \\
\varepsilon_{2,t} &= \mu_2 + \nu_{2t} + e_{2t} \\
\varepsilon_{3,t} &= \mu_3 + \nu_{3t} + e_{3t}
\end{align*}$$

Where the $\mu_j$ are permanent unobserved individual heterogeneity, $\nu_{jt}$ are individual time varying heterogeneity and the $e_{jt}$ are the idiosyncratic errors.

Assuming that the $\varepsilon_{1,t}^e$ are additively separable, mutually independent, and Type-I Extreme Value distributed error terms, the log odds ratio of $E_t = e$ relative to the outcome $E_t = 0$, conditional on is $\mu_1^e + \nu_{1t}^e$.

$$\ln \left[ \frac{\Pr( E_t = e )}{\Pr( E_t = 0 )} \right] = \delta_0^e + \delta_1^e Q_t + \delta_2^e A_t + \delta_3^e M_t + \delta_4^e A_t \ast M_t + \delta_5^e Z_t + \delta_6^e X_t + \mu_1^e + \nu_{1t}^e \quad (21)$$

yielding a multinominal logit estimation specification. The logit model of engaging in abusive drinking, conditional on $\mu_2^a + \nu_{2t}^a$ is

$$\ln \left[ \frac{\Pr( a_t = 1 )}{\Pr( a_t = 0 )} \right] = \delta_0^a + \delta_1^a Q_t + \delta_2^a A_t + \delta_3^a M_t + \delta_4^a A_t \ast M_t +$$

$$\delta_5^a Z_t + \delta_6^a X_t + \delta_7^a E_t + \delta_8^a W_t + \mu_2^a + \nu_{2t}^a$$

With assumptions about $\varepsilon_{3,t}^m$ similar to those for $\varepsilon_{1,t}^e$.
\[
\ln \left[ \frac{\Pr(M_t = m)}{\Pr(M_t = 0)} \left| \mu_3^m + \nu_3^m \right] \right] = \\
\delta_0^m + \delta_1^m Q_t + \delta_2^m A_t + \delta_3^m M_t + \delta_4^m A_t \ast M_t + \\
\delta_5^m Z_t a^m + \delta_6^m X_t + \delta_7^m E_t + \delta_8^m \nu_t + \mu_3^m + \nu_3^m
\] (25)

yields the log odds of each treatment choice. An individual’s likelihood contribution is thus

\[
L_i(\Theta, \psi | \mu_1, \mu_2, \mu_3) = \left[ \Pr(a_1 = 1 | \mu_2) a_1 (1 - \Pr(a_1 = 1 | \mu_2))^{1-a_1} \right] \\
\times \left[ \Pr(m_1 = 1 | \mu_3) m_1 (1 - \Pr(m_1 = 1 | \mu_3))^{1-m_1} \right] \\
\times \prod_{e=0}^3 \Pr(E_i = e | \mu_1^e, \nu_1^e) \ast I[E_i = e] \\
\times \prod_{t=1}^7 \sum_{l=1}^L \psi_l \\
\times \left[ \Pr(a_{t+1} = 1 | \mu_2, \nu_2) a_{t+1} (1 - \Pr(a_{t+1} = 1 | \mu_2, \nu_2))^{1-a_{t+1}} \right] \\
\times \left[ \Pr(m_{t+1} = 1 | \mu_3, \nu_3) m_{t+1} (1 - \Pr(m_{t+1} = 1 | \mu_3, \nu_3))^{1-m_{t+1}} \right]
\]

where \( \Theta \) is the vector of variables that will be estimated. The distribution of \( \psi \) is

\[
\psi = \Pr(\nu_{1t}^e = \nu_{1t}^e, \nu_{2t} = \nu_{2t}, \nu_{3t} = \nu_{3t}) \ \forall l = 1, \ldots, L, \forall e
\]

Where \( L \) is the number of time varying mass points. The likelihood function for the sample is thus written as

\[
L(\Theta, \psi, \pi) = \prod_{i=1}^N L_i(\Theta, \psi, \pi)
\]

In which

\[
L_i(\Theta, \psi, \pi) = \sum_{k=1}^K \pi_k \ast L_i(\Theta, \psi | \mu_{1k}, \mu_{2k}, \mu_{3k})
\]

And the distribution of the permanent unobserved heterogeneity is
\[\pi_k = \Pr(\mu_{1k} = \mu_{1k}^e, \mu_{2k} = \mu_{2k}^e, \mu_{3k} = \mu_{3k}^e) \quad \forall l = 1, \ldots, L, \forall e\]

**Initial Conditions**

The initial period likelihoods \(\Pr(a_l = 1 | \mu_2)\) and \(\Pr(m_l = 1 | \mu_3)\) show that beginning drinking and treatment choices are functions of permanent heterogeneity and the state variables \(A_0\) and \(M_0\). Since I do not observe the pre-sample choices leading to these states I will estimate them within the likelihood function above as reduced form functions of historical variables. Specifically, the starting state variables \(A_0\) and \(M_0\) will be estimated using randomized treatment assignment that was completed before my study begins as well as historical prices, historical treatment capacity, policy variables that have varied over time interacted with an individual’s age, and family drinking history variables. The policy variables will include minimum legal drinking laws and insurance parity laws.\(^{15}\)

**V. Data**

The main inclusion criterion for the study included a DSM-IV diagnosis of alcohol dependence. Participants were excluded if another substance was deemed to be the primary drug of dependence, had a severe psychiatric illness or had certain serious physical conditions (COMBINE Protocol). The COMBINE trial randomized 1,383 participants to nine different combinations of two pharmacotherapies (Acamprosate and Naltrexone) and one Cognitive Behavioral Intervention. The trial also included Medication Management for all but one group that received no pills or placebos. Randomization took place within eleven different treatment sites in the United States between 2001 and 2003. Treatment lasted for sixteen weeks after which the individuals’ only study experience was incentivized follow-up data collection. Data collection continued at four-month intervals for three years after randomization for a subset of

\(^{15}\) I will not actual have data on migration between states in the past. Nonetheless, as policy changes over time represent broader national changes these might still be useful covariates. I am still hoping to obtain some genetic predisposition data from the study.
willing participants whose data was collected for an economic study of COMBINE. Only nine of the original eleven sites chose to continue data collection for the economic study. This study estimates models using only responses from interviews that occurred after the 16 week study treatment because of inconsistent data collection techniques and questions between the study period and the follow-up period and uncertainty about the measurement of self-reported treatment seeking decisions during a clinical trial. Table 2. describes the sample size and observations of the original COMBINE study. Of 991 participants who completed 16 weeks of treatment in the nine sites that continued the study, 792 chose to participate in the three-year economic study and completed 6,138 interviews. Attrition within this group was limited.\textsuperscript{16} Moreover, there were relatively few missing interviews because the data collection instruments were designed to capture outcomes since the previous interview. The clinical staff conducting the interviews were trained in techniques to improve recollection (COMBINE Protocol). In accordance with the original study, if too much time passed between interviews, the clinical staff attempted to reconstruct the outcomes as of the time of the missed interview. Finally, there was virtually no non-response to any particular question in the primary data collection instrument other than logical survey skips. The current analysis sample is 775 individuals with 5,769 interviews after removing inconsistent or missing observations and individuals with fewer than four of the eight possible follow-up interviews. Of these, 601 interviews were flagged as having been reconstructed. A third column of Table 1 describes the number of observations in which patients responded to a second survey instrument that was administered less frequently but which has several useful measures described below.

One of the strengths of this study is the quality of its measures. The data were collected using the Economic Form 90 instrument (Bray et al, 2007) which asked about labor market outcomes, sub-

\textsuperscript{16} The reasons include the frequency of follow-up interviews, incentives, the rapport established between the study participants and the study staff during the main study period, and the amount of grant resources provided to the study sites to support data collection. Finally, the participants eligible for the follow-up study had successfully completed 16 weeks of study treatment and thus may have been selected on unobserved characteristics that were correlated with study adherence.
stance use and mental health treatment seeking and health since the last interview. All treatment information collected by the Form 90 refers to self reported treatment seeking and is unrelated to COMBINE study treatment. The Form 90 also collected detailed alcohol use using a calendar follow-back method which is considered the most accurate survey method for substance use. The length of the time since the previous interview averages four months. This is useful because it not only supports a longitudinal and dynamic statistical model but it also supports a theory-driven statistical model that depends on simultaneous alcohol consumption and treatment decisions. In other words, it supports modeling the key behaviors that theory and common sense would predict. No other survey contains such detailed measures of treatment use, substance use, and employment outcomes over such fine periods of time. The second instrument of interest is the World Health Organization’s Quality of Life (WHO-QoL) that yields validated and reliable scales of physical, mental and environmental health.

Table 3 describes the characteristics of the 775 individuals in the analysis sample. Marital status and Education variables are mutually exclusive. Of note, this sample is fairly well educated with fewer than 7% not having achieved a high school degree or better, and over 40% having a college degree or higher. Likewise, they have relatively high wages. These facts are consistent with the substance abuse literature in that they are a high functioning group of individuals. This is particularly true of substance abusers who do not have a co-occurring severe psychiatric condition, criteria by which this sample was selected.
References


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George MS et al. Activation of Prefrontal Cortex and Anterior Thalamus in Alcoholic Subjects on Exposure to Alcohol-Specific Cues. *Archives of General Psychiatry.* April 2001;58:345-352.


Grüsser SM et al. Cue-Induced Activation of the Striatum and Medical Prefrontal Cortex is Associated with Subsequent Relapse in Abstinent Alcoholics. *Psychopharmacology.* 2004;175:296-302.


Sher L. Depression and Alcoholism. QJMEd. 2004;97: -240.


### Table 1. Determination of Sample

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Number of Individuals in Sample</th>
<th>Number of Observations</th>
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<tr>
<td>Randomized into COMBINE Study Groups</td>
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<tr>
<td>Completed COMBINE Study Treatment</td>
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<tr>
<td>Completed COMBINE Study Treatment in the 9 Continuing Sites</td>
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<tr>
<td>Participated in 3 Year Economic Data Collection</td>
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<td><strong>Time Periods</strong></td>
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<tr>
<td>0 - Prior to Randomization</td>
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<td>775</td>
</tr>
<tr>
<td>1 - Randomization to End of Study Treatment</td>
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<td>775</td>
</tr>
<tr>
<td>2 - After Study Treatment - Data Collection</td>
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<tr>
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<td>Final Analysis Sample After End of Study</td>
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Table 2. Variable Definitions

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<th>Employment Outcomes</th>
<th>Description</th>
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<td>Not Employed</td>
<td>Reported no days of work during entire period.</td>
</tr>
<tr>
<td>Employed Less Than Full Period</td>
<td>Employed less than 90% of the length of the period</td>
</tr>
<tr>
<td>Employed Full Period, Part-time</td>
<td>Employed more than 90% of the length of the period, ( \geq 35 ) hours per week</td>
</tr>
<tr>
<td>Employed Full Period, Full-time</td>
<td>Employed more than 90% of the length of the period, (&lt; 35 ) hours per week</td>
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<table>
<thead>
<tr>
<th>Current Alcohol Consumption Profile</th>
<th>Description</th>
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<tr>
<td>Abstinent</td>
<td>Dichotomous variable equal to 1 if days of drinking were reported during the period.</td>
</tr>
<tr>
<td>Percent of Days Drinking</td>
<td>Total number of days of reported drinking divided by the period length.</td>
</tr>
<tr>
<td>Any Heavy Drinking</td>
<td>Dichotomous variable equal to 1 if any days of heavy drinking were reported during the period.</td>
</tr>
<tr>
<td>Percent of Weeks Heavy Drinking</td>
<td>Number of weeks with any reported heavy drinking divided by the period length.</td>
</tr>
<tr>
<td>Any Intensive Drinking Weeks</td>
<td>Dichotomous variable equal to 1 if more than 14 drinks were consumed during any week in the period.</td>
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<tr>
<td>Percent of Weeks Heavy Drinking</td>
<td>Number of weeks in which more than 14 drinks were consumed divided by the period length.</td>
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<td>Variable</td>
<td>Mean (Std. Dev.)</td>
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<tr>
<td>-----------------------------------------------</td>
<td>------------------</td>
</tr>
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<td>775</td>
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<tr>
<td>Female</td>
<td>.302 (.459)</td>
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<tr>
<td>Age</td>
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<td>Black</td>
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<td>Other Race</td>
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<tr>
<td>Married or Cohabitating</td>
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<td>Divorced, Separated, or Widowed</td>
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</tr>
<tr>
<td>High School Degree</td>
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<tr>
<td>Undergraduate Degree</td>
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<tr>
<td>Graduate Degree</td>
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<tr>
<td>Any Non-labor Income</td>
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<tr>
<td>Conditional Non-labor Income ($/Month)</td>
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</tr>
<tr>
<td>Currently Employed Fulltime (&gt;=35 Hours/Week)</td>
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</tr>
<tr>
<td>Fulltime Conditional Wage ($/Hour)</td>
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<tr>
<td>Currently Employed Parttime (&lt;35 Hours/Week)</td>
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<tr>
<td>Parttime Conditional Wage ($/Hour)</td>
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<td>Employed in Past 90 days and Not Currently</td>
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<td>Employed</td>
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Table 3. Sample Summary Statistics at Time of Enrollment in COMBINE Trial
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<th>Period 5</th>
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<td><em>N</em></td>
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<table>
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<th><em>N</em></th>
<th><em>N</em></th>
<th><em>N</em></th>
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Table A: Employment Proportions (5-Year) By Time Period and Age Category for Full Sample
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<th>5</th>
<th>6</th>
<th>7</th>
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<th>9</th>
<th>10</th>
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<th>12</th>
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<td>0-10</td>
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<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
<td>F</td>
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<td>O</td>
<td>P</td>
<td>Q</td>
<td>R</td>
<td>S</td>
<td>T</td>
<td>U</td>
<td>V</td>
<td>W</td>
<td>X</td>
<td>Y</td>
<td>Z</td>
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Table 5: Reproductive and Condensation Properties of Female (SU) of Drying Outcomes by Time and Application for Laboratory Samples.
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>All Periods</th>
<th>Periods 2-8</th>
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<tbody>
<tr>
<td>N</td>
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<td>775</td>
<td>775</td>
<td>775</td>
<td>775</td>
<td>745</td>
<td>704</td>
<td>650</td>
<td>570</td>
<td>6544</td>
<td>4994</td>
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<tr>
<td>Naltrexone Provided by Study - Not Placebo</td>
<td>0.001</td>
<td>0.043</td>
<td>0.057</td>
<td>0.066</td>
<td>0.067</td>
<td>0.059</td>
<td>0.058</td>
<td>0.054</td>
<td>0.058</td>
<td>0.056</td>
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<td></td>
<td>(2.0)</td>
<td>(2.0)</td>
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<td>(13.9)</td>
<td>(11.3)</td>
<td>(11.5)</td>
<td>(14.3)</td>
<td>(14.6)</td>
<td>(10.6)</td>
<td>(13.9)</td>
<td>(5.0)</td>
</tr>
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<td>90</td>
<td>90</td>
<td>90</td>
<td>90</td>
<td>90</td>
<td>90</td>
<td>90</td>
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<tr>
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<td>0.059</td>
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<td>0.054</td>
<td>0.058</td>
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<td>(8)</td>
<td>(13.9)</td>
<td>(11.3)</td>
<td>(11.5)</td>
<td>(14.3)</td>
<td>(14.6)</td>
<td>(10.6)</td>
<td>(13.9)</td>
<td>(5.0)</td>
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<tr>
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<td>0.450</td>
<td>0.450</td>
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<td>(1.9)</td>
<td>(1.9)</td>
<td>(1.9)</td>
<td>(1.9)</td>
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<td>(1.9)</td>
<td>(1.9)</td>
<td>(1.9)</td>
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</tr>
<tr>
<td>Acamprosate Provided by Study - Placebo</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
<td>0.434</td>
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<tr>
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<td>(1.9)</td>
<td>(1.9)</td>
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<td>(1.9)</td>
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<tr>
<td>Acamprosate - Private^d</td>
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<td>0.019</td>
<td>0.019</td>
<td>0.019</td>
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<td>0.023</td>
<td>0.026</td>
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<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
<td>(0.121)</td>
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<td>Disulfiram - Private</td>
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<td>0.015</td>
<td>0.018</td>
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<td>0.023</td>
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<td>0.025</td>
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<td>Other^e - Private</td>
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<td>0.097</td>
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<td>Any Medication Consumed to Prevent or Control Drinking - Excluding Placebo</td>
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<td>0.474</td>
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<td>0.592</td>
<td>0.746</td>
<td>0.773</td>
<td>0.676</td>
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<td>0.777</td>
<td>0.498</td>
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<td>(0.090)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.042)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.047)</td>
</tr>
<tr>
<td>Any Medication Consumed to Prevent or Control Drinking - Including Placebo</td>
<td>0.018</td>
<td>0.496</td>
<td>0.749</td>
<td>0.592</td>
<td>0.746</td>
<td>0.773</td>
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<td>0.810</td>
<td>0.777</td>
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<td>(0.042)</td>
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<td>(0.042)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.042)</td>
<td>(0.047)</td>
<td>(0.047)</td>
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</tbody>
</table>

Notes: a. Sample includes individuals who did not complete all interview waves.  
b. Time period 1 covers from study randomization through the end of COMBINE study treatment.  
c. Represents prescription use of individuals during the period after COMBINE treatment ended.  
e. Includes Quetiapine, Topiramate, Gabapentin and Neurontin when prescribed for alcohol use.
Table 5. Transition Probabilities in Primary Outcome Categories within Individuals over Time

<table>
<thead>
<tr>
<th>Outcome Category in Period</th>
<th>Prop of Individuals in Outcome Category in Period</th>
<th>Not Employed</th>
<th>Employed Less Than Full Period</th>
<th>Employed Full Period, Part-time</th>
<th>Employed Full Period, Full-time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not Employed (16.4)</td>
<td></td>
<td>0.771</td>
<td>0.163</td>
<td>0.033</td>
<td>0.033</td>
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<tr>
<td>Employed Less Than Full Period (17.4)</td>
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<td>0.168</td>
<td>0.393</td>
<td>0.141</td>
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<tr>
<td>Employed Full Period, Part-time (12.6)</td>
<td></td>
<td>0.051</td>
<td>0.175</td>
<td>0.590</td>
<td>0.184</td>
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<tr>
<td>Employed Full Period, Full-time (53.5)</td>
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<td>0.015</td>
<td>0.102</td>
<td>0.038</td>
<td>0.845</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Abstinent (26.1)</th>
<th>Abstinent, No Problem Drinking (0.26)</th>
<th>Problem Drinking, Less than Entire Period (0.12)</th>
<th>Problem Drinking, During Entire Period (0.02)</th>
</tr>
</thead>
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<tr>
<td>0.813</td>
<td>0.057</td>
<td>0.128</td>
<td>0.002</td>
</tr>
<tr>
<td>0.153</td>
<td>0.504</td>
<td>0.305</td>
<td>0.038</td>
</tr>
<tr>
<td>0.143</td>
<td>0.073</td>
<td>0.560</td>
<td>0.223</td>
</tr>
<tr>
<td>0.008</td>
<td>0.018</td>
<td>0.271</td>
<td>0.703</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No Medication Consumed to Prevent or Control Drinking (78.1)</th>
<th>Medication Consumed to Prevent or Control Drinking (21.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.977</td>
<td>0.023</td>
</tr>
<tr>
<td>0.624</td>
<td>0.376</td>
</tr>
</tbody>
</table>

Notes: a. The sample is 775 individuals. Calculations are based on 4,994 observations. The \( t \) periods are 1 through 7, \( t + 1 \) are 2 through 8. b. Problem drinking includes either a week in which a heavy drinking day occurred (5+ drinks for men, 4+ for woman) or a week in which 14 or more drinks were consumed.