Rational Self-Medication*

Michael E. Darden
Johns Hopkins University and NBER

Nicholas W. Papageorge
Johns Hopkins University, IZA and NBER

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Abstract

We develop a theory of rational self-medication. The idea is that forward-looking individuals, lacking access to better treatment options, attempt to manage the symptoms of mental and physical pain outside of formal medical care. They use substances that relieve symptoms in the short run but that may be harmful in the long run. For example, heavy drinking could alleviate current symptoms of depression but could also exacerbate future depression or lead to alcoholism. Rational self-medication suggests that, when presented with a safer, more effective treatment, individuals will substitute towards it. To investigate, we use forty years of longitudinal data from the Framingham Heart Study and leverage the exogenous introduction of selective serotonin reuptake inhibitors (SSRIs). We demonstrate an economically meaningful, arguably causal reduction in alcohol consumption when SSRIs became available. Additionally, we show that addiction to alcohol inhibits substitution. Our results suggest a role for rational self-medication in understanding the origin of substance abuse. Furthermore, our work suggests that punitive policies targeting substance abuse may backfire, leading to substitution towards even more harmful substances to self-medicate. In contrast, policies promoting medical innovation that provide safer treatment options could obviate the need to self-medicate with dangerous or addictive substances. More broadly, our findings illustrate how the effects of medical innovation operate in part through behavior changes that are not measured in clinical trials.

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1 Introduction

Beginning with Grossman (1972), economists have envisioned health as a form of human capital that increases survival rates, raises productivity, and improves the quality of life. Accordingly, behaviors that can improve health, such as exercise, healthy eating, abstaining from risky behavior, or medication usage, can be viewed as costly investments in human capital. Rational individuals invest in their health until the long-term benefits of doing so cease to outweigh the upfront costs. This basic model has been expanded upon to incorporate the realities of many health-related decisions. Examples include uncertainty and learning about how well a drug will work (Crawford & Shum, 2005), treatment decisions when faced with an acute illness (Gilleskie, 1998), and addiction that encourages use of harmful substances (Darden, 2017), among others.

Research using the Grossman framework has focused almost exclusively on investments that unambiguously improve health. This need not be the case. Some health investments can be both beneficial and harmful to health.[1] An example is *self-medication* with potentially dangerous substances.[2] Lacking better options, individuals take matters into their own hands, turning to substances that are potentially destructive (e.g., alcohol or opioids) in an effort to manage symptoms of illnesses (e.g., chronic pain or depression). Understanding how, and under what circumstances, people self-medicate is important because self-medication is socially costly, especially if it leads to addiction. Yet, focusing solely on the downsides of self-medication or treating the behavior as an error in judgment or an act of desperation — which earlier literature on self-medication has done — rather than treating it as a plausibly optimal if costly investment given prevailing technology, can lead to the wrong policy conclusions. Viewing problem drinking as purely irrational behavior suggests policies to curb drinking. Viewing it as rational self-medication suggests such policies could backfire if, for example, people substitute towards substances that are even more addictive or destructive. A better policy response would be to promote treatment innovations that obviate the need to self-medicate and thus induce rational actors to substitute towards less harmful substances.

In this paper we test the rational self-medication hypothesis. In particular, we ask whether the

[1] For example, radiation therapy is an effective way to treat cancer that increases the likelihood of sustained remission. Yet, it can actually *cause* other forms of cancer in the future (Wallis et al. , 2016). Thus, young patients with treatable forms of cancer (e.g., testicular cancer) often forgo radiation in favor of surgery.

[2] It is important to note that there are two definitions of self-medication. One encompasses any self-administered medication that is prescribed by a physician. A second definition is the use of potentially dangerous substances in order to alleviate symptoms outside of formal medical care (i.e., without a prescription). While the first is often used in the medical literature, the second is more aligned to a layperson's notion of self-medication and is also discussed at length in the psychological literature. For example, Khantzian (1985) introduces the concept of self-medication, in which an individual manages her ailment outside of formal prescription medicine or therapy. In this paper, we use the second definition.

emergence of effective medication obviates the need to self-medicate with riskier substances. In the case we study, we leverage a technological advancement — the 1988 Food and Drug Administration (FDA) approval of Selective Serotonin Reuptake Inhibitors (SSRIs) — as an arguably exogenous expansion of the choice set for the management of depression. If alcohol consumption is in part a form of self-medication, we predict that drinking should fall following the introduction of SSRIs.[3] If we are unable to detect such substitution patterns as better medications emerge, heavy drinking is less likely to be a form of self-medication. Broadly, this analysis illustrates a central contribution of health economics, which is to move beyond quantifying the direct impacts of new medicines (e.g., treatment effects on health or the harms of risky substances) by incorporating additional factors, such as uptake and compliance decisions along with substitution patterns in other potentially relevant health behaviors. In the context we examine, if alcohol is used to self medicate, a potentially overlooked social benefit of SSRIs is a reduction in heavy drinking.

Self-medication using substances such as alcohol is not a new idea and is a widely-documented phenomenon (Khantzian, 1985). It is also deeply frowned upon in public health in ways that could potentially lead to counter-productive policies. To clarify how, this paper re-assess self-medication — in part by modeling it as rational behavior and then in part by testing key model implications to provide (to our knowledge) the first causal evidence that individuals substitute away from self-medication with harmful substances to less harmful ones when they come available. We are thus able to make the following point: efforts to curb self medication that fail to recognize it as a rational behavior can lead to negative unintended consequences, such as shunting people towards even worse substances. To provide a current example, there is considerable public health concern regarding stress-induced alcohol consumption as a result of the COVID-19 pandemic (Clay & Parker, 2020). The World Health Organization has recommended that "Existing rules and regulations to protect health and reduce harm caused by alcohol, such as restricting access, should be upheld and even reinforced during the COVID-19 pandemic and emergency situations; while any relaxation of regulations or their enforcement should be avoided."[4] The rise in alcohol use during Covid may in part be due to self-medication due to alcohol and could lead to addiction problems in the future. Yet, blunt policy responses (e.g., prohibiting alcohol) could make things even worse if people turn to more addictive, illegal or dangerous substances.

To begin our analysis of self-medication we formalize the concept with a simple two-period

[3] In Section 2, we document a strong correlation between depression and heavy alcohol consumption using NHANES data, and we review the significant literature on alcohol self-medication. For example, Bacolod et al. (2017) study minimum drinking age laws and show that the largest increase in drinking at age 21 (for those in the military) comes from the most depressed.

[4] https://www.euro.who.int/en/health-topics/disease-prevention/alcohol-use/news/news/2020/04/alcohol-does-not-protect-against-covid-19-access-should-be-restricted-during-lockdown

model in which an agent makes health investment decisions, jointly choosing alcohol and antidepressant medications to maximize utility.[5] Poor mental health generates symptoms which reduce utility. Health investments have contemporaneous effects on symptoms along with inter-temporal effects on the stock of mental health. In our case, alcohol relieves current-period symptoms, but may also exacerbate future mental health problems, which produce future symptoms. The model also permits the possibility that substances, such as alcohol, are enjoyable in their own right. The key factor underlying self-medication is a complementarity: the current-period marginal benefit of substances rises with the symptoms of illness.[6] One way to achieve this is that alcohol is more effective at reducing more severe symptoms. Alternatively, if the utility cost of symptoms is larger as symptoms increase, the same reduction via alcohol has a larger utility benefit. In either case, the complementarity between alcohol and immediate symptoms of depression generates the following testable hypothesis. By decreasing symptoms and thus the marginal benefits of improving symptoms with alcohol, the introduction of SSRIs should cause alcohol usage to decline. Alternatively, if alcohol is simply an enjoyable good, it should not be affected by the introduction of SSRIs.

To test this hypothesis, we use data from the Framingham Heart Study Offspring Cohort. The data include longitudinal information on alcohol, tobacco, and antidepressant consumption, as well as depression measures, for roughly 5,000 individuals over a forty-year period. We estimate a series of within-individual estimators in which we measure how trends in alcohol consumption vary before and after the arguably exogenous arrival of SSRIs in 1988. For example, we study how these trends differ by CES-Depression score, measured prior to 1988. We also estimate models of alcohol consumption on antidepressant usage while allowing trends to vary by whether someone is ever observed to take an antidepressant. The main results from these analyses provide arguably causal evidence of substitution away from alcohol and towards antidepressants once SSRIs become available.[7] In particular, taking an antidepressant leads to a statistically significant 3.9 percentage point (12.5%) increase in abstinence from alcohol and a 9.5% reduction in the number of drinks per week conditional on drinking.[8] Effects are stronger for men and are increasing in depression.

[5] Our model formalizes the argument that the type of substance being used depends on the type and severity of mental health ailment (Khantzian, 1985).

[6] The notion that harmful substance use is explained through a complementarity is similar to Becker & Murphy (1988), who model dynamic complementarities in the marginal utility of consumption as a necessary condition for addiction. We discuss further links to this paper below.

[7] Event studies around an antidepressant prescription demonstrate statistically indistinguishable pre-trends, and our results are robust to allowing trends to vary by a variety of time-invariant characteristics, including pre-1988 depression.

[8] We note that some individuals who self-medicate with alcohol prior to the introduction of SSRIs could continue to do so after. However, this behavior is also consistent with simply enjoying alcohol rather than using it to self-medicate. Thus, individuals who reduce or stop drinking in response to SSRIs provide stronger evidence of the self-medication hypothesis.

The latter finding underscores the self-medication hypothesis since it suggests that, until better options emerge, alcohol is an effective way to combat depression.[9]

Our reduced-form results establish the average treatment effect of SSRIs; however, they ignore addiction, which could hamper substitution to SSRIs. Moreover, an implication of the theoretical model is that addictive stock is potentially endogenous to depression, which could also affect SSRI uptake. Clean identification of an interaction between addiction and substitution from alcohol to SSRIs would require exogenous variation in addiction at the time of SSRI introduction, which we do not have. Thus, to investigate a role for addiction, we augment our analysis to estimate a system of dynamic equations. Specifically, we estimate dynamic equations for alcohol, tobacco, and antidepressants jointly, along with depression, attrition, and mortality equations, and we allow for correlation in the error structure across equations to capture unobserved heterogeneity in the joint determination of these behaviors and outcomes (Heckman & Singer, 1984; Mroz, 1999).[10] The aim is to account for various forms of selection that could undermine a causal interpretation of heterogeneous treatment effects of SSRI introduction on drinking. Estimates from the model incorporating dynamics and unobserved heterogeneity are generally consistent with findings from basic regressions: SSRI usage causes a reduction in alcohol. Moreover, estimates show that addiction may significantly hinder substitution away from alcohol when better technologies (i.e., SSRIs) arrive.

To quantify these substitution effects, we use the estimated system of dynamic equations to perform two sets of counterfactual policy simulations. First, we impose antidepressants on the entire sample relative to our baseline simulation. Heavy drinking declines by 3.4 percentage points, which is primarily driven by men. Moreover, while we show that the reduction in heavy drinking is largest in those simulated to be moderately depressed, we find no change in heavy alcohol consumption, in any period, for those simulated to be in the highest baseline tercile of depression. Our second set of simulations leverages the dynamics more explicitly to explore addiction, which may explain the lack of substitution for those simulated to be the most depressed. To do so, we set lagged alcohol consumption to zero in the contemporaneous alcohol demand equation, regardless

[9] An alternative explanation of our results is that substitution away from alcohol could reflect doctors' recommendations to avoid combining alcohol and SSRIs. Yet, there is little evidence that this contraindication was widely known when SSRI's were first introduced (Weathermon & Crabb, 1999). Furthermore, the FDA highlights of prescribing information for Prozac, the first SSRI to be approved and by far the market leader, did not list alcohol under the contraindications nor under warnings or precautions (http://pi.lilly.com/us/prozac.pdf), so it is unlikely that doctor recommendations drive the substitution patterns we identify. It is also worth noting that use of both SSRIs and alcohol is widespread, and, for depressed individuals with a strong preference for alcohol, SSRIs may have *increased* alcohol consumption as their interaction is significantly less risky than with previous generation antidepressants.

[10] The empirical framework is similar to the dynamic seemingly unrelated regression (SUR) model in Darden et al. (2018), who use FHS data to study the effect of cigarette smoking on expected longevity.

of simulated behavior in the previous period. Overall, regardless of gender or mental health, heavy alcohol consumption drops enormously. Antidepressant usage (which is chosen endogenously in this simulation) increases by 5.5 percentage points by the final exam of FHS, and the magnitude of this substitution is increasing in depression severity. Indeed, in the absence of dependence on past alcohol consumption, those simulated to be in the most severe depression category increase SSRI usage the most. Predicated on the idea that persistent alcohol use could reflect addiction, we interpret these results to suggest that alcohol addiction may significantly hinder substitution away from alcohol when better technologies (i.e., SSRIs) arrive. Finally, we demonstrate that the simulated reduction in heavy drinking is equivalent to a roughly 10% increase in alcohol prices.

Our study adds to a vast literature on self-medication that documents the phenomenon and emphasizes its harms.[11] We contribute to this literature in two key ways. First, we provide arguably causal estimates that imply that the introduction of a new medical technology can reduce use of a potentially harmful substance, which provides novel evidence of self-medicating behavior. Second, our theoretical model suggests a new look at policy surrounding self-medication. Existing literature suggests policies be implemented to curb self-medication, such as making substances used to self-medicate more difficult to obtain. These policies follow from the idea that self-medication is unambiguously harmful rather than a rational decision with costs and benefits. In contrast, the theory of rational self-medication that we present, coupled with our empirical evidence, suggests such policies may be ill-conceived, especially if rational individuals respond to them by turning to more harmful ways to self-medicate. This possibility echoes recent medical literature assessing the possibility that the 2010 reformulation of OxyContin may have increased heroin usage(Dart et al. , 2015). Moreover, our results are consistent with Powell et al. (2018), who show that medical marijuana laws, and in particular the number of marijuana dispensaries, is associated with fewer opioid overdoses. The underlying idea is that rational individuals substitute towards safer options when they emerge.[12]

We also contribute to a literature in health economics that moves beyond assessing the direct effects of medical innovation (e.g., lower mortality and better health) to incorporate a more complete set of indirect effects. This type of work is a crucial complement to findings from clinical trials, which measure treatment effects under controlled conditions, but are ill-suited to analyze additional relevant factors, such as changes in other health behaviors and impacts on longer-run lifecycle outcomes, (e.g. employment), all of which contribute to the full social impact of medical innovation. For example, Papageorge (2016) shows that an important benefit of new HIV treat-

[11] A Google Scholar search for research with "self-medication" in the title yields 3,760 results.

[12] Relatedly, Dinardo (2001) and Crost (2012) use minimum drinking age regulations to show clear substitution patterns between alcohol and marijuana.

ments emerging in the mid-1990s was to raise productivity and increase labor supply. Conversely, Kaestner et al. (2014) show evidence of technological substitution away from diet and exercise with the introduction Statin pharmaceuticals introduced to combat cholesterol. In either case, failing to account for these indirect, behavioral effects would lead to a biased evaluation of the innovation's social value. In our case, to the extent that alcohol consumption is a form of self-medication that harms health, the net benefit of SSRIs on long-term mental health has likely been understated because randomized trials do not account for long-term shifts in alcohol consumption.

More broadly, our paper contributes to our understanding of addiction. In the seminal paper on rational addiction, Becker & Murphy (1988) posit that under addiction, a person has a low level of utility while addicted but a high marginal utility of usage of addictive substances, which incentivizes continued use. While the model explains why forward-looking and addicted individuals continue to use an addictive substance, it is silent on why they would ever become addicted in the first place. Our paper suggests one possible reason. Initial usage of an addictive substance need not be an error in judgment or due to lack of perfect foresight or a large exogenous shock. An individual in pain may assess the probability of future addiction and rationally medicate her pain with available technology, fully aware that doing so can lead to a Becker-style addictive spiral with some probability. Moreover, providing evidence of rational self-medication has implications for understanding the dramatic increase in mortality rates of white non-Hispanic men since 1998, the so-called "Deaths of Despair" documented in Case & Deaton (2015). However, whereas "despair" technically suggests a lack of hope, self-medication suggests the opposite: heavy alcohol use or addiction may reflect an earlier, rational and hopeful attempt to medicate away pain.[13] If so, the appropriate policy response is to stop punishing people who use risky substances to self-medicate and instead work to develop treatments that are less addictive so that people can rationally substitute away from harmful self-medicating behavior.

2 Background on Depression and Self-Medication

Depression is a chronic mental health condition that, while highly treatable, is the leading cause of disability globally[14]. Depression produces symptoms that include feelings of sadness, pessimism, guilt, and anxiety, while also causing decreased energy, loss of interest in daily activities, and indecisiveness. Clinical diagnosis of Major Depressive Disorder (MDD) includes a set of daily symp-

[13] According to the online etymology dictionary, "despair" comes from the French-Anglo *despeir*, originally the French *despoir*, referring to "hopelessness" or a "total loss of hope." See https://www.etymonline.com/word/despair.

[14] http://www.who.int/en/newsroom/fact-sheets/detail/depression

toms plus some functional impairment with respect to family and peer relationships, school/work performance, and stress and anxiety levels (O'Connor et al., 2009).[15]

Depression is an ideal context to study self-medication through alcohol for several reasons. One, it is prevalent. In the United States, in any given two-week period between 2013 and 2016, 8.1% of Americans suffered from depression, ranging from 5.5% for men to 10.4% for women, and there exists a strong gradient between depression and income: 19.8% of women earning less than 100% of the Federal poverty line (FPL) exhibit depressive symptoms compared to only 4.8% of women at or above 400% of the FPL (Brody et al., 2018). Two, while alcohol is not recommended for the treatment of depression, it is well-understood to be a highly effective way to treat the immediate symptoms of depression, which makes it an attractive option for people who lack alternatives (Khantzian, 1990). Three, depression affects many facets of life, including human capital accumulation, productivity, family structure, risky behaviors, and employment, along with other physical health outcomes, such as cancer, cardiovascular disease, and diabetes. Therefore, it is little surprise that individuals would engage in potentially costly attempts to alleviate their immediate symptoms. Four, there is massive stigma surrounding mental health treatment, which might make self-medication via a socially-acceptable behavior, such as drinking, an attractive option.[16] Finally, and key to our empirical work, there are large changes in treatment options over time, in particular the emergence of SSRIs, which replaced earlier drugs that, while effective, had massively adverse side effects that precluded widespread use.

Unsurprisingly, depression is associated with a wide variety of mental and physical ailments, including sleep problems, irritability, persistent physical pain, and risk of suicide (U.S. HHS, 2015). Beck et al. (2011) show that depression is associated with significantly lower levels of fundamental economic building blocks such as workforce productivity, which they measure with the Work Productivity and Activity Impairment Questionnaire, and Berndt et al. (1998) demonstrate that depressed workers have lower levels of perceived at-work productivity and performance. Furthermore, Kessler (2012) shows that depression is associated with low educational attainment, teen pregnancy, martial disruption, unemployment, functional status, early mortality, and suicide. Un-

[15] In the middle 20th century, anxiety was the leading mental illness in the United States. Horwitz (2010) describes how, through a series of reclassifications, as well as the introduction of SSRIs, anxiety has given way to a focus and prevalence of depression.

[16] Another issue, which we do not explore given a relatively homogeneous sample, is that prevalence of depression is heterogeneous across socio-economic groups. Depression is about four times more likely for poor versus non-poor individuals. For those below 100% of the Federal Poverty Line (FPL), the rate was 15.8% between 2013 and 2016, while the rate was only 3.5% for those at or above 400% of the FPL (Brody et al., 2018). This is especially concerning in the context of self-medication if low-income individuals have less access to medical care, safer medications, or treatment options, such as therapy. Moreover, low-income individuals may face other challenges that encourage use of addictive substances, compounding the risks of self-medication.

surprisingly, there is a strong correlation between depression and alcoholism.[17] Indeed, Figures 1a. and 1b. present National Health and Nutrition Examination Survey (NHANES) data on heavy alcohol consumption for men and women by the tercile of the Patient Health Questionnaire (PHQ-9) depression score between 2007 and 2013. For both men and women, more severely depressed individuals are persistently and significantly more likely to engage in heavy alcohol consumption, which is defined here as more than three drinks per day on days in which the respondent drank alcohol.

Depressed individuals have a clear incentive to manage, maintain, and improve mental health. Antidepressant pharmaceuticals have existed since the initial Monoamine Oxidase Inhibitors (MAOI) developed in the 1950s. Most antidepressants function by preventing or slowing the re-uptake of neurotransmitters (such as Serotonin) in the brain, without which depression is more likely. MAOI antidepressants were effective at relieving symptoms of depression, but these, along with Tricyclic antidepressants (TCA) developed in the 1960s, prevent reuptake of many types of neurotransmitters, not only those that regulate mood, and the associated side effects of MAOIs and TCAs include risk of stroke, cardiovascular ailments, and sexual dysfunction, among others. Reflecting these side effects, which prevented certain groups from using antidepressants (e.g., the elderly), as well as public stigma associated with antidepressants, only 2-3% of Americans used an antidepressant through the middle 1980s.[18]

A major advancement in the management of depressive symptoms came with the 1988 FDA approval of Selective Serotonin Reuptake Inhibitors (SSRIs), which, as the name suggests, effectively inhibit the re-uptake of serotonin selectively, making more serotonin available in the brain without affecting the levels of other neurotransmitters. SSRIs significantly altered the perception of antidepressants, reducing stigma, and expanding the set of individuals for whom an antidepressant is considered safe.[19] As a result, rates of antidepressants have increased dramatically since 1988 — up to 12.7% of Americans were prescribed an antidepressant between 2011 and 2014, and of those taking an antidepressant, 25.3% have been taking an antidepressant for more than 10 years (Brody et al., 2018). Researchers now use SSRI prescriptions to *gauge* the rates of depression, mental health, and happiness. For example, Blanchflower & Oswald (2016) study the well-known u-shaped well-being curve with respect to age and show a similar inverse pattern between antidepressants and age.

[17] For example, see Bolton et al. (2009), who use nationally representative survey data from the National Epidemiologic Survey on Alcohol and Related Conditions to document cross-sectional correlations between alcohol and drug use and a variety of mental health conditions.

[18] See Hillhouse & Porter (2015) for an excellent overview of the history of antidepressants.

[19] Despite a significant literature finding positive correlations between SSRIs and teen suicide, Ludwig et al. (2009) provides evidence that the relationship is unlikely to be causal, showing that SSRIs reduce suicides across 25 countries after controlling for the selection of depressed individuals into antidepressant use.

A significant body of work in psychology, medicine, and public health studies the management of depressive symptoms outside of formal prescription drugs, a hypothesis known as self-medication. Khantzian (1985) introduced the idea that the kind of substance used to self-medicate is not random but depends on the type of illness, and that those in states of pain will experiment with different types of substances, some of which may lead to addiction. While the application of Khantzian (1985) was on self-medication with hard drugs (heroin and cocaine), Khantzian (1990) extended the notion of self-medication to the consumption of alcohol, which he described as "a means to achieve and maintain self-regulation." This presentation of self-medication connects "intense affects, such as rage, shame, loneliness, and depression" with the "use of drugs and alcohol to cope with these emotions."

In the absence of safe medication for depression (historically), significant cross-sectional survey evidence suggests that depressed individuals consume alcohol to cope with the symptoms of depression. For example, Crum et al. (2013) show that mental illness is a significant rationale for alcohol consumption and that coping with depressive symptoms with alcohol is associated with the development of alcohol use disorders. Indeed, the consumption of alcohol induces short-term anxiolysis, which produces feelings of relaxation. Deykin et al. (1987) were the first to demonstrate that major depressive disorder typically predates alcohol use disorders in adolescents, providing some evidence on the direction of causality for the robust and pervasive correlation between heavy alcohol consumption and depression.

To summarize, major depressive disorder is the most common mood disorder in the United States, affecting over 16.2 million adults in 2016. SSRIs significantly expanded the choice set with respect to the management of depression, which is frequently medicated outside of the medical system with potentially harmful and addictive substances. These endogenous investments into the mental health production function may have important implications for a variety of outcomes, including labor market productivity and long-term health.

3 Theory

Before proceeding to our empirical analysis, we provide a model of self-medication. The model formalizes the idea that underlying self-medication is a complementarity: the marginal benefit of alcohol use is larger when people are depressed. This generates the hypothesis that the introduction of a medication that reduces depression should lead to a reduction in alcohol use.

S_t denotes symptoms of mental health and M_t denotes underlying mental health stock. It is useful to distinguish between the two in the context of self-medication to allow for a substance such

as alcohol to both reduce immediate symptoms, but also cause future mental health to deteriorate. The production of symptoms is a function of mental health, alcohol A_t and anti-depressants D_t:

$$S_t = f_s(M_t, A_t, D_t) \tag{1}$$

where symptoms are more likely to occur when M_t is lower. Alcohol can improve symptoms, which is the "self-medication" effect, and antidepressants can also improve symptoms. Mental health evolves according to the following production function

$$M_{t+1} = f_m(M_t, A_t, D_t) \tag{2}$$

where the argument M_t captures persistence in mental health stock, A_t captures how alcohol usage can have negative impacts on future mental health and D_t captures how antidepressants may change long-run mental health.

Agents solve a two-period problem, where periods are denoted t and $t+1$.[20] An agent enters period t with state variable M_t, which is the stock of mental health and where lower values of M_t imply worse mental health. Agents choose whether or not to take an antidepressant, denoted $D_t \in \{0, 1\}$ and how much alcohol to drink $A_t \in \mathbf{R}^+$. For ease of exposition, we assume that the agent chooses non-zero alcohol consumption.

Agents have preferences over alcohol consumption A and antidepressant consumption D, where the latter includes the price of antidepressants along with side effects, stigma and other non-pecuniary costs of SSRI use. They do not have preferences over mental health *per se*, but instead over symptoms of poor mental health S. Agents choose A and D to solve:

$$\max_{A_t, D_t} \Big(u(S_t, A_t, D_t) + \beta v(S_{t+1}) \Big) \tag{3}$$

where we assume that S and D enter negatively and A enters positively into both u and v. Period $t+1$ is effectively a "terminal" period in which no decisions are made and $v(S_{t+1})$ is thus a continuation payoff affected by period-t choices which thus provides dynamic incentives to improve mental health.

To characterize self-medicating behavior, we use the model to make the following three points. First, we show conditions under which $D^\star = 1$. Second, we characterize optimal alcohol usage. Finally, we discuss conditions under which lowering the costs associated with antidepressant usage — through the approval of SSRIs — would lead to decreases in alcohol usage. The third point is

[20] Our model is similar to Becker (2007), who distills the Grossman (1972) model into a two-period framework.

consistent with a reduction in self-medication through alcohol when medication becomes a more attractive option.

To show optimal antidepressant usage, denote optimal alcohol consumption A^* and A^{**}, when using antidepressants and not using antidepressants, respectively. Agents use antidepressants when the benefits of doing so exceed the costs:

$$\begin{array}{rcl} u(S(D_t=1),A^*,D_t=1) & + & \beta v(S_{t+1}(M_{t+1}(D_t=1))) \geq \\ u(S(D_t=0),A^{**},D_t=0) & + & \beta v(S_{t+1}(M_{t+1}(D_t=0))) \end{array} \tag{4}$$

To fix ideas, suppose we make the simplifying assumption on period-t utility that the costs of medication usage are additively separable from other utility components, e.g., $u(S_t, A_t, D_t) = \tilde{u}(S_t, A_t) - \phi(D_t)$ where $\phi(D_t = 1) = \phi$ and $\phi(D_t = 0) = 0$.[21] The agent uses antidepressants if and only if

$$\begin{array}{rcl} \tilde{u}(S_t(D_t=1),A_t^*)+\phi & + & \beta v(S_{t+1}(M_{t+1}(D_t=1))) \geq \\ \tilde{u}(S_t(D_t=0),A_t^{**}) & + & \beta v(S_{t+1}(M_{t+1}(D_t=0))) \iff \\ \tilde{u}(S_t(D_t=1),A_t^*)-\tilde{u}(S(D=0),A^{**}) & + & \beta[v(S_{t+1}(M_{t+1}(D_t=1)))-v(S_{t+1}(M_{t+1}(D=0)))] \geq \phi \end{array} \tag{5}$$

The last line implies that the benefits must outweigh the costs in order for antidepressant usage to occur, where the benefits include current period utility of fewer symptoms along with discounted $t+1$ reductions in symptoms due to increased mental health stock. For a given level of antidepressant effectiveness, antidepressant usage increases if the flow utility costs decline, e.g., through side effects, stigma or price reductions. Moreover, as long as $\phi > 0$, antidepressant usage only occurs if there are benefits in the form of improved symptoms, either currently or in the future.

Next, we characterize optimal alcohol consumption, in which the relevant first order condition is:

$$\frac{\delta u}{\delta S_t}\frac{\delta S_t}{\delta A_t} + \frac{\delta u}{\delta A_t} + \frac{\delta v}{\delta S_{t+1}}\frac{\delta S_{t+1}}{\delta M_{t+1}}\frac{\delta M_{t+1}}{\delta A_t} = 0 \tag{6}$$

or

$$\frac{\delta u}{\delta A_t} + \frac{\delta u}{\delta S_t}\frac{\delta S_t}{\delta A_t} = -\beta\frac{\delta v}{\delta S_{t+1}}\frac{\delta S_{t+1}}{\delta M_{t+1}}\frac{\delta M_{t+1}}{\delta A_t} \tag{7}$$

The left hand side captures the marginal benefits of alcohol use, including both the enjoyment of alcohol along with reduction in symptoms from self-medicating. The right hand side captures

[21] Additive separability implies that the marginal utility of alcohol is unaffected by SSRI usage. While this assumption is unrealistic, it simplifies the exposition for optimal SSRI usage, and it does not affect our comparative dynamics analysis presented below.

marginal costs: higher A reduces M' and lower M' reduces continuation payoffs captured by v. Optimal alcohol usage occurs when the marginal benefit of an additional unit of A is equal to the marginal cost.

Finally, we use our simple model to derive conditions under which antidepressant usage should lead to decreases in alcohol usage. It is convenient to define a function for the marginal utility of symptoms for both periods as follows:

$$\frac{\delta v}{\delta S_t} = \frac{\delta u}{\delta S_t} \equiv \alpha(S_t) \tag{8}$$

For example, if $\alpha(S) = \alpha S$ and $\alpha > 0$, then utility is a concave function with increasingly negative marginal utility of S. Having done this, the first-order condition above can be rewritten as:

$$\frac{\delta u}{\delta A_t} = -\alpha(S_t)\left[\frac{\delta S_t}{\delta A_t} + \beta \frac{\delta S_t}{\delta M_{t+1}} \frac{\delta M_{t+1}}{\delta A_t}\right] \tag{9}$$

If alcohol usage decreases with SSRIs, it must be the case that SSRIs lead to a decline in the left-hand-side of the last equation or an increase in the right-hand-side. We do not allow the enjoyment of alcohol to be a function of symptoms, so the left hand side does not change. Thus, for SSRIs to lower alcohol usage, it must be the case that the right hand side rises or that -1 times the right hand side falls. Thus, to understand reduced self-medication in the form of drinking, we examine why the following expression should decline when symptoms decline:

$$\alpha(S_t)\left[\frac{\delta S_t}{\delta A_t} + \beta \frac{\delta S_{t+1}}{\delta M_{t+1}} \frac{\delta M_{t+1}}{\delta A_t}\right] \tag{10}$$

There are four possibilities:

1. $\alpha(S_t)$ is lower when $D_t = 1$. Given that utility is a declining function of S_t, this suggests that costs of S_t rise with S_t. The implication is that medication leads to a decline in symptoms. This reduces the marginal cost of symptoms, which means that the marginal benefit of technology that reduces symptoms is lower.

2. A second possibility is that $\frac{\delta S_t}{\delta A_t}$ is lower when $D_t = 1$. This could occur if alcohol is less productive at reducing symptoms at lower symptom levels.

3. The third possibility is that $\frac{\delta S_{t+1}}{\delta M_{t+1}}$ is smaller when $D_t = 1$. This means that improvements to mental health reduce symptoms more so when mental health is better.

4. Finally $\frac{\delta M_{t+1}}{\delta A_t}$ is lower when $D_t = 1$ which suggests that alcohol reduces future mental health more so if mental health is better.

In each of these instances, there is a complementarity in the sense that use of medication reducing depression makes use of alcohol less attractive. The model thus generates a testable hypothesis. In the absence of self-medication, there should be no causal link between alcohol consumption and the introduction of SSRIs. In contrast, self-medicating behavior implies that the introduction of SSRIs should reduce alcohol consumption. The model also provides guidance on policy. Restricting access to substances used to self-medicate without improving underlying health problems or resolving underlying motivations to self-medicate could backfire, inducing people to turn to other, more dangerous options. Alternatively, policies that promote safer medications can obviate the need to self-medicate. Finally, the model formalizes our notion of self-medication as arising from rational responses to a complementarity between alcohol consumption utility and depression. Yet, the exact source of the complementarity is difficult to pinpoint with the data we have. We return to this point in the conclusion, where we discuss avenues for future research. We now turn to our empirical investigation of self-medication.

4 Empirical Evidence

4.1 Data, Sample Construction and Preliminary Analysis

To study self-medication empirically, we turn to the Offspring Cohort of the Framingham Heart Study (FHS). The Offspring Cohort data are ideal for our purposes as they include longitudinal information on alcohol, antidepressant medication, and mental health over nine detailed health exams over 40 years. Begun in 1971, the Offspring Cohort includes roughly 5,000 offspring of the FHS Original Cohort, which began in 1948 in Framingham Massachusetts, and their spouses. Both cohorts of individuals have received detailed health examinations at 2-4 year intervals into the 21st century, and both cohorts have made significant contributions to the understanding of cardiovascular disease.[22]

Participants range from 13 to 62 years of age at the first exam, which reflects the wide age variation in the Original Cohort. The Original Cohort restricted its sampling to white residents of Framingham Massachusetts, and, while no restriction was placed on the ethnicity or residency of the spouses of the offspring, data are not available on these characteristics. As the FHS was not meant to be representative of any larger population, we restrict our final estimation sample to 2,497 individuals for whom we have consistent exam participation and information.[23] To enter our

[22]See Mahmood et al. (2014) for a detailed history of the Study. See Darden et al. (2018) and Darden (2017) for economic studies of the Original and Offspring Cohorts, respectively.

[23]Kaestner et al. (2014) and Darden (2017) construct very similar samples from FHS Offspring Data.

sample, an individual must have completed exams one through three and must not have skipped exams in the subsequent periods. Following the third exam, individuals may leave the sample through either death or attrition. Because of an eight year gap between exams one and two, and because of data limitations discussed below, we restrict our analysis to exams two through nine. All FHS Offspring participants completed exam two between 1979 and 1983.

Table 1 presents summary statistics of the Offspring Cohort at our initial exam (exam two) by gender and by whether an individual is ever, over the subsequent seven exams, observed to be on any type of antidepressant. Of the 1,241 men in our sample, 12.17% are observed at some point to be taking antidepressants; for women, that statistic is 24.52%. The FHS asks respondents the number of 12oz beers, 5oz glasses of wine, and 1.5oz liquor drinks they typically consume per week. We aggregate these to a drinks per week measure, and we follow the National Institute on Alcohol Abuse and Alcoholism guidelines for light and heavy alcohol consumption based on gender: light drinking is defined as up to seven drinks per week for women and 14 drinks per week for men; heavy drinking is any number above the gender-specific thresholds.[24] At the second exam, men drink more heavily than women (despite the higher threshold for heavy drinking), and rates of heavy drinking are higher for those ever-observed to take an antidepressant (although these differences are not statistically significant). Generally, there are not statistical differences between ever and never antidepressant users, although a notable exception is cancer and mortality incidence for women, which are both statistically higher among the never users, despite the fact that women taking antidepressants are more likely to smoke.

At exam three, Offspring Cohort participants took the Center for Epidemiological Services - Depression (CES-D) test for depression, which aggregates 20 clinically verified depression questions (each on 0 to 3 Likert Scale) into a depression summary score (Radoff, 1977).[25] We break the continuous depression score at exam three into terciles, and we present the fraction of individuals in each exam three tercile by gender and whether they are ever observed to take an antidepressant in the last three rows of Table 1. Not surprisingly, the fraction of both men and women in higher CES-D terciles are higher for those who go on to take an antidepressant, but we emphasize the sizable fraction of those in the lowest tercile of depression in exam three who eventually use antidepressants as foreshadowing of the heterogeneity in results presented below.[26] Importantly, antidepressants are prescribed for a wide variety of conditions other than depression, including bipolar disorder, bulimia, fibromyalgia, insomnia, PTSD, and social anxiety disorder (CMS, 2013).

[24] See NIAAA, which was accessed for this paper on November 7th, 2018.

[25] The clinically verified threshold for depression is any score at or above 16.

[26] Wulsin et al. (2005) use FHS Offspring Cohort data to relate the exam three CES-D score to future health outcomes. They find that, relative the lowest tercile, CES-D score is statistically related to all-cause mortality but not coronary heart disease.

Table 2 shows means and proportions of key variables over the eight exams. Each FHS exam was administered within a three to four year window, and, while we do not have information on the date that an individual took an exam, Table 2 displays the year ranges in which all participants completed each exam. Unfortunately, we do not observe antidepressant medication usage at exam two, however, the absence of this information likely stems from the observed trends in their use: at exam three, only 1.0% of men and 2.1% of women used antidepressants. Importantly, exam three was completed prior to 1988, when the FDA approved SSRIs, after which antidepressant medication usage grows considerably within our sample over time for both men and women. Light and heavy alcohol use decline over our sample period and cigarette smoking plummets. Between exams two and nine, we lose roughly 48% and 38% of men and women, respectively, to sample attrition or death; thus, trends in behaviors in Table 2 reflect both changing behavior and the changing composition of the sample, which we emphasize below in our dynamic system of equations model.

4.2 Evidence of Rational Self-Medication

To test the rational self-medication hypothesis that consumption of risky goods should decline following an improvement in the choice set of treatment options, we begin by regressing binary indicators for none, light, and heavy drinking on FHS exam binary variables, where we allow trends in consumption to vary by exam three CES-Depression terciles. The idea is to exploit the plausibly exogenous introduction of SSRIs - the improvement in technology - and look for differential trends in alcohol consumption around their introduction by groups that are more likely to use SSRIs. Formally, we estimate:

$$\begin{aligned} y_{it} = \mu_i + x_{it}^{'}\beta + \sum_{j=3}^{9} 1\{t=j\}\theta_j + \sum_{j=3}^{9} 1\{t=j\}1\{\text{Moderately Depressed}_{it=3}\}\psi_j + \\ + \sum_{j=3}^{9} 1\{t=j\}1\{\text{Heavily Depressed}_{it=3}\}\nu_j + \epsilon_{it}, \end{aligned} \tag{11}$$

where y_{it} is alcohol variable y for person i in year t, μ_i represents an individual specific effect, x_{it} are time-varying individual characteristics, θ_t are parameters on exam binary variables, and ϵ_{it} is an i.i.d. error component. Parameters ψ and ν allow the trend in consumption to differ by those with a CES-D score between 5 and 10, which we call "moderately depressed," and those with a CES-D score more of than 10, which we call "heavily depressed", respectively. Our focus on the exam three CES-D score is because exam three took place between 1983 and 1987, just before the introduction of SSRIs in 1988. Thus, the exam three score is a baseline metric of depression, prior

to the improved technology. We argue that deviations from the trend in alcohol consumption of those in the low depression group after exam three would provide suggestive evidence that SSRIs generated changes in alcohol consumption for those in worse mental health. We estimate Equation 11 on data from exams two through nine, where exam four represents the first exam to be taken after the introduction of SSRIs.

The top panel of Table 3 presents estimates of the ψ and ν parameters from Equation 11 after exam three for our four alcohol behavioral outcomes, where we estimate separate linear probability models for none, light, and heavy drinking, and a continuous equation for the log of drinks per week conditional on drinking.[27] We find suggestive evidence that alcohol consumption decreases for those with heavy depression. For example, by exam nine, heavy alcohol consumption declines by 4.5 percentage points, roughly 26.3%, for those with heavy depression relative to those in good mental health. The identification assumption is that trends in alcohol consumption by depression would evolve in a parallel fashion in the absence of the introduction of SSRIs. While this assumption is untestable, we present the p-value on the F-test whose null hypothesis is that ψ_3 and ν_3, the deviations in trend prior to the introduction of SSRIs, are jointly zero. For each dependent variable, we fail to reject differential pre-trends.

Results in the top panel of Table 3 are consistent with our rational self-medication theory, but the estimates are fairly imprecise. Thus, we model alcohol consumption directly as a function of antidepressent usage - recognizing that nearly all of the observed antidepressant medication usage in our data occurs after the introduction of SSRIs. Specifically, we estimate:

$$y_{it} = \mu_i + x_{it}^{'}\beta + \delta d_{it} + \sum_{j=4}^{9} 1\{t = j\}\theta_j + \epsilon_{it}, \tag{12}$$

where y_{it} is alcohol variable y for person i in year t, μ_i represents an individual specific effect, x_{it} are time-varying individual characteristics, θ_t are parameters on exam binary variables, and ϵ_{it} is an i.i.d. error component. Our variable of interest is d_{it}, which equals one if person i in exam t is taking an antidepressant. Because we do not observe antidepressant use in exam 2, we estimate Equation 12 on data from exams 3 through 9; thus, the θ parameters are relative to exam 3. Results are conditional on age, education, and other health metrics, including blood pressure, obesity, cardiovascular disease, cancer, and exam fixed effects.[28] Standard errors are clustered at

[27] Multinomial and ordered logit estimators yield similar results as those from estimation of Equation 12 without the individual fixed effect, μ_i. Because of the incidental parameters problem associated with logit estimators with fixed effects, we focus on linear probability models in this section.

[28] Throughout our paper, our results are not sensitive to the inclusion of these endogenous health outcomes. Especially in the dynamic model presented below, we include these outcomes as controls as there is evidence that own health shocks may alter health behaviors. (Arcidiacono et al. , 2007)

the individual level.

The bottom panel of Table 3 offers more concrete evidence of substitutability between antidepressants and alcohol. For each alcohol measure, the top two rows present separate estimates of δ with and without individual fixed effects (i.e., $\mu_i = \mu \ \forall i$), respectively. Results without individual fixed effects are presented in row one, where antidepressants are associated with a 9.9 percentage point increase in alcohol abstinence, a result that is mainly driven by a reduction in light drinking. Conditional on positive alcohol consumption, the intensive margin of drinks per week decreases by 16.3% when taking an antidepressant. Adding individual fixed effects (row 2) attenuates the results, however the results remain statistically significant and economically meaningful: alcohol abstinence increases by 3.9 percentage points and the intensive margin measure of drinks per week drops by 9.5%.

The identification argument in Equation 12 such that δ may take a causal interpretation is that there is no *time-varying* unobserved heterogeneity that affects both the decision to take antidepressants and behavior. While we cannot directly test this assumption, we can explore differential trends based on time invariant characteristics. For example, conditional on contemporaneous antidepressant usage, differential trends between those ever and never using antidepressants would be suggestive of time-varying unobserved heterogeneity. Equation 13 demonstrates this model, where the τ parameters capture exam-specific deviations from trend.

$$y_{it} = \mu_i + x_{it}^{'}\beta + \delta d_{it} + \sum_{j=4}^{9} 1\{t = j\}\theta_j + \sum_{j=4}^{9} 1\{t = j\}1\{\text{Ever Takes Antidepressants}_i\}\tau_j + \epsilon_{it}, \quad (13)$$

Row 3 of Table 3 presents estimates of δ from Equations 13. Relative to row 2, where trends are restricted to be the same by whether someone ever takes antidepressants, estimates of δ are attenuated; however, the drinks per week along the intensive margin declines significantly by 5.9%. Furthermore, the p-values for the respective F-tests that all of the trend deviation parameters (i.e., the τ parameters in Equation 13) are zero all suggest that we fail to reject parallel trends *conditional on antidepressant usage*. Again, evidence of differential trends would be suggestive of important time-varying unobserved heterogeneity, but our estimates of δ are robust to allowing for differential trends. The bottom row of Table 3 presents estimates of δ when we allow trends to vary by exam three depression status, as in Equation 11, and the results are similar to those in row 3.

To further demonstrate that alcohol trends prior to antidepressant usage did not differ systematically from those not taking antidepressants, we estimate a series of event studies which exploit

within-individual variation on the timing of antidepressant usage. Specifically, we estimate

$$\begin{aligned} y_{it} = \mu_i + x_{it}'\beta + \sum_{j=4}^{9} 1\{t=j\}\theta_j + \sum_{j=-3}^{-1} \tau_{j+4} 1\{\text{First Takes Antidepressants in t-j}\} + \\ + \tau_4 1\{\text{First Takes Antidepressants in t}\} + \\ + \tau_5 1\{\text{Took Antidepressants Prior to t}\} + \epsilon_{it}. \end{aligned} \tag{14}$$

Conditioning on individual and time fixed effects and observable characteristics x, Equation 14 captures the timing of antidepressant usage. Coefficients τ_1, τ_2, and τ_3 represent any deviation from trend for those individuals who will take an antidepressant in four (or more), three, and two periods, respectively. We omit a parameter for the period prior to the first antidepressant usage (i.e., event time 0), and we estimate parameters for the first period of antidepressant usage (τ_4) and an aggregate of all periods after the first period of antidepressant usage (τ_5).[29] Figure 2 graphically presents our estimates of the τ parameters. As shown, none of the parameters representing differential pre-trends (i.e., τ_1 through τ_3) are significantly different from zero for any of our dependent variables. Furthermore, we fail to reject the null hypothesis that all of the pre-period parameters are zero, as the p-values on the associated F-tests are all large. Consistent with our results in Table 3, we find significant evidence of substitution away from alcohol. Alcohol abstinence increases significantly following initiation of antidepressants, and drinks per week decline by 11.2% in the first exam taking antidepressants. For example, Figure 2d shows estimates and 95% confidence intervals of the τ parameters in a model of the log of drinks per week conditional on nonzero drinking. In the first exam in which a person is reported to be taking an antidepressant, drinks per week fall significantly.

To summarize, we provide evidence that the introduction of SSRIs led individuals to substitute from alcohol to SSRIs. Results from our baseline estimator in Equation 12 reveal a 3.9 percentage point increase in alcohol abstinence and a 9.5 percent reduction in drinks per week conditional on drinking. We find no evidence that these effects are biased due to differential trends.

5 Evidence on Heterogeneity

The theoretical model, in particular, the complementarity between the marginal utility of alcohol and depression, predicts that the introduction of SSRIs should lead to lower alcohol consumption. Consistent with the model, empirical results from the previous section establish that the intro-

[29] Most individuals in our sample who ever use antidepressants initiate usage towards the end of the nine exams; thus, we are only able to estimate parameters for the first period of use and an aggregate of the following periods.

duction of SSRIs reduced alcohol use. However, addiction to alcohol may hamper this type of substitution, which we aim to explore in this section.[30]

Exogenous variation in addiction coupled with the exogenous arrival of SSRIs would allow us to test for this type of interaction directly. However, lacking a measure of addiction (let alone exogenous variation in accumulated addiction stock), we rely on the idea that persistent use of alcohol can reflect addiction. In particular, we estimate a dynamic system of equations in which we explicitly model not only alcohol, but also cigarettes, antidepressants, along with equations for depression, attrition, and mortality. The dynamic system of equations approach allows current behavior to depend on previous alcohol usage, and it also allows for correlation across equations in both observed and unobserved heterogeneity. The system of equations addresses several problems that could bias results. For example, the composition of our sample is changing over time through mortality and attrition. Especially because (i) the behaviors being modeled may cause mortality or attrition; and (ii) significant antidepressant medication usage is not observed until the end of our sample period, selective exits may significantly bias our results. Finally, estimation of each equation separately does not allow for correlation in unobserved heterogeneity across equations, which could bias our effects of interest if unobserved factors drive both the decision to use alcohol and the probability of either death or attrition. As we will show, results presented in this section are consistent with those from the static model, which provides an important robustness check of the estimates presented in Table 3, i.e, they are not driven by factors such as shifts in the composition of the sample over time.

A benefit of an estimated system of equations incorporating dynamics is that it expands the set of counterfactual policies we can simulate, albeit at the expense of making stronger assumptions. We use the dynamic alcohol demand equation to investigate a role for addiction in explaining substitution patterns, essentially simulating behavior supposing previous use of alcohol is zero. The validity of this exercise requires the assumption that the system of equations incorporating unobserved heterogeneity adequately accounts for selection into previous alcohol use. Our simulation provides speculative evidence that addiction, proxied by persistent use of alcohol, hampers substitution towards SSRIs.

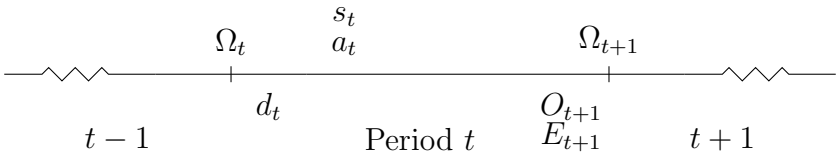
5.1 Model Specification

In the spirit of our two-period theoretical model presented above, and to address the limitations of our static empirical model, we estimate a dynamic system of equations for antidepressants,

[30]Indeed, a large and growing empirical literature recognizes the inherent dynamics in addictive goods (Arcidiacono et al., 2007; Darden, 2017), the failure of which to model will likely lead to an overestimate on the effect of antidepressants on behavior.

alcohol and tobacco consumption, sample attrition, and mortality. The empirical model is an approximation of a more general structural model of behavior and outcomes in which an individual optimally selects a bundle of investments in health, and health, both mental health and mortality, is a function of behavior. In what follows, we briefly outline the timing of our dynamic system of equations.

The following time line presents a representative exam period t of an individual's problem in which we suppress the individual subscript i for ease of notation:



Here, Ω_t captures the period t state vector, which sufficiently summarizes measures of past behavior. Given her state Ω_t, an individual begins period t by choosing whether or not to take an antidepressant, d_t. Conditional on d_t, an individual chooses whether to smoke s_t and the intensity of alcohol consumption $a_t \in \{None, Light, Heavy\}$.[31] Alcohol and cigarette decisions follow the antidepressant decision to allow the marginal utility of alcohol and cigarettes to depend on antidepressant consumption.[32] Following these decisions, at the end of period t, a person may attrit from the sample, E_{t+1} or die, O_{t+1}, but conditional on remaining in the sample, the state variable S updates.

While solution of such a model is beyond the scope of this paper, such a solution would generate demand equations for antidepressants, alcohol, and cigarettes, as well as outcome equations for attrition and mortality. Specifically, solution would theoretically yield the following probabilities for each behavior:

$$p(d_t = d) = d(\Omega_t, X_t, c_3, \mu^d, \epsilon_t^d) \tag{15}$$

$$p(a_t = a) = a(\Omega_t, d_t, X_t, P_t, c_3, \mu^a, \epsilon_t^a) \tag{16}$$

$$p(s_t = s) = s(\Omega_t, d_t, X_t, P_t, c_3, \mu^s, \epsilon_t^s) \tag{17}$$

The demand for antidepressants is a function of past behavior (alcohol, cigarettes, and antidepressants), as well as exogenous characteristics X_t. The final two terms, μ^d and ϵ_t^d, represent a

[31] While smoking is not our main behavior of interest, we model smoking behavior because of the obvious implications of smoking on life-cycle health.

[32] We model alcohol sequentially with antidepressants because an antidepressant requires a prescription and is therefore a less flexible input. Furthermore, this modeling decision allows the marginal utility of alcohol (and tobacco) consumption to depend on contemporaneous antidepressant consumption.

permanent, individual specific component and an i.i.d. error component, respectively.[33] The demand for alcohol and cigarettes are chosen simultaneously as a function of the same arguments, including a price vector P_t, lagged behavior, exogenous characteristics, and antidepressants, which again captures the potential for negative interaction effects between these behaviors and antidepressants. Similar to the antidepressant equation, the final two terms, $\mu^{\cdot}$ and $\epsilon^{\cdot}_t$, represent permanent, individual specific components and i.i.d. error components, respectively.

The structural equation framework above suggests that an outcome equation for mental health should be a function of the state vector Ω_t, which includes lagged mental health, and period t behavior. Unfortunately, we do not consistently observe the CES-D score in the Framingham data.[34] Our solution is to estimate a time invariant measure of depression based on the exam three CES-D terciles presented above. Specifically, we estimate:

$$p(c_3 = c) = c(a_2, s_2, X_3, \mu^c, \epsilon^c_3) \tag{18}$$

where $c \in \{Low, Medium, Heavy\}$. Importantly, the exam three CES-D is measured prior to the introduction of SSRIs in 1988; thus, we interpret c_3 as a baseline measure of depression which is predictive of future mental health. Because our baseline measure of mental health may itself be a function of past alcohol and tobacco consumption, we allow the probability of each depression state to be a function of lagged alcohol and tobacco consumption, a_2 and s_2, respectively. Furthermore, as discussed in more detail below, estimating Equation 18 jointly with the demand/outcome system allows us to jointly estimate the distribution of permanent unobserved heterogeneity, μ.

In addition to Equation 18, we estimate equations for sample attrition and mortality, respectively:

$$p(E_{t+1} = e) = e(\Omega_t, a_t, s_t, d_t, c_3, X_t, \mu^e, \epsilon^e_t) \tag{19}$$

$$p(O_{t+1} = o) = o(\Omega_t, a_t, s_t, d_t, c_3, X_t, \mu^o, \epsilon^o_t). \tag{20}$$

Finally, because we observe individuals between the ages of 17 and 72 at exam two, we observe very different initial histories of alcohol and cigarette consumption. Thus, we estimate initial conditions

[33] Prices of antidepressants are not included because the sample lacks spatial variation - most individuals live in Framingham, and precise location is unobserved. We allow for a time trend in estimation to attempt to absorb the temporal component of prices. Furthermore, our data lack income, so we are unable to construct the usual budget constraint. The price effect in the antidepressant equation is likely second-order, as most respondents have health insurance and the sample represents a very well-educated and homogenous population.

[34] We observe the CES-D measure in exams 3, 6, 7, and 9. While estimation of a dynamic production function for the CES-D score is technically possible, the parameter estimates were highly unstable when estimating jointly with other behavioral/outcome equations.

equations for alcohol consumption and cigarette smoking at exam two:

$$p(a_2 = a) = a(X_2, \mu^{a\prime}, \epsilon^{a\prime}) \tag{21}$$

$$p(s_2 = s) = s(X_2, \mu^{s\prime}, \epsilon^{s\prime}). \tag{22}$$

Included in X_2 is a coarse cohort control for initially entering our sample over the age of 50. Under the assumption that each ϵ term takes an extreme value type 1 distribution, equations 15 through 22 become a system of dynamic logit equations.[35]

The μ terms represent equation specific permanent unobserved heterogeneity, and we allow the μ terms to be correlated across equations, yielding the familiar seemingly unrelated regression framework. Conditional on the assumption that each ϵ term takes an i.i.d. extreme value distribution, we treat the joint distribution of $(\mu^{a'}, \mu^{s'}, \mu^c, \mu^a, \mu^s, \mu^e, \mu^O)$ non-parametrically. Following Heckman & Singer (1984) and Mroz (1999), we estimate a step-function for an assumed number of points of support for each term. Subject to the normalization that the first point of support is zero in all equations, we jointly estimate each point of support and the probability of each type. While μ takes the form of a random effect (i.e., we are estimating the distribution of the permanent component of the error structure), μ is not independent of the *endogenous* right-hand side variables because the latent factor μ helps to determine past realizations of the endogenous behaviors and outcomes.

To estimate the system, we maximize the log-likelihood function with respect to the parameters that dictate initial conditions, exam three depression, behavior, and outcomes. The latent factor approach allows individual characteristics that are unobserved by the researcher to impact all jointly estimated equations (in a non-linear way) and integrates over their distributions when constructing the likelihood function. The weighted-sum of likelihood contributions for each individual i at time t is:

[35] Equations for alcohol and the exam three CES-D tercile are multinomial logit equations.

$$
\begin{aligned}
L_i(\Theta, \mu, \rho) \quad = \quad & \sum_{k=1}^{K} \rho_k \Bigg\{ \prod_{s=0}^{1} p(s_2 = s|\mu_k^{s'})^{1\{s'=s\}} \prod_{a=0}^{2} p(a_2 = a|\mu_k^{a'})^{1\{a'=a\}} \prod_{j=0}^{2} p(c = j|\mu_k^{c})^{1\{c=j\}} \times \\
& \times \prod_{t=3}^{9} \Bigg[\prod_{d=0}^{1} p(d_{it} = d|\mu_k^{d})^{1\{d_{it}=d\}} \prod_{a=0}^{2} p(a_{it} = a|\mu_k^{a})^{1\{a_{it}=a\}} \prod_{s=0}^{1} p(s_{it} = s|\mu_k^{s})^{1\{s_{it}=s\}} \times \\
& \times \prod_{e=0}^{1} p(E_{it+1} = e|\mu_k^{e})^{1\{E_{it+1}=e\}} \prod_{o=0}^{1} p(O_{it+1} = o|\mu_k^{o})^{1\{O_{it+1}=o\}} \Bigg] \Bigg\}
\end{aligned} \tag{23}
$$

where Θ defines the vector of parameters of the model. Here, ρ_k denotes the probability of the k^{th} mass-point, which is estimated jointly with the k^{th} permanent mass point $\mu_{\dot{k}}$ in each equation. After taking the log of each individual's unconditional likelihood contribution, we add the contributions to form the sample log-likelihood function and maximize with respect to Θ.

Estimation of our dynamic system of equations uses both within-individual and across-individual variation in behavior and outcomes (as opposed to results in Table 3, which focus only on within-individual variation), which we argue provides a richer test of the rational self-medication hypothesis, in addition to addressing the limitations of the static reduced-form approach listed above. Because of the presence of both sources of variation, identification of the system comes from three sources. First, as a natural set of exclusion restrictions, prices of cigarettes and alcohol appear only in the demand equations for cigarettes and alcohol.[36] The assumption is that any effect of prices on antidepressant behavior and our endogenous outcomes works through alcohol and cigarette behavior, and in what follows, we show that prices significantly shift these behaviors. Furthermore, following the logic in Arellano & Bond (1991), time-varying exogenous variables such as prices serve as implicit instruments for behavior.

Second, as discussed above, the FDA's 1988 approval of SSRIs dramatically lessened the side-effects of taking an antidepressant and opened antidepressants to new demographic markets (e.g., the elderly). We argue that the full price of antidepressants shifted exogenously between exams three (taken between 1983 and 1987) and four (taken between 1987 and 1991) as a result of this

[36] While we do not observe an individual's location, most of our sample remain in Massachusetts, so the only variation in average prices is temporal. We interact all prices with age to generate cross-sectional variation and to allow the price elasticity and cross-price elasticities of demand for alcohol and cigarettes to vary with age. We use the alcohol specific Consumer Price Index for urban consumers from the Federal Reserve Bank of St. Louis' Federal Reserve Economic Data, which is seasonally adjusted and relative to 1982-1984. https://fred.stlouisfed.org. Accessed on April 2nd, 2018. Price data for cigarettes represent the mean cigarette price in Massachusetts in a given year over all cigarette brands. We merge these data to the median year in which an individual may have taken each exam. See Darden et al. (2018) for further information. We thank Koleman Strumpf for sharing these data.

innovation. While we do not observe antidepressant use in exam two (or exam one), the absences of these questions in FHS surveys is likely due to the national prevalence of antidepressants. Indeed, in exam three, only 1.6% of our sample was taking an antidepressant. We model antidepressant usage as a function of past depression, among other things, to capture observable types of individuals most likely to select into subsequent antidepressant usage, and we argue that the diffusion of antidepressants documented in Table 2 was due to SSRIs.

Finally, by estimating Equations 15- 22 jointly, we allow permanent unobserved heterogeneity to influence both initial and per-period behavior and outcomes. We argue that modeling the distribution of unobserved heterogeneity is important because permanent unobserved characteristics such as genetic endowments may affect both behaviors and outcomes. For example, if some individuals are less likely to consume alcohol, and thus are observed at exam two not drinking alcohol, but are more likely to take an antidepressant later in the sample for permanent unobserved reasons, we allow for this correlation.

5.2 Estimates from the Dynamic Model

Table 4 provides selected estimates from the multinomial logit equation for per-period alcohol consumption relative to the omitted category of not drinking.[37] For example, for light drinking, Table 4 presents the estimated coefficients on selected right-hand-side variables and the associated standard errors for both a model without unobserved heterogeneity (i.e., where we set k=1) and for a model in which we assume four points of support for the joint distribution of μ (subject to the normalization that the first point of support is zero in all equations). While the coefficients are difficult to interpret, the Table demonstrates a negative relationship between antidepressants and both light and heavy drinking. Table 4 also demonstrates the importance of allowing for unobserved heterogeneity. The coefficient on heavy depression at Exam three (i.e. $CES - D \in [11, 51]$) flips from negative to positive and statistically significant with respect to heavy drinking, which suggests that the marginal utility of heavy alcohol consumption is higher for those with depression.

Table 5 presents the estimated points of support for the joint distribution of μ and the associated probabilities of each "type."[38] Our preferred specification includes four points of support for the distribution of μ, and we normalize the first point in each equation to zero. For example, type four individuals are significantly more likely to be highly depressed at exam three, they are significantly more likely to take antidepressants and smoke, but they are significantly less likely to drink, both lightly and heavily. Because parameters in both Tables 4 and 5 are difficult to interpret on their

[37] Tables 7-10 present the entire set of parameter estimates and standard errors for all estimated equations.

[38] Not reported are the estimated points of support in the initial conditions alcohol and cigarette equations. These are presented in Table 10

own, we now turn to simulation exercises to investigate rational self-medication.

5.3 Policy Simulations

To evaluate our model, we simulate both the extent to which our model can recover the time path of each behavior/outcome and the extent to which it can capture transitions between behaviors. To proceed, we replicate the baseline sample, complete with their baseline characteristics, 50 times, which implies a sample of 50*2,497=124,850 simulated observations. Using the estimated distribution of μ, we endow each simulated individual with a complete set of draws of the error structure (as well as independent draws of ϵ). We begin by using the estimated initial conditions and exam three CES-D equations to simulate starting points for our simulation.[39] Conditional on these and the assigned draws of the error structure, we simulate behavior and outcomes forward from exam two, taking care to update the state vector with endogenous variables and associated interaction terms. For example, when an individual is simulated to drink lightly, his or her next period lagged light drinking variable is updated accordingly, regardless of if the person actually drank lightly. To summarize, our sample of 2,497 Framingham Heart Study participants provide their sets of baseline exogenous characteristics, and we simulate the endogenous variables of our model, updating the simulated state vector, to study substitution from alcohol to antidepressants.

Figure 3 evaluates the model fit of our estimated system by graphing the evolution of different simulated behaviors and outcomes and their counterparts in the data.[40] In all cases, our model produces the observed patterns quite well. To further demonstrate that our model does a good job in capturing the data, Table 6 presents simulated transitions for each behavior along with the analogous transition proportion in the data for both men and women. For example, conditional on drinking heavily in period $t-1$, 61.7% of individuals are simulated to be drinking heavily in period t. In the data, that percentage is 58.7%. Capturing transitions is more difficult than capturing averages, yet our model does a good job of recovering the transitions in the data. Finally, Figure 4 demonstrates the importance of modeling the unobserved heterogeneity distribution. For example, Figure 4a shows a significantly higher fraction of Type 4 individuals using antidepressants while these same individuals are much less likely to be drinking heavily. Importantly, despite the fact that each μ term shifts the respective logit equation intercept, the time paths by type are not perfectly parallel. This highlights selection out of the sample by type. While the μ terms enter each equation as linear intercept shifters, they have dynamic, nonlinear effects through their influence on past and future behaviors and outcomes.

[39] Simulating the initial conditions equations prevents us from breaking the link between the initial conditions, the unobserved heterogeneity, and the per-period equations.

[40] In simulation, we assign the median year in the range of years in which each exam could have occurred.

To test our theory of rational self-medication, we simulate our estimated dynamic model under two counterfactual scenarios. As a natural first step, we evaluate a counterfactual in which all sample participants take an antidepressant as soon as SSRIs become available and there onward (i.e., exam 4 through 9). Figure 5a presents results for the entire sample. Heavy drinking declines by approximately five percentage points by the end of the ninth exam. Figures 5b and 5c break the results from Figure 5a by gender, which demonstrates that men are primarily driving our heavy drinking result. Figures 5d, 5e, and 5f break the results from Figure 5a by simulated exam three CES-D tercile. Surprisingly, the reduction in heavy drinking associated with antidepressants is driven by those in the middle tercile, with no reduction in heavy drinking for those simulated to be highly depressed.

One potential explanation for the lack of substitution away from heavy drinking for those simulated to be highly depressed is that with depression comes addiction. If highly depressed individuals face significant reinforcement, tolerance, and withdrawal mechanisms, then alcohol consumption may not change despite improvements in mental health. To investigate, Figure 6 presents results in which we simulate our model setting the value of lagged alcohol consumption to zero (regardless of simulated lagged behavior). Not surprisingly, relative to the baseline simulation, heavy alcohol consumption plummets while light drinking remains unchanged — a roughly equal fraction of light drinkers quit as compared to the fraction of heavy drinkers who move to light drinking. Figure 7 presents the simulated time paths of endogenously chosen antidepressant medication under this counterfactual relative to the baseline simulation. Overall, Figure 7a demonstrates a 5.5 percentage point increase in antidepressant usage by the end of the sample. Figures 7b-7f demonstrate that substitution towards antidepressants is increasing in simulated depression — the largest increase in antidepressant use is in those simulated to be the most depressed. Figure 7 provides clear evidence that addiction inhibits substitution within our estimated system of equations. These results are consistent with rational self-medication.

Finally, Figure 8b contrasts our main finding in Figure 5a, repeated in Figure 8a for comparison, of a roughly five percentage point decline in heavy drinking when antidepressants are imposed on the entire sample with a similar simulation in which we both impose antidepressants and decrease alcohol prices by 10%. Figure 8b shows that the 10% price decrease completely nullifies the antidepressant effect by exam nine. The simulation also demonstrates that prices, which serve an important role with respect to identification of our dynamic system, significantly affect long-term alcohol consumption.

6 Conclusion

We develop a theory of rational self-medication that suggests a relationship between optimal investments in health and the degree of negative symptoms generated by a stock of health. We test our hypothesis by studying alcohol and tobacco consumption when the choice set for the management of depression expands due to technological advancement (i.e., SSRIs). Using a variety of estimators that each make different assumptions, we show that alcohol consumption decreased in the Framingham Heart Study following the introduction of SSRIs. The dynamic model allows us to simulate antidepressant behavior under the counterfactual that alcohol is less addictive, which shows that antidepressant consumption increases by five to six percentage points, and, consistent with the rational self-medication hypothesis, this increase is increasing in the severity of depression.

To the extent that rational self-medication accurately characterizes behavior, our theory has important implications for addiction and health policy. For example, there is considerable public health concern regarding stress-induced alcohol consumption as a result of the COVID-19 pandemic (Clay & Parker, 2020), yet the World Health Organization WHO has recommended that "Existing rules and regulations to protect health and reduce harm caused by alcohol, such as restricting access, should be upheld and even reinforced during the COVID-19 pandemic and emergency situations; while any relaxation of regulations or their enforcement should be avoided."[41] Our work suggests that restrictions to alcohol during COVID-19 may lead anxious and stressed individuals to substitute towards more socially harmful substances. Similarly, given the growing literature on the significant effects of technological innovation on health behaviors, policy should promote treatment innovations that obviate the need to self-medicate and thus induce rational actors to substitute towards less harmful substances. This prescription stands in stark contrast to the vast literature on self-medication.

We acknowledge four main limitations of our work. First, even with forty years of longitudinal data on alcohol, tobacco, and antidepressant consumption, FHS lacks a consistently measured metric of mental health. Ideally, a representative period of our dynamic empirical model would include a time-varying mental health production function which is a function of period t health investments. However, this limitation does not detract from our reduced-form results, presented in Table 3 and Figure 14. Second, while our theory has important implications for current policy, FHS is not representative of a larger population, and thus our results may not extend to at-risk populations in other areas of the United States or for underrepresented groups. Third, our data do not allow us to distinguish between different underlying factors that could drive the comple-

[41] https://www.euro.who.int/en/health-topics/disease-prevention/alcohol-use/news/news/2020/04/alcohol-does-not-protect-against-covid-19-access-should-be-restricted-during-lockdown

mentarity between depression and alcohol consumption, which would be useful to understand to conduct more specific counterfactual policy analysis. Finally, our dynamic system of equations abstracts from an explicitly forward-looking decision-making process. In a fully structural model, an individual's decision to consume alcohol or tobacco would depend on the present discounted value of being in different possible future states, about which an individual would form expectations conditional on contemporaneous behavior. For example, fully-rational self-medicating agents should consider the possibility of future addiction when considering current management of pain, and we are unable to address these expectations with our current estimator. We leave specification and estimation of such a model for future work.

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A Main Tables

Table 1: Baseline Characteristics by Gender and Ever Antidepressant Usage

	Men = 1,241			Women = 1,256		
	Never (87.83%)	Ever (12.17%)	p-value	Never (75.48%)	Ever (24.52%)	p-value
Alcohol Consumption						
None	0.177	0.205	0.398	0.284	0.286	0.947
Light	0.573	0.556	0.691	0.506	0.529	0.485
Heavy	0.250	0.238	0.767	0.210	0.185	0.347
Smokes	0.417	0.430	0.745	0.296	0.370	0.015
Ever Has Cancer	0.414	0.411	0.941	0.343	0.276	0.030
Ever Has CVD	0.372	0.397	0.540	0.203	0.234	0.243
Dies Before Exam 9	0.336	0.285	0.212	0.214	0.091	0.000
Age	45.025	44.093	0.291	44.872	41.292	0.000
Education						
Less than HS	0.017	0.026	0.440	0.006	0.003	0.528
HS Grad.	0.304	0.272	0.419	0.379	0.390	0.732
Some College	0.423	0.404	0.659	0.461	0.481	0.551
College or More	0.185	0.252	0.053	0.098	0.078	0.290
BMI	26.799	27.170	0.230	24.391	24.509	0.702
Obese	0.162	0.199	0.263	0.114	0.120	0.767
Exam 3 CES-Depression Tercile [Range]						
Low [0,4]	0.397	0.291	0.012	0.350	0.250	0.001
Medium [5, 10]	0.350	0.351	0.990	0.349	0.302	0.128
High [11, 51]	0.252	0.358	0.006	0.301	0.448	0.000

Notes: $n = 2,497$. With the exception of the CES-D score, statistics are calculated from exam 2, which took place between 1979 and 1983. The sample is constructed such that an individual must be present for exams 2 and 3, after which an individual may leave the sample through death or attrition. Rows for never and ever antidepressant usage reflect whether the person was ever observed to take an antidepressant. Depression is measured by the CES-D scale, which is broken into terciles. Light drinking is defined as seven or fewer drinks per week for women and 14 or fewer drinks per week for men. Heavy drinking is defined as more than seven drinks per week for women and more than 14 drinks per week for men.

Table 2: Sample Behaviors over Time by Gender.

Men, $n = 8,345$								
Exam	Count	Year Range	Age	Antidepressant	None	Light	Heavy	Smoke
2	1241	1979-1983	44.911	.	0.180	0.571	0.248	0.418
3	1241	1983-1987	49.267	0.010	0.212	0.555	0.233	0.269
4	1198	1987-1991	52.422	0.013	0.264	0.539	0.197	0.234
5	1122	1991-1995	55.603	0.020	0.266	0.546	0.188	0.178
6	1043	1995-1998	59.301	0.036	0.291	0.548	0.161	0.129
7	1005	1998-2001	61.867	0.056	0.276	0.554	0.170	0.116
8	845	2005-2008	67.424	0.088	0.249	0.591	0.161	0.090
9	650	2011-2014	71.462	0.105	0.269	0.554	0.177	0.055
Women, $n = 8,913$								
Exam	Count	Year	Age	Antidepressant	None	Light	Heavy	Smoke
2	1256	1979-1983	43.994	.	0.284	0.512	0.204	0.314
3	1256	1983-1987	48.362	0.021	0.350	0.473	0.177	0.278
4	1225	1987-1991	51.740	0.036	0.343	0.507	0.150	0.219
5	1183	1991-1995	55.173	0.049	0.332	0.525	0.143	0.174
6	1131	1995-1998	59.034	0.084	0.450	0.417	0.133	0.141
7	1107	1998-2001	61.822	0.112	0.388	0.451	0.162	0.114
8	972	2005-2008	67.418	0.186	0.321	0.515	0.164	0.099
9	783	2011-2014	71.775	0.217	0.354	0.469	0.178	0.056

Notes: $n = 17,258$. Statistics are calculated from eight exams, which took place between 1979 and 2011. The sample is constructed such that an individual must be present for exams 2 and 3, after which some individuals are lost to death or attrition. Light drinking is defined as seven or fewer drinks per week for women and 14 or fewer drinks per week for men. Heavy drinking is defined as more than seven drinks per week for women and more than 14 drinks per week for men.

Table 3: Reduced-Form Estimates of Antidepressants on Behavior

	Alcohol Binary Variables			Log Drinks/Week
	None	Light	Heavy	| Drinking
Mean	0.313	0.516	0.171	1.105
St. Dev.	0.464	0.500	0.377	1.142
	Estimates of τ and ν parameters: Equation 11			
Exam 3 CES∈[5, 10] *				
Exam 4	-0.011	0.009	0.002	0.026
	(0.022)	(0.028)	(0.020)	(0.045)
Exam 5	-0.019	0.004	0.016	0.031
	(0.023)	(0.030)	(0.022)	(0.049)
Exam 6	-0.022	0.014	0.008	0.036
	(0.025)	(0.031)	(0.022)	(0.052)
Exam 7	0.006	-0.005	-0.001	-0.021
	(0.025)	(0.031)	(0.022)	(0.052)
Exam 8	-0.039	0.035	0.004	0.014
	(0.024)	(0.031)	(0.023)	(0.056)
Exam 9	-0.022	0.020	0.002	-0.033
	(0.026)	(0.035)	(0.026)	(0.062)
Exam 3 CES∈[11, 51]*				
Exam 4	-0.004	0.034	-0.030	-0.016
	(0.024)	(0.030)	(0.021)	(0.048)
Exam 5	-0.024	0.038	-0.015	-0.003
	(0.025)	(0.031)	(0.022)	(0.051)
Exam 6	0.005	0.030	-0.035	-0.036
	(0.026)	(0.032)	(0.022)	(0.055)
Exam 7	0.012	0.007	-0.018	-0.068
	(0.027)	(0.032)	(0.023)	(0.055)
Exam 8	-0.003	0.034	-0.031	-0.085
	(0.027)	(0.033)	(0.025)	(0.059)
Exam 9	0.034	0.011	-0.045*	-0.094
	(0.030)	(0.036)	(0.027)	(0.065)
F-Test p-value	0.348	0.682	0.478	0.364
Estimator	Estimates of δ: Equations 12 and 13			
OLS	0.099***	-0.078***	-0.021	-0.163***
	(0.024)	(0.022)	(0.017)	(0.054)
OLS + Ind. FE	0.039**	-0.026	-0.013	-0.095***
	(0.017)	(0.019)	(0.014)	(0.036)
OLS + Ind. FE+	0.030	-0.015	-0.015	-0.059*
+ Ever Trends	(0.019)	(0.021)	(0.013)	(0.036)
F-Test p-value	0.310	0.123	0.836	0.433
OLS + Ind. FE+	0.036**	-0.024	-0.012	-0.089**
+ Depression Trends	(0.017)	(0.019)	(0.014)	(0.037)
F-Test p-value	0.563	0.983	0.941	0.792

Notes: The top panel of results are estimated on data from exams two through nine, n=17,258 person/exam observations. All regressions in the bottom panel are estimated on data from exams 3 through 9 (n=14,761). Estimators in both panels include controls for age, education, cardiovascular disease, cancer, body mass index, and exam binary variables. All binary dependent variable results are from linear probability models. P-values in the top panel are with respect to an F-test that $\tau_3 = \nu_3 = 0$. P-values in the bottom panel are with respect to an F-test with null hypothesis that interactions between ever taking a medication (row 3), medium and high baseline depression (row 4) and each exam binary variable are jointly zero. * $p < 0.1$, ** $p < 0.05$ *** $p < 0.01$.

Table 4: Selected Parameter Estimates

	Light Drinking				Heavy Drinking			
	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.
Antidepressant	-0.321	0.219	-0.314	0.303	-0.871	0.336	-1.178	0.444
Antidepressant*								
CES-D$\in [5, 10]$	-0.180	0.237	-0.192	0.319	-0.367	0.375	-0.529	0.500
CES-D$\in [11, 51]$	-0.232	0.222	-0.240	0.303	0.029	0.344	0.331	0.465
Female	0.174	0.190	0.286	0.257	0.765	0.304	1.047	0.400
CES-D$\in [5, 10]$	0.053	0.056	0.183	0.117	0.088	0.078	0.248	0.157
CES-D$\in [11, 51]$	-0.060	0.059	0.482	0.119	-0.147	0.085	1.281	0.237
Female	-0.250	0.049	-0.676	0.085	-0.227	0.070	-0.836	0.116
L. Heavy Drinking	2.476	0.047	1.217	0.070	3.789	0.159	2.567	0.179
L. Light Drinking	2.887	0.092	1.624	0.137	6.795	0.174	4.216	0.198
L. Smoking	-0.122	0.079	0.019	0.111	0.027	0.109	0.083	0.150
Years Smoking	0.001	0.002	-0.006	0.003	0.013	0.003	-0.002	0.004
Years Smoking Cessation	0.008	0.002	0.008	0.003	0.013	0.003	0.021	0.004
Age								
(35, 40]	-0.017	0.178	0.013	0.213	0.401	0.253	0.373	0.293
(40, 45]	-0.135	0.168	-0.151	0.202	0.331	0.238	0.355	0.273
(45, 50]	-0.012	0.176	-0.017	0.210	0.537	0.247	0.695	0.281
(50, 55]	-0.123	0.186	-0.122	0.220	0.572	0.261	0.809	0.296
(55, 60]	-0.060	0.203	-0.111	0.237	0.589	0.287	0.769	0.322
(60, 65]	-0.050	0.228	-0.124	0.264	0.717	0.322	0.877	0.359
(65, 70]	-0.038	0.258	-0.174	0.298	0.751	0.370	0.879	0.412
(70, 75]	-0.194	0.294	-0.461	0.339	0.578	0.423	0.455	0.474
>75	-0.264	0.355	-0.773	0.409	0.377	0.513	-0.083	0.572
Education								
High School	0.185	0.099	0.341	0.171	0.139	0.146	0.246	0.272
Some College	0.410	0.099	0.783	0.170	0.396	0.145	0.699	0.266
College or More	0.530	0.113	1.013	0.192	0.577	0.162	0.905	0.293
(Alcohol CPI * Age)/100	-0.008	0.005	-0.006	0.006	-0.029	0.008	-0.033	0.009
(Cents/cig. Pack * Age)/100	-0.001	0.002	0.001	0.002	0.007	0.002	0.015	0.003
Constant	-1.862	0.228	1.076	0.375	-5.405	0.359	-2.110	0.537
μ_1			0.000	.			0.000	.
μ_2			-1.500	0.271			0.553	0.328
μ_3			-2.116	0.207			-4.841	0.318
μ_4			-4.712	0.247			-4.136	0.346

Notes: $n = 17,258$. Selected parameter estimates are from models estimated on data in exams 2-9.

Table 5: Unobserved Heterogeneity Distribution

	Probability	Medium Dep.	High Dep.	Anti-depressants	Light Drinking	Heavy Drinking	Smoking	Attrition	Death
μ_1	0.241	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
μ_2	0.185	0.070	-1.062***	0.437*	-1.500***	0.553*	0.546***	0.167	0.318
μ_3	0.375	0.081	0.811***	0.269	-2.116***	-4.841***	0.241	-0.314	0.208
μ_4	0.198	0.160	1.084***	0.595***	-4.712***	-4.136***	0.356*	-0.368	0.067

Notes: $n = 17,258$. Selected parameter estimates are from models estimated on data in exams 2-9, with the exception of the multinomial logit for exam 3 depression. Also estimated jointly, but not listed here, are initial conditions equations for drinking and smoking in exam 2. * $p < 0.1$, ** $p < 0.05$ *** $p < 0.01$.

Table 6: Model Fit: Transitions.

	Period t Behavior									
	No Drinking		Light Drinking		Heavy Drinking		Antidepressants		Smoking	
Lagged Behavior $t-1$	Data	Sim.	Data	Sim.	Data	Sim.	Data	Sim.	Data	Sim.
No Drinking	0.671	0.730	0.148	0.156	0.044	0.050	0.372	0.421	0.288	0.304
Light Drinking	0.218	0.259	0.690	0.738	0.295	0.333	0.366	0.440	0.414	0.475
Heavy Drinking	0.008	0.011	0.094	0.106	0.587	0.617	0.118	0.138	0.204	0.220
Antidepressants	0.092	0.117	0.060	0.073	0.065	0.069	0.623	0.748	0.078	0.086
Smoking	0.124	0.139	0.114	0.123	0.195	0.190	0.144	0.152	0.694	0.682

Notes: $n = 17,258$. Results are from models estimated on data in exams 2-9.

Table 7: Antidepressant Parameter Estimates

	Antidepressant Logit Estimates			
	Beta	S.E.	Beta	S.E.
L. Antidepressant	3.814	0.178	3.798	0.188
L. Antidepressant*				
Female	-0.120	0.214	-0.113	0.226
L. Light Drinking * CES-D$\in [5, 10]$	0.453	0.234	0.458	0.238
L. Light Drinking * CES-D$\in [11, 51]$	0.555	0.221	0.573	0.226
L. Heavy Drinking * CES-D$\in [5, 10]$	0.070	0.312	0.091	0.315
L. Heavy Drinking * CES-D$\in [11, 51]$	0.364	0.296	0.496	0.305
L. Light Drinking	-0.680	0.171	-0.445	0.193
L. Heavy Drinking	-0.469	0.222	-0.400	0.285
CES-D$\in [5, 10]$	0.026	0.171	0.011	0.176
CES-D$\in [11, 51]$	0.322	0.159	0.272	0.164
Female	0.657	0.096	0.683	0.102
L. Smoking	0.037	0.145	0.002	0.146
Years Smoking	0.012	0.003	0.012	0.003
Years Smoking Cessation	0.003	0.003	0.003	0.003
Age				
(35, 40]	1.170	1.071	1.156	0.666
(40, 45]	1.800	1.025	1.776	0.586
(45, 50]	2.149	1.018	2.127	0.572
(50, 55]	2.015	1.017	1.988	0.569
(55, 60]	1.747	1.018	1.725	0.568
(60, 65]	1.504	1.020	1.474	0.570
(65, 70]	1.268	1.024	1.238	0.575
(70, 75]	1.460	1.027	1.433	0.578
>75	1.112	1.030	1.095	0.581
Education				
High School	-0.074	0.178	-0.093	0.187
Some College	-0.052	0.178	-0.098	0.188
College or More	0.082	0.204	0.023	0.211
CVD Last Period	0.404	0.213	0.404	0.216
Any History of CVD	-0.037	0.154	-0.029	0.157
Cancer Last Period	0.452	0.191	0.453	0.194
Any History of Cancer	-0.043	0.148	-0.034	0.151
Obese	0.033	0.093	0.033	0.095
Currently Working	-0.261	0.104	-0.264	0.107
Work Missing	0.025	0.109	0.028	0.114
Married	0.371	0.119	0.376	0.124
Married Missing	-0.118	0.170	-0.124	0.182
Exam Trend	0.358	0.030	0.360	0.032
Constant	-7.631	1.044	-8.036	0.646
μ_1			0.000	.
μ_2			0.437	0.244
μ_3			0.269	0.170
μ_4			0.595	0.210

Notes: $n = 17,258$. Selected parameter estimates are from models estimated on data in exams 2-9.

Table 8: Behavior Parameter Estimates

	Light Drinking				Heavy Drinking				Smoking			
	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.
Antidepressant	-0.321	0.219	-0.314	0.303	-0.871	0.336	-1.178	0.444	1.473	0.411	1.452	0.475
Antidepressant*												
CES-D$\in [5, 10]$	-0.180	0.237	-0.192	0.319	-0.367	0.375	-0.529	0.500	-0.904	0.451	-0.944	0.521
CES-D$\in [11, 51]$	-0.232	0.222	-0.240	0.303	0.029	0.344	0.331	0.465	-1.125	0.407	-1.125	0.466
Female	0.174	0.190	0.286	0.257	0.765	0.304	1.047	0.400	-0.719	0.345	-0.707	0.351
CES-D$\in [5, 10]$	0.053	0.056	0.183	0.117	0.088	0.078	0.248	0.157	0.196	0.095	0.206	0.100
CES-D$\in [11, 51]$	-0.060	0.059	0.482	0.119	-0.147	0.085	1.281	0.237	0.386	0.098	0.430	0.105
Female	-0.250	0.049	-0.676	0.085	-0.227	0.070	-0.836	0.116	0.159	0.081	0.162	0.086
L. Light Drinking	2.476	0.047	1.217	0.070	3.789	0.159	2.567	0.179	-0.157	0.092	-0.051	0.119
L. Heavy Drinking	2.887	0.092	1.624	0.137	6.795	0.174	4.216	0.198	0.090	0.110	0.002	0.157
L. Smoking	-0.122	0.079	0.019	0.111	0.027	0.109	0.083	0.150	3.438	0.115	3.430	0.128
Years Smoking	0.001	0.002	-0.006	0.003	0.013	0.003	-0.002	0.004	0.087	0.004	0.087	0.005
Years Smoking Cessation	0.008	0.002	0.008	0.003	0.013	0.003	0.021	0.004	-0.028	0.011	-0.028	0.011
Age												
(35, 40]	-0.017	0.178	0.013	0.213	0.401	0.253	0.373	0.293	-0.074	0.219	-0.090	0.221
(40, 45]	-0.135	0.168	-0.151	0.202	0.331	0.238	0.355	0.273	-0.290	0.215	-0.301	0.215
(45, 50]	-0.012	0.176	-0.017	0.210	0.537	0.247	0.695	0.281	-0.397	0.236	-0.392	0.234
(50, 55]	-0.123	0.186	-0.122	0.220	0.572	0.261	0.809	0.296	-0.685	0.265	-0.669	0.261
(55, 60]	-0.060	0.203	-0.111	0.237	0.589	0.287	0.769	0.322	-0.945	0.305	-0.931	0.300
(60, 65]	-0.050	0.228	-0.124	0.264	0.717	0.322	0.877	0.359	-1.292	0.359	-1.275	0.353
(65, 70]	-0.038	0.258	-0.174	0.298	0.751	0.370	0.879	0.412	-1.466	0.432	-1.446	0.423
(70, 75]	-0.194	0.294	-0.461	0.339	0.578	0.423	0.455	0.474	-1.661	0.508	-1.639	0.501
>75	-0.264	0.355	-0.773	0.409	0.377	0.513	-0.083	0.572	-2.003	0.633	-1.962	0.630
Education												
High School	0.185	0.099	0.341	0.171	0.139	0.146	0.246	0.272	0.021	0.153	0.025	0.162
Some College	0.410	0.099	0.783	0.170	0.396	0.145	0.699	0.266	-0.072	0.154	-0.090	0.164
College or More	0.530	0.113	1.013	0.192	0.577	0.162	0.905	0.293	-0.266	0.185	-0.314	0.196
CVD Last Period	-0.264	0.130	-0.293	0.155	-0.443	0.198	-0.466	0.241	-0.448	0.211	-0.453	0.212
Any History of CVD	-0.132	0.088	-0.262	0.125	-0.123	0.135	-0.361	0.189	0.024	0.149	0.033	0.150
Cancer Last Period	-0.192	0.128	-0.145	0.152	-0.281	0.187	-0.138	0.222	-0.200	0.272	-0.178	0.279
Any History of Cancer	0.185	0.095	0.063	0.126	0.126	0.138	-0.188	0.180	-0.338	0.209	-0.353	0.214
Obese	-0.180	0.052	-0.135	0.076	-0.247	0.077	-0.113	0.112	-0.450	0.088	-0.452	0.094
Currently Working	0.117	0.062	0.142	0.078	-0.032	0.090	-0.032	0.112	-0.057	0.108	-0.058	0.111
Work Missing	0.096	0.064	0.098	0.087	0.134	0.095	0.159	0.125	-0.196	0.111	-0.193	0.114
Married	0.387	0.076	0.513	0.095	0.453	0.113	0.695	0.142	0.387	0.147	0.399	0.154
Married Missing	0.287	0.086	0.347	0.101	-0.010	0.124	0.029	0.149	0.369	0.147	0.374	0.154
Exam Trend	0.247	0.052	0.169	0.061	0.373	0.076	0.204	0.091	0.508	0.101	0.512	0.103
(Alcohol CPI * Age)/100	-0.008	0.005	-0.006	0.006	-0.029	0.008	-0.033	0.009	-0.028	0.009	-0.028	0.010
(Cents/cig. Pack * Age)/100	-0.001	0.002	0.001	0.002	0.007	0.002	0.015	0.003	-0.005	0.004	-0.005	0.004
Constant	-1.862	0.228	1.076	0.375	-5.405	0.359	-2.110	0.537	-4.459	0.372	-4.754	0.415
μ_1			0.000	.			0.000	.			0.000	.
μ_2			-1.500	0.271			0.553	0.328			0.546	0.194
μ_3			-2.116	0.207			-4.841	0.318			0.241	0.164
μ_4			-4.712	0.247			-4.136	0.346			0.356	0.198

Notes: $n = 17,258$. Selected parameter estimates are from models estimated on data in exams 2-9.

Table 9: Outcome Parameter Estimates

	Sample Attrition				Mortality			
	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.
Antidepressant	-0.133	0.435	-0.131	0.598	0.661	0.358	0.647	0.378
Antidepressant*								
CES-D$\in [5, 10]$	0.204	0.459	0.176	0.629	-0.174	0.453	-0.163	0.479
CES-D$\in [11, 51]$	0.097	0.432	0.114	0.586	-0.317	0.421	-0.312	0.450
Female	0.214	0.369	0.206	0.389	-0.403	0.344	-0.392	0.352
CES-D$\in [5, 10]$	0.309	0.135	0.328	0.143	0.146	0.113	0.152	0.115
CES-D$\in [11, 51]$	0.422	0.140	0.509	0.149	0.085	0.123	0.114	0.126
Female	0.107	0.114	0.090	0.119	-0.430	0.104	-0.446	0.109
Light Drinking	-0.024	0.116	-0.197	0.162	-0.390	0.102	-0.390	0.143
Heavy Drinking	-0.061	0.162	-0.466	0.246	-0.260	0.134	-0.362	0.224
Smoking	0.470	0.177	0.477	0.182	0.347	0.144	0.330	0.145
Years Smoking	0.006	0.004	0.004	0.004	0.012	0.003	0.011	0.003
Years Smoking Cessation	-0.003	0.004	-0.002	0.004	-0.007	0.004	-0.006	0.004
Age								
(40, 45]	-0.654	0.348	-0.644	0.386	1.176	0.771	1.170	0.476
(45, 50]	-1.068	0.343	-1.050	0.384	1.770	0.736	1.769	0.410
(50, 55]	-1.371	0.333	-1.350	0.378	1.965	0.727	1.970	0.390
(55, 60]	-1.168	0.314	-1.148	0.363	2.304	0.721	2.300	0.375
(60, 65]	-0.854	0.312	-0.834	0.365	2.534	0.720	2.533	0.371
(65, 70]	-0.949	0.333	-0.932	0.387	2.724	0.725	2.719	0.375
(70, 75]	-0.921	0.350	-0.900	0.407	3.060	0.729	3.057	0.381
>75	0.241	0.341	0.254	0.399	3.706	0.732	3.700	0.387
Education								
High School	-0.052	0.204	-0.028	0.216	-0.177	0.149	-0.177	0.155
Some College	-0.158	0.205	-0.124	0.218	-0.344	0.152	-0.349	0.159
College or More	-0.344	0.244	-0.311	0.258	-0.564	0.198	-0.575	0.205
CVD this period	-0.062	0.172	-0.063	0.178	1.838	0.105	1.843	0.108
Any History of CVD	0.115	0.141	0.112	0.144	0.484	0.105	0.485	0.108
Cancer this period	-0.024	0.166	-0.028	0.167	1.632	0.105	1.638	0.107
Any History of Cancer	-0.340	0.143	-0.359	0.145	0.888	0.111	0.885	0.112
Obese	0.143	0.119	0.152	0.120	-0.081	0.109	-0.085	0.110
Currently Working	0.041	0.140	0.047	0.147	-0.381	0.135	-0.381	0.140
Work Missing	0.150	0.178	0.151	0.193	-0.150	0.133	-0.147	0.135
Married	0.674	0.186	0.691	0.196	-0.140	0.145	-0.132	0.149
Married Missing	0.536	0.248	0.547	0.288	0.115	0.159	0.116	0.163
Exam Trend	0.703	0.057	0.703	0.061	-0.076	0.036	-0.076	0.039
Constant	-7.769	0.455	-7.520	0.522	-5.336	0.764	-5.467	0.488
μ_1			0.000	.			0.000	.
μ_2			0.167	0.231			0.318	0.241
μ_3			-0.314	0.216			0.208	0.209
μ_4			-0.368	0.272			0.067	0.263

Notes: $n = 17,258$. Selected parameter estimates are from models estimated on data in exams 2-9.

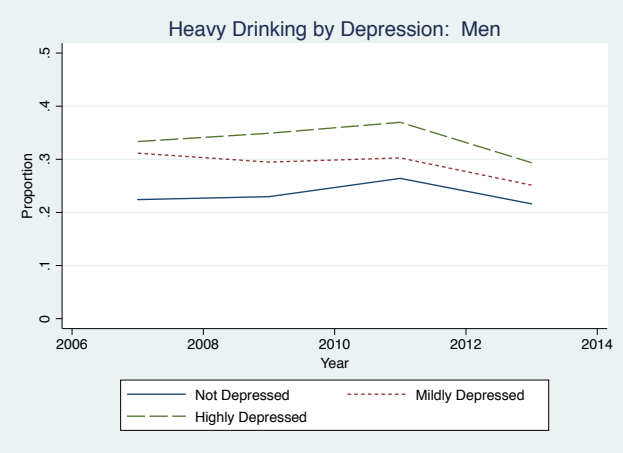
Table 10: Initial Conditions Parameter Estimates

	Light Drinking		Heavy Drinking		Medium Depression		Heavy Depression		Smoking	
	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.	Beta	S.E.
Age	-0.038	0.011	-0.025	0.014	-0.005	0.009	-0.028	0.010	-0.016	0.008
Female	-0.739	0.126	-0.869	0.163	0.150	0.101	0.477	0.108	-0.524	0.087
Education										
High School	0.303	0.242	-0.025	0.334	-0.181	0.218	-0.321	0.222	-0.481	0.172
Some College	0.612	0.242	0.060	0.328	-0.265	0.217	-0.679	0.223	-0.870	0.172
College or More	1.146	0.289	0.219	0.380	-0.253	0.242	-0.827	0.259	-1.283	0.201
Age > 50	0.397	0.215	0.542	0.268	-0.059	0.180	0.116	0.191	-0.067	0.156
					-0.005	0.154	0.059	0.155		
					0.027	0.216	0.936	0.249		
					-0.251	0.146	-0.043	0.153		
					0.015	0.005	0.022	0.005		
					-0.005	0.007	-0.011	0.007		
Constant	3.952	0.583	3.025	0.726	0.227	0.514	0.501	0.544	1.107	0.364
μ_1	0.000	.	0.000	.	0.000	.	0.000	.	0.000	.
μ_2	-0.184	0.574	1.410	0.613	0.070	0.269	-1.062	0.330	0.744	0.160
μ_3	-1.463	0.351	-3.944	0.523	0.081	0.205	0.811	0.227	-0.139	0.157
μ_4	-3.512	0.340	-3.560	0.439	0.160	0.275	1.084	0.277	-0.065	0.163

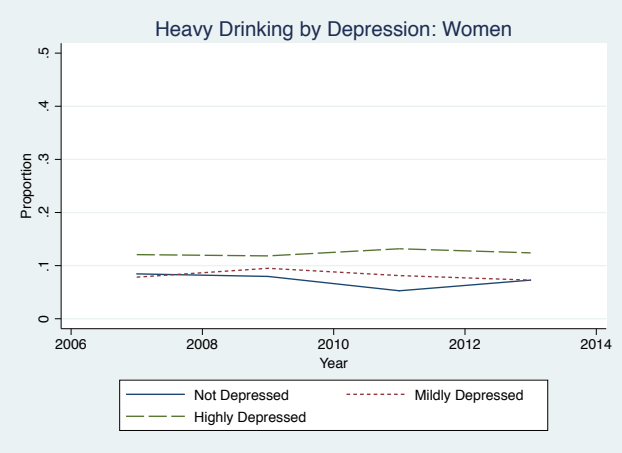
Notes: $n = 17,258$. Selected parameter estimates are from initial condition models. For smoking and drinking, models are estimated on data from exam 2. For depression, data come from the exam 3 CES-D survey.

B Main Figures

Figure 1: Alcohol and Depression: Evidence from NHANES



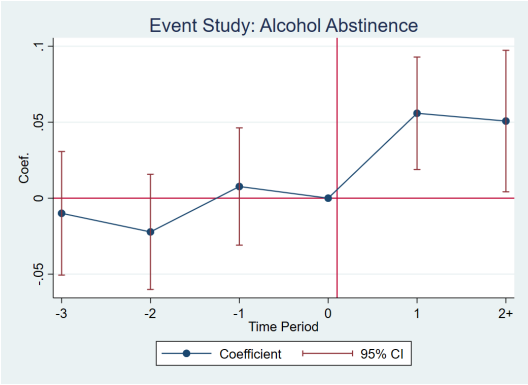
a. Heavy Drinking by Depression, Men



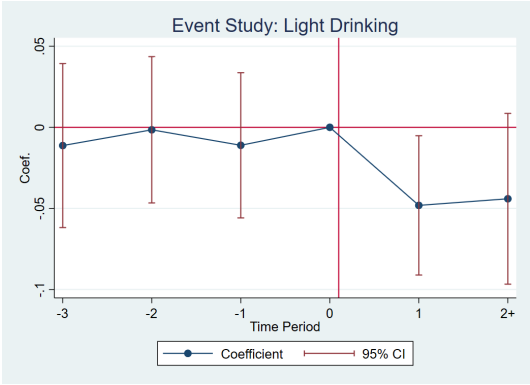
b. Heavy Drinking by Depression, Women

Notes: Author's calculations from NHANES data from 2007-2013. Heavy drinking is defined here as more than three drinks per day on days in which the respondent drank alcohol. Proportions are weighted by the NHANES full sample 2-year interview weight. Proportions are presented by terciles of the Patient Health Questionnaire (PHQ-9) Depression Score. $n = 16,940$.

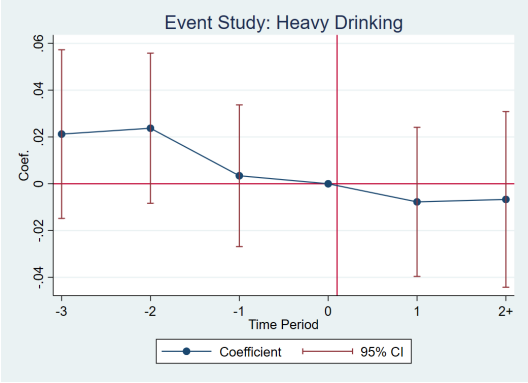
Figure 2: Event Study Analysis



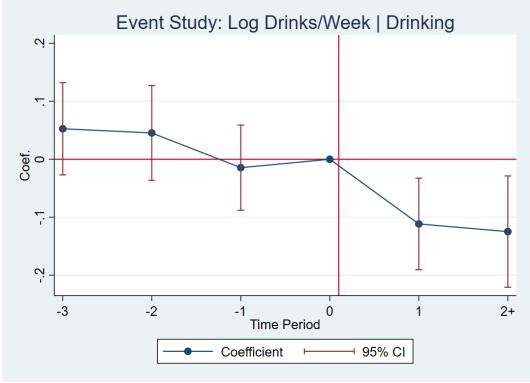
a. No Alcohol



b. Light Drinking



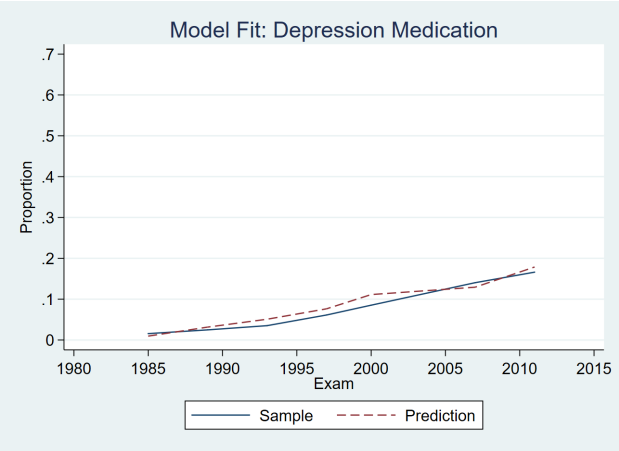
c. Heavy Drinking



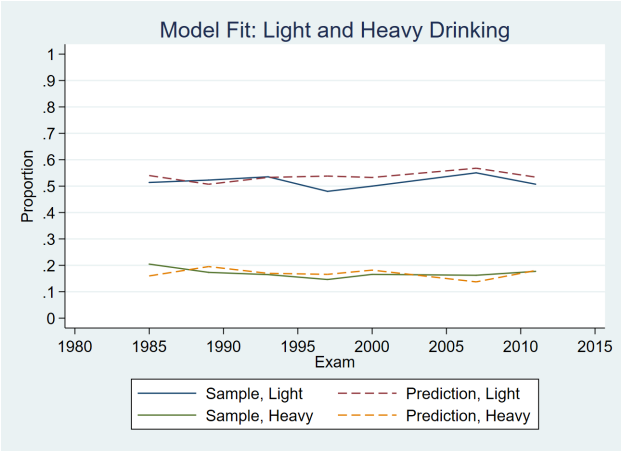
d. Log Drinks/Week Conditional on Drinking

Notes: The figures present estimated event study coefficients for different dependent variables. Period 0, which represents the exam prior to the first exam in which an individual is observed to be taking antidepressants. Because most individuals who take antidepressants initiate use towards the end of our sample, we combine periods greater than or equal to two periods after initial antidepressant usage. The p-values on the F-Test that all coefficients prior to taking an antidepressant are zero are 0.318, 0.782, 0.643, and 0.408, respectively.

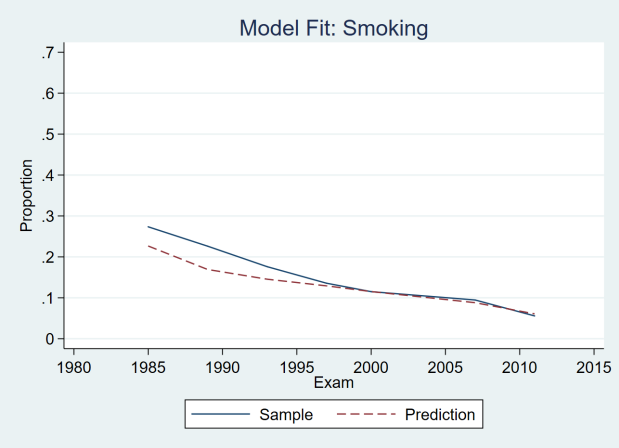
Figure 3: Model Fit



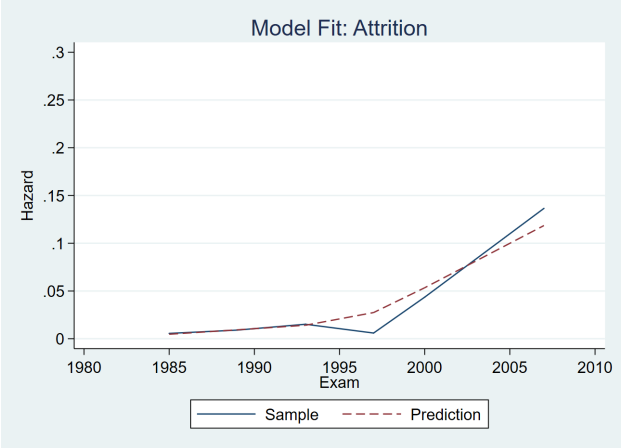
a. Antidepressants



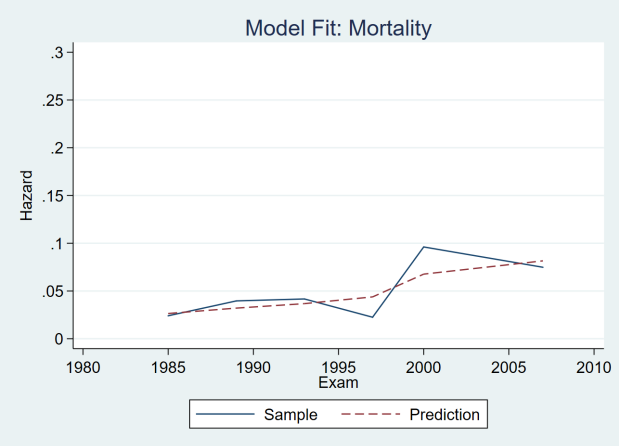
b. Light and Heavy Drinking



c. Smoking



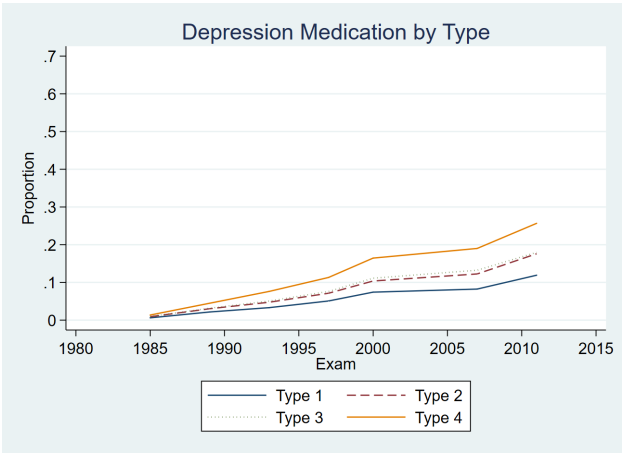
d. Sample Attrition



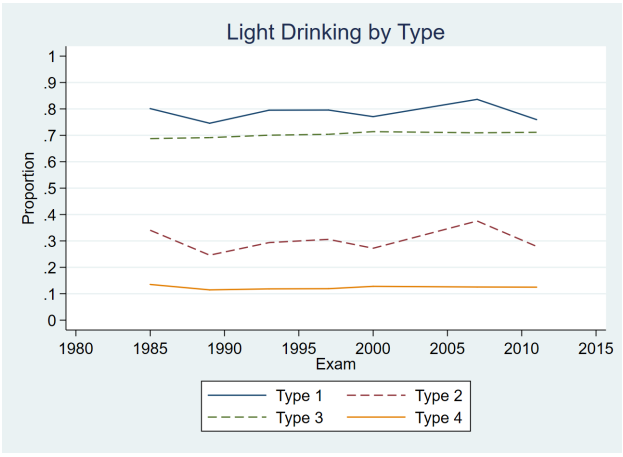
e. Mortality

Notes: Each figure presents results from the baseline simulation of our estimated dynamic model relative to sample data.

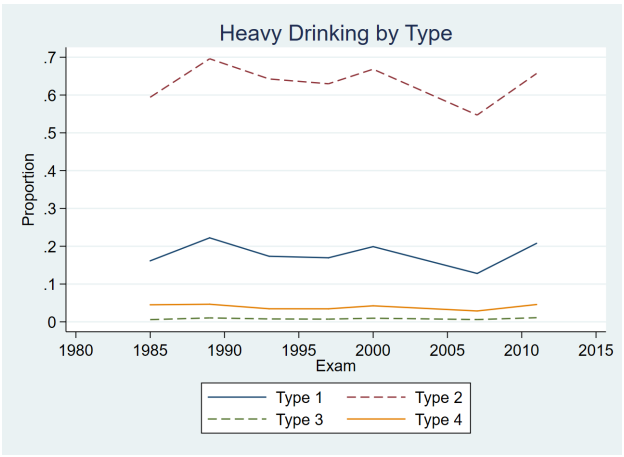
Figure 4: Behaviors and Outcomes by Unobserved Type



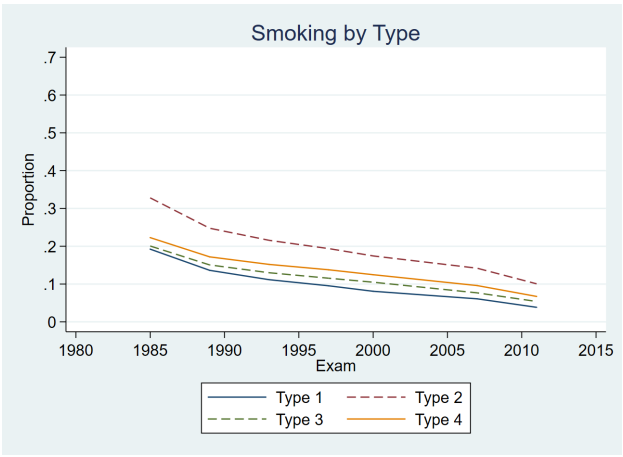
a. Antidepressants



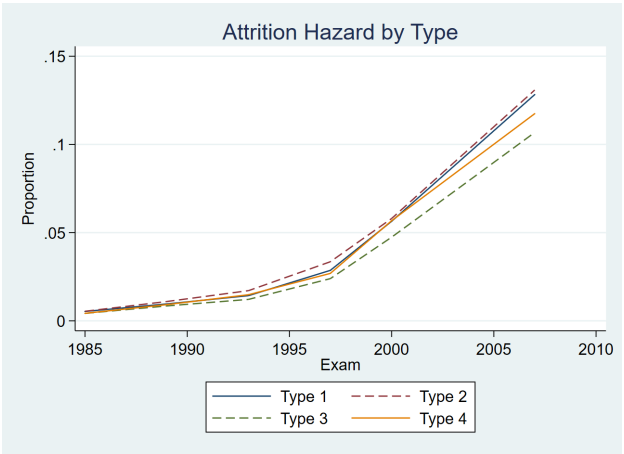
b. Light Drinking



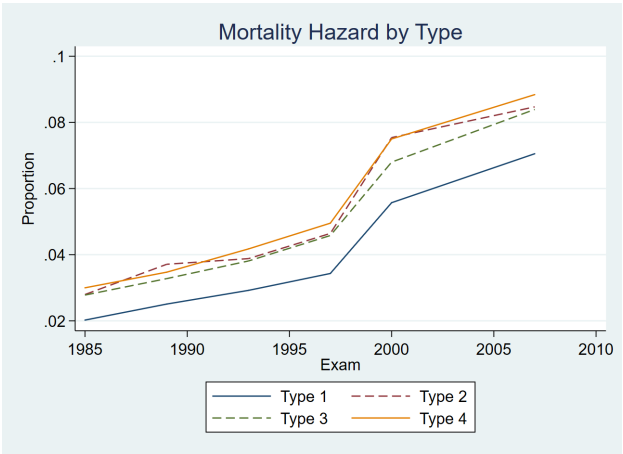
c. Heavy Drinking



d. Smoking



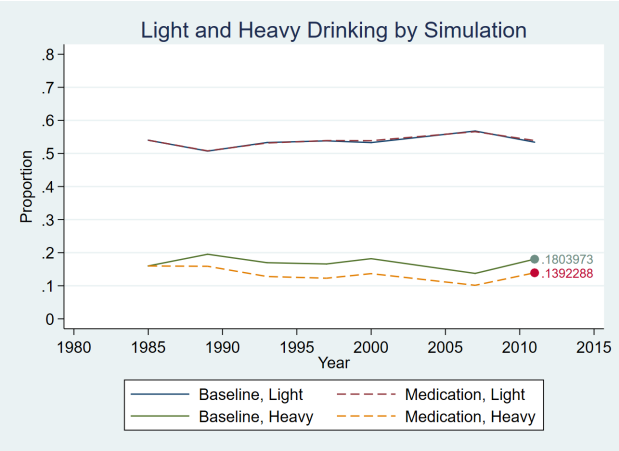
e. Sample Attrition



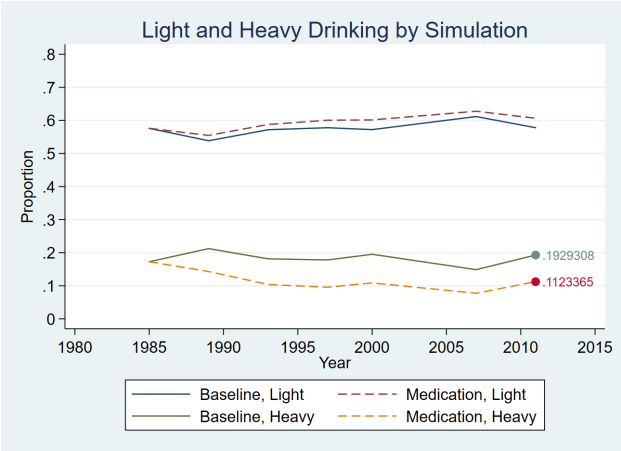
f. Mortality

Notes: Each figure presents results from the baseline simulation of our estimated dynamic model by each of the four unobserved types.

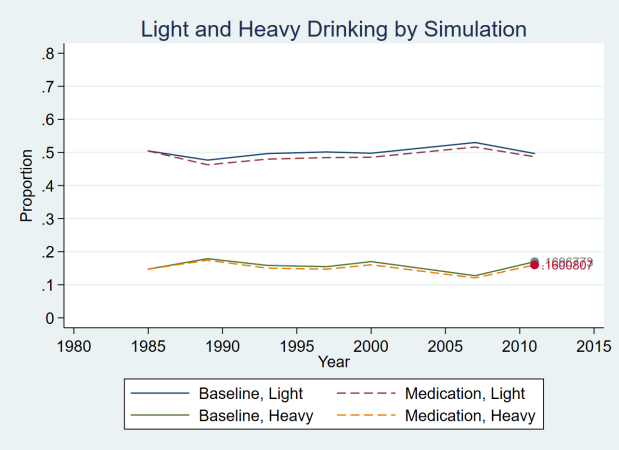
Figure 5: Comprehensive Antidepressants vs. Baseline: Alcohol Consumption



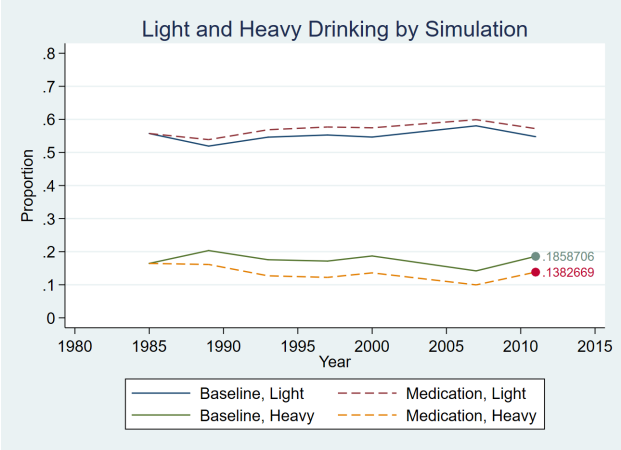
a. Overall



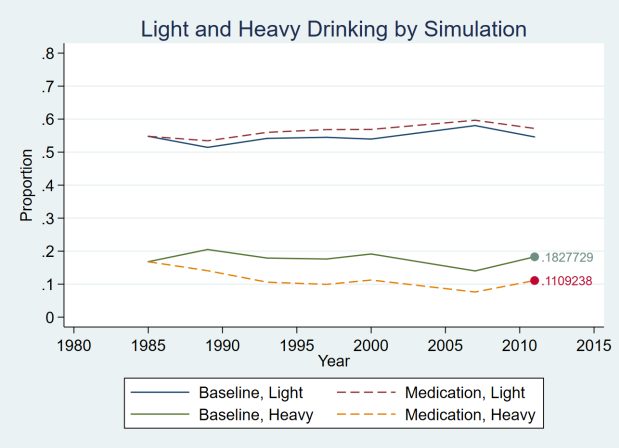
b. Men



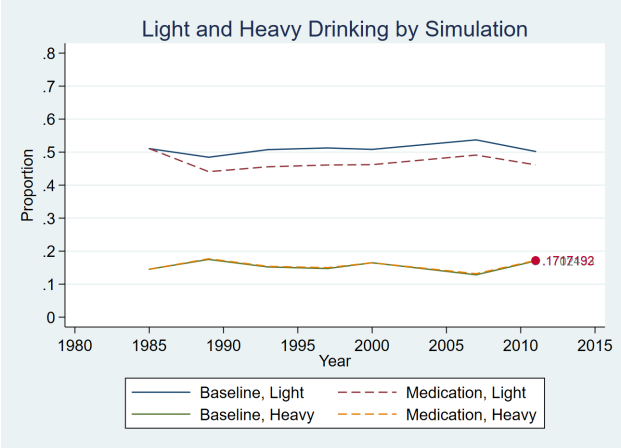
c. Women



d. Low Depression



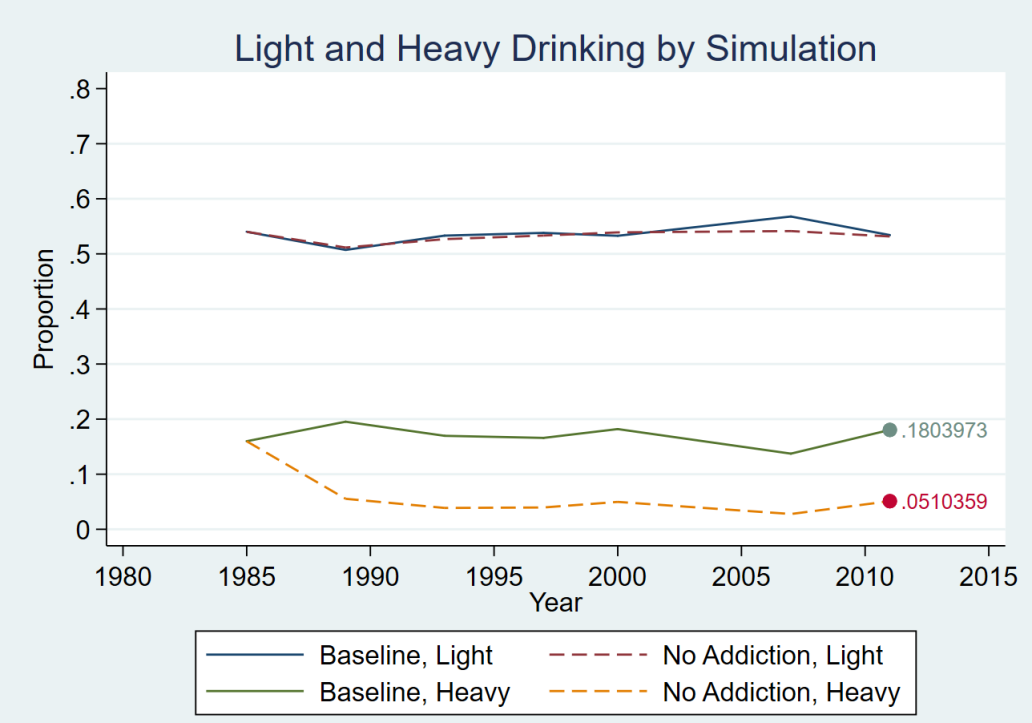
e. Medium Depression



f. High Depression

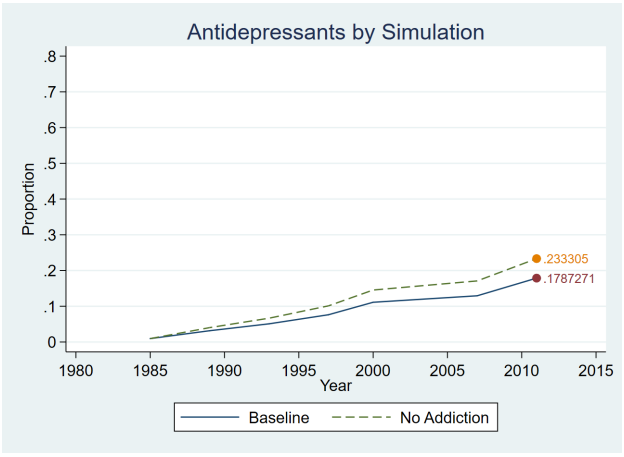
Notes: Each figure presents baseline simulated trends in light and heavy drinking as well as those behaviors when we impose that all individuals take an antidepressant from exam 4 onwards. Figure 5a presents the simulations for the entire sample. Figures 5b and 5c present results separately for men and women. Figures 5d, 5e, 5f present results for those simulated at exam 3 to be in the low, medium, or high terciles of CES-Depression score.

Figure 6: Alcohol Consumption by Simulation

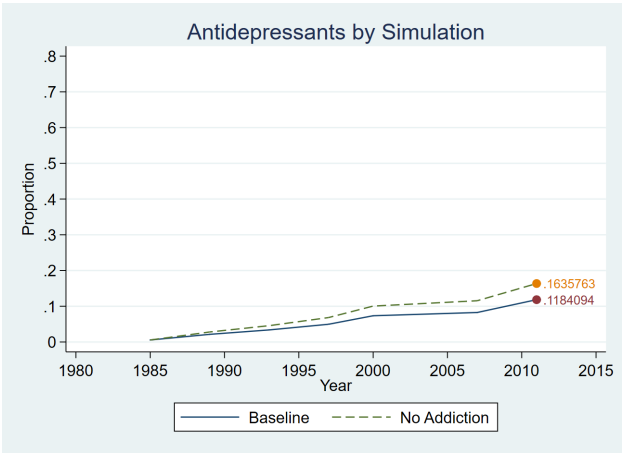


Notes: Figure displays light and heavy smoking under the counterfactual scenario that past alcohol consumption does not factor in any of the contemporaneous period behavioral equations. Results are presented relative to the baseline simulation.

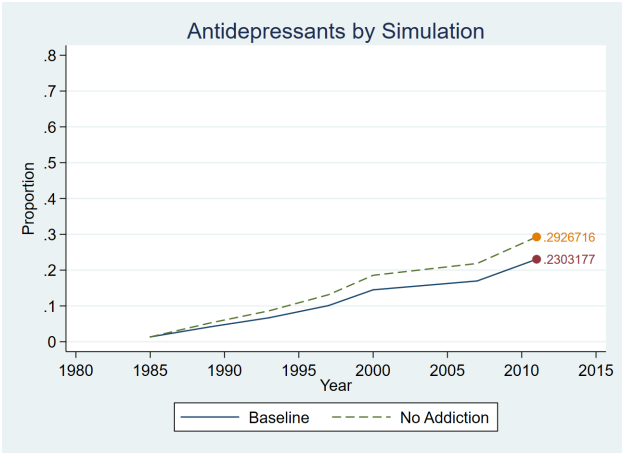
Figure 7: Antidepressant Consumption by Simulation



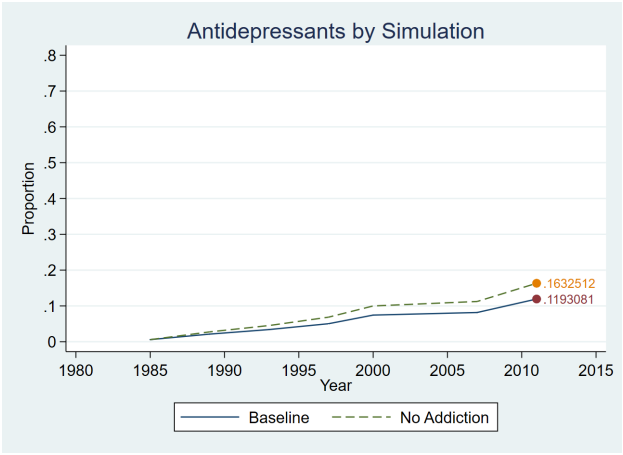
a. Overall



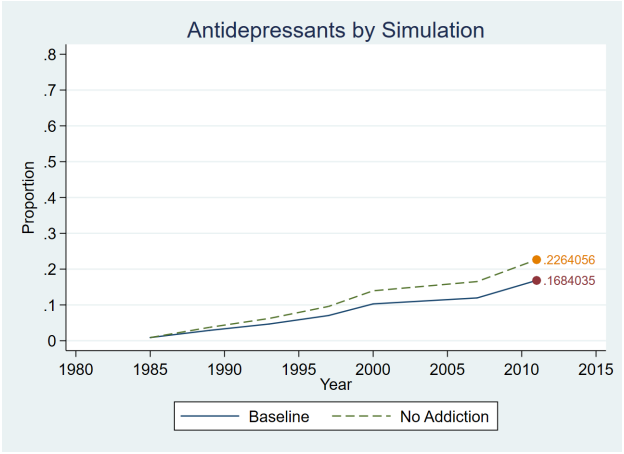
b. Men



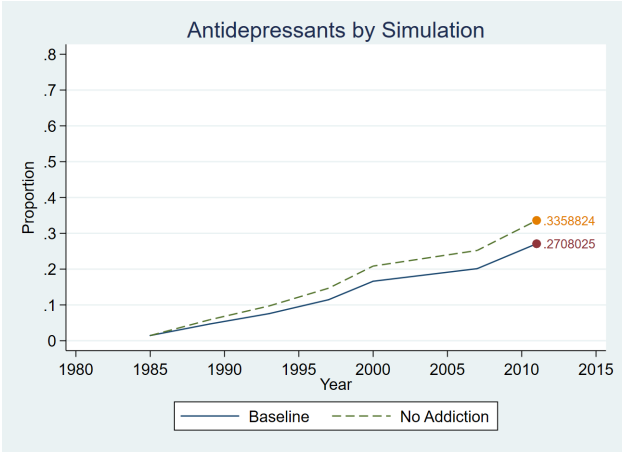
c. Women



d. Low Depression



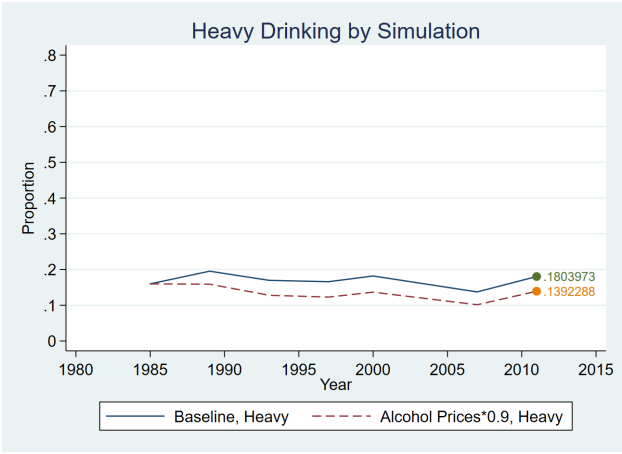
e. Medium Depression



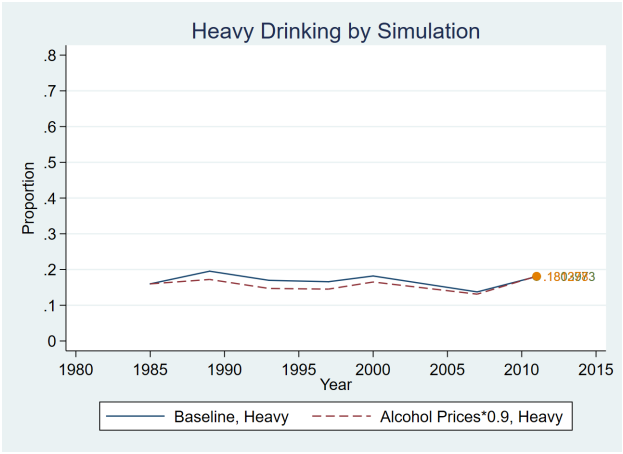
f. High Depression

Notes: Each figure presents simulated trends in antidepressant usage under the baseline scenario as well as under the counterfactual in which we remove the dependence on past alcohol consumption in all behavioral equations. Figure 7a presents the simulations for the entire sample. Figures 7b and 7c present results separately for men and women. Figures 7d, 7e, 7f present results for those simulated at exam 3 to be in the low, medium, or high terciles of CES-Depression score.

Figure 8: The Role of Alcohol Prices



a. Simulation 1



b. Simulation 1 + price effect

Notes: Figure 8a presents simulated trends in heavy alcohol consumption under the baseline scenario as well as under the counterfactual in which we impose antidepressants on all participants at exam 4. This figure is identical to the heavy drinking trend presented in Figure 5. Figure 8b presents the same baseline simulation in heavy drinking along with imposed antidepressants and a decrease in alcohol prices by 10% of baseline levels in all exams after the third.