NBER WORKING PAPER SERIES

RATIONAL SELF-MEDICATION

Michael E. Darden
Nicholas W. Papageorge

Working Paper 25371
http://www.nber.org/papers/w25371

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
December 2018, Revised November 2023

We thank Robert Kaestner, John Mullahy, Jon Skinner, Tom Mroz, Matthew Harris, Melinda Pitts, Peter Savelyev, Matthew Zahn, and seminar participants at the Atlanta Federal Reserve Bank, University of Tennessee, 2018 Workshop on the Economics of Risky Behavior, the 2018 American Society of Health Economists Conference, the 2018 European Health Economics Association Conference, IUPUI, the Southeastern Health Economics Working Group, UNC-Chapel Hill, and the University of South Carolina. The Framingham Offspring Study (FOS) is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the FOS Investigators. This manuscript was prepared using a limited access data set obtained from the NHLBI and does not necessarily reflect the opinions or views of the FOS or the NHLBI. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

Rational Self-Medication
Michael E. Darden and Nicholas W. Papageorge
NBER Working Paper No. 25371
December 2018, Revised November 2023
JEL No. I10,I12

ABSTRACT

We develop a model of rational self-medication in which individuals use potentially dangerous or addictive substances (e.g., alcohol) to manage symptoms of illness (e.g., depression) outside of formal medical care. A model implication is that the emergence of better treatments reduces incentives to self-medicate. 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 reduction in alcohol consumption when SSRIs became available. Our findings illustrate how the effects of medical innovation operate, in part, through changes in behavior.

Michael E. Darden
Carey School of Business
Johns Hopkins University
100 International Dr.
Baltimore, MD 21202
and NBER
mdarden4@jhu.edu

Nicholas W. Papageorge
Department of Economics
Johns Hopkins University
3400 N. Charles Street
Baltimore, MD 21218
and IZA
and also NBER
papageorge@jhu.edu

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 exercising, eating healthy, abstaining from risky behavior, or using medication, can be viewed as costly investments in human capital. In the basic model, individuals invest in their health until the expected marginal long-term benefits of doing so cease to outweigh the marginal upfront costs. The model highlights how human behavior and the tradeoffs people face contribute to health outcomes at both the individual and the population level. The initial phases of COVID-19 offer a stark example. Across the globe, people faced a tradeoff between their health and their livelihoods, and their decisions not only affected their own lives, but also the economy and the spread of illness.[1]

The original Grossman model has been extended to incorporate various features reflecting the reality of health decisions, including addiction, side effects of medication, information, and retirement. We explore an idea that is often discussed non-scientifically and has been examined outside of economics, but that has received scant attention in economic studies of health behaviors: *self-medication* with potentially dangerous or addictive substances. Khantzian (1985) introduces a concept of self-medication in which an individual manages their ailments outside of formal prescription medicine or therapy (e.g., drinking to manage depression or anxiety). However, most studies cast such behavior as problematic and as something to be curbed. This perspective overlooks how self-medication can be an optimal choice. The idea is that in the absence of good treatment options individuals may take matters into their own hands to alleviate their symptoms, even if doing so has potential costs. Viewed within the Grossman framework, they do so rationally in the sense that their behavior takes account of the full set of dynamic costs and benefits. We refer to this behavior as "rational self-medication." The question of whether self-medication is appropriately viewed as a rational decision to alleviate symptoms of illness has implications for policy. For example, restricting access to substances people use to manage their symptoms could compel them to substitute towards even more dangerous substances.

In this paper we examine rational self-medication in the context of alcohol and depression. We study alcohol use before and after the 1987 Food and Drug Administration approval of selective serotonin reuptake inhibitors (SSRIs), a major advancement in the management of depressive symptoms.[2] While SSRIs were not more effective than earlier antidepressants, they produced less harm to long-run health. A lower "shadow price" led to rapid increases in the use of antidepressants once SSRIs were introduced.

To begin, we develop a theoretical link between SSRI introduction and self-medication with alcohol. We present a simple Grossman-style model of rational self-medication that formalizes the idea that risky behaviors (i.e., enjoyable activities with potentially long-run negative consequences) can also provide

[1] In the context of vaccine hesitancy, Francis Collins, the outgoing director of NIH, stated, "Maybe we underinvested in research on human behavior." See https://www.pbs.org/newshour/show/dr-collins-reflects-on-career-at-nih-covid-response-effort-work-on-genome-sequencing.

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

therapeutic benefits. In the model, people may drink despite potential future harm to health (e.g., liver damage) not only because they enjoy being inebriated but also because doing so relieves symptoms of depression. A key implication we derive is that if alcohol is used in part as self-medication, the introduction of SSRIs should render this role redundant and thus lead to less alcohol consumption. Alternatively, if alcohol is not a form of self-medication and its sole benefit is that people enjoy it, the introduction of SSRIs should have no impact on drinking. The key assumption underlying this implication is that alcohol is less effective at reducing symptoms that SSRIs have already reduced or eliminated.

To test this prediction of the model we examine forty years of longitudinal data on alcohol, antidepressants, and depression from the Framingham Heart Study (FHS). We show that SSRIs led to a decline in alcohol consumption, especially on the extensive margin among people with clinical depression, as measured by the Center for Epidemiological Studies - Depression (CES-D) score. Prior to the development of SSRIs, roughly 14% of FHS participants met the CES-D criteria for depression, and we show that these individuals drank significantly more on the intensive margin relative to non-depressed participants. These individuals were also much more likely to use antidepressants following the introduction of SSRIs. Overall, we find that antidepressant usage is associated with an increase in alcohol abstinence of 9.5% for men and 7.2% for women; for those meeting the CES-D criteria for depression, an antidepressant increases alcohol abstinence by 22.3% for men and 11.3% for women. These results are consistent with a complementarity between depression and alcohol—when SSRIs were introduced, their usage led to a reduction in depressive symptoms, which, for some, obviated the need for alcohol.

In summary, our empirical work coupled with the model we offer provide evidence that, for some people, self-medication is the optimal choice given the set of options and constraints they face, including the lack of better ways to manage their symptoms. Our findings have implications for policy and the evaluation medical innovation. Regarding policy, we first note that a vast literature on self-medication documents the phenomenon.[3] Much of this literature is concerned with whether self-medication is a reasonable way to explain problem use of addictive substances (sometimes called the "self-medication hypothesis"). Thus, a contribution to this literature is to provide evidence that it is. Furthermore, most policy proposals from earlier literature on self-medication amount to suggestions to curb it (e.g., restricting access; see, e.g., Twombly & Holtz (2008)) and fail to account for endogenous behavioral responses. Such proposals seem to follow from the idea that self-medication is unambiguously harmful or that the prospect of short-term relief driving the choice to self-medicate does not merit serious enough consideration to affect policy. By modeling self-medication as a rational choice under uncertainty, our work suggests caution in developing policy surrounding self-medication. Restrictions to access can backfire if rational individuals respond by turning to even more harmful substances that have some likelihood of relieving current symptoms. This is consistent with evidence that the reformulation of OxyContin to curb addiction may have increased heroin usage (Dart et al., 2015). Relatedly, Powell et al. (2018) show that

[3] A Google Scholar search for research with "self-medication" in the title yields 209,000 results. This search was conducted on 10/30/2023.

less restrictive medical marijuana laws, and in particular higher numbers of marijuana dispensaries, are associated with fewer opioid overdoses.

Another implication of our findings pertains to the evaluation of medication innovation. While other papers, such as Grossmann & Strulik (2021), explore the socioeconomic factors that cause mental distress, our work takes depression as given and asks how technological innovation (i.e., SSRIs), causes behavioral changes. A literature in health economics moves beyond assessing the direct effects of medical innovation (e.g., lower mortality and better health) to incorporate a more complete set of factors. 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 expected impacts on longer-run life cycle 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 HIV treatments 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 of Statin pharmaceuticals 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 health has likely been understated because randomized trials do not account for subsequent shifts in alcohol consumption.

2 A Model of Rational Self-Medication in the Context of Mental Health

We present a simple two-period model to formalize intuition and develop hypotheses about the impact of an improvement to antidepressants on both antidepressant use and alcohol use. SSRIs were as effective as earlier antidepressants at reducing symptoms of mental illness and improving mental health but had fewer adverse side effects deleterious to future health (i.e., they were safer) which can be seen as a reduction in the "shadow price" of antidepressants. A lower shadow price means usage should either increase or stay the same. Since antidepressants are effective, increased use should lead to improvements in mental health, which obviated the need to use inferior modes of treatment to manage mental health symptoms. If alcohol had been used to treat symptoms of depression (i.e., as way to self-medicate in the absence of alternative viable options), the introduction of SSRIs diminished or eliminated this benefit, which should lead to a decline in the use of alcohol.

Agents in the model choose whether or not to use antidepressants, denoted $d \in \{0, 1\}$ and how much alcohol to drink $a \in [0, \infty)$. Individuals are either depressed or not, which we denote as $M \in \{0, 1\}$, where $M = 0$ means they are not depressed and $M = 1$ means they are. We assume a two period model, where

lifetime utility is expressed as follows:

$$V(M) = \max_{a,d} u(a) - \nu(d) - S(a, d; M) + \beta F(a, d; M). \tag{1}$$

Here, $V(M)$ is the value function, the maximum attainable lifetime utility given state variable M. $u(a)$ is the period utility the agent derives from consuming a and is an increasing and concave function. $\nu(d)$ is the utility cost of consuming an antidepressant where $\nu(d = 1) > \nu(d = 0)$ and includes monetary costs along with non-pecuniary costs, such as side effects, time costs obtain a prescription, concerns related to stigma, etc. Both u and ν are assumed to be independent of M. Moreover, the marginal utility of alcohol, denoted u_a does not depend on d, i.e., how much individuals enjoy an additional unit of alcohol does not depend on whether or not an individual is depressed or taking antidepressants and the costs of taking antidepressants do not depend on mental health.

$S(a, d; M)$ captures how either alcohol and antidepressants serve as treatments in the sense that they reduce depression symptoms. $S(a, d; M)$ is a decreasing and convex function in a (i.e., alcohol reduces the symptoms of depression). We further assume that $S(a, d = 1; M = 1) < S(a, d = 0; M = 1)$ and that $S(\cdot; M = 0) = 0$. These conditions mean that taking antidepressants lowers symptoms for people with depression and has no impact on symptoms for people without depression, for whom symptoms are assumed equal to zero. How a and d interact to reduce symptoms will be discussed below.

$F(a, d; M)$ captures the second-period payoff from a and d, which is discounted by factor β. It is decreasing and concave in a and we assume that $F(a, d = 1; M) < F(a, d = 0; M)$, which means alcohol and antidepressant use can lower future utility. Finally, we assume that the impact of alcohol on the payoff F, denoted F_a, is independent of d and M. Notice, under the stated assumptions, the value function is concave (the negative of a decreasing and convex function S is increasing and concave).

We now use this model to develop three hypotheses. To do so, we characterize optimal alcohol use when agents optimally do not choose antidepressants ($d^\star = 0$) and when they do ($d^\star = 1$) with the following first order conditions:

$$u_a(a^\star) - S_a(a^\star, d^\star = 0) + \beta F_a(a^\star, d^\star = 0) = 0 \tag{2}$$

and

$$u_a(a^{\star\star}) - S_a(a^{\star\star}, d^\star = 1) + \beta F_a(a^{\star\star}, d^\star = 1) = 0 \tag{3}$$

where $a^\star$ and $a^{\star\star}$ are optimal alcohol when $d^\star = 0$ and $d^\star = 1$, respectively.

1. A reduction in the shadow price of antidepressants through the introduction of a safer version, SSRIs, should not affect uptake of antidepressants or shifts in alcohol use among people who do not suffer depression. To see this, first note that for a person who is not depressed, $S(a, d = 1; M =$

$0) = S(a, d = 0; M = 0) = 0$, so the first-order-conditions for alcohol use are:

$$u_a(a^\star) + \beta F_a(a^\star, d = 0) = 0 \tag{4}$$

and

$$u_a(a^{\star\star}) + \beta F_a(a^{\star\star}, d = 1) = 0. \tag{5}$$

F_a and u_a are independent of d, which means that $a^{\star\star} = a^\star$ for non-depressed people. That is, whether or not they use antidepressants, individuals without depression will choose the same amount of alcohol. Thus, we need only evaluate the lifetime utility of $d = 1$ versus $d = 0$ for the same value of $a^\star$. Non-depressed agents are worse off consuming antidepressants if:

$$\begin{aligned} u(a^\star) - \nu(d = 1) + \beta F(a^\star, d = 1; M = 0) &< u(a^\star) - \nu(d = 0) + \beta F(a^\star, d = 0; M = 0) \\ -\nu(d = 1) + \beta F(a^\star, d = 1; M = 0) &< -\nu(d = 0) + \beta F(a^\star, d = 0; M = 0) \end{aligned} \tag{6}$$

which holds because the first (second) term on the left-hand-side of the inequality is less than the first (second) term on the right-hand-side.

2. We now focus on individuals with depression ($M = 1$). If there is a reduction in the shadow price of antidepressants because of smaller-in-magnitude negative future consequences, captured by F, antidepressant use will stay the same or increase among people with depression. Consider the case in which an individual is indifferent between taking or not taking antidepressants prior to the introduction of SSRIs. The introduction of SSRIs means there is an upward shift to $F(a^\star, d = 1; M = 1) - F(a^\star, d = 0; M = 1)$, which means that, keeping alcohol use constant at $a^\star$, the agent would prefer antidepressant use since the costs have gone down. A shift to a new optimal level of alcohol consumption given antidepressant usage ($a^{\star\star}$) would further increase lifetime utility. However, it is not clear absent further assumptions how alcohol use would change, a question we turn to now.

3. Under what conditions would uptake of antidepressants lead to a decrease in the use of alcohol? In particular, using the first order conditions and notation from above, under what conditions does it hold that $a^{\star\star} < a^\star$. To investigate, we consider the first order condition when the individual uses anti-depressants $d^\star = 1$ but we hold alcohol use at the optimal level when the individual is not using anti-depressants $a^\star$. We write:

$$H(a^\star, d^\star = 1) \equiv u_a(a^\star) - S_a(a^\star, d^\star = 1) + \beta F_a(a^\star, d^\star = 1) \tag{7}$$

This expression captures the marginal change in lifetime utility when $d^\star = 1$, starting from optimal alcohol use when $d^\star = 0$ ($a^\star$). If it is negative, it is optimal to use less alcohol. If it is positive, it is optimal to use more alcohol. Thus, we want to know when $H(a^\star, d^\star = 1) < 0$. Using equation (2),

we can write:

$$H(a^\star, d^\star = 1) < H(a^\star, d^\star = 0) = u_a(a^\star) - S_a(a^\star, d^\star = 0) + \beta F_a(a^\star, d^\star = 0) = 0 \tag{8}$$

Next, we note that u_a and F_a are independent of d, which reduces the condition to:

$$-S_a(a^\star, d^\star = 1) < -S_a(a^\star, d^\star = 0) \tag{9}$$

or

$$S_a(a^\star, d^\star = 1) > S_a(a^\star, d^\star = 0) \tag{10}$$

Recall, S is decreasing and convex. Thus, if it is larger when $d^\star = 1$, it means that it is less steeply negative (flatter) when people use antidepressants compared to when they do not. Put another way, the inequality means that a marginal increase in alcohol leads to a smaller marginal decrease in symptoms when the individual uses antidepressants. In summary, a necessary condition for optimal alcohol use to decline with the uptake of antidepressants is that alcohol use is less effective at reducing symptoms when individuals are on antidepressants versus when they are not.

Combining the three points made above, we argue that we should observe reductions in alcohol use among people who are depressed and who go from not taking antidepressants to taking antidepressants.

3 Empirical Evidence

3.1 The Framingham Heart Study

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 consumption, antidepressant medication use, and mental health from more than 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 received detailed health examinations at 2–4 year intervals and have made significant contributions to the understanding of cardiovascular disease.[4]

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. No sampling ethnicity or residency restrictions were imposed for the Offspring Cohort and their spouses. Moreover, information on these characteristics are not available. As the FHS was not meant to be representative of any larger population, we restrict our final estimation sample to 2,986

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

individuals for whom we have consistent information.[5] To enter our sample, an individual must have completed exam three and they must have complete information on antidepressants and alcohol in all subsequent exams that they complete. Individuals may leave the sample through either death or attrition. Given our data availability requirements, in practice we focus on exams three through nine. At exam three, which was conducted between 1983 and 1987, Offspring Cohort participants took the Center for Epidemiological Studies - 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). Higher scores indicate worse mental health. The clinically verified threshold for depression is any score at or above 16. Exam three was also the first time that the FHS measured antidepressant usage. Importantly, exam three was completely prior to the introduction of SSRI antidepressants. We use information at exam three and alcohol information at exam one to describe a baseline period prior to the introduction of a new technology.

Table 1 presents summary statistics of the Offspring Cohort at our initial exam (exam three) by gender and CES-D score. Of the 1,451 men in our sample, the mean exam three CES-D score is 7.697; for the 1,535 women, this mean is 9.264, indicating worse baseline mental health for women. For each gender, Table 1 presents sample averages of different variables for the first (best mental health) and fourth (worst mental health) quartile of exam three CES-D score, as well as for those at or above the CES-D threshold of 16. At exam three, antidepressant usage is rare for both men and women but it is increasing in CES-D severity; the fraction of individuals ever observed (in exams three through nine) to take antidepressants ranges from 8.1%, for the first quartile men, to 41.1% for the clinically depressed women. The FHS asks respondents the number of 12oz beers, 5oz glasses of wine, and 1.5oz spirits drinks they typically consume per week. We construct extensive and intensive margin consumption measures of each type of alcoholic drink, as well as an aggregate measure of the sum of all drinks per week. On the extensive margin, the share of individuals who drink is *lower* for those with CES-D defined depression for both men and women. Of men in the first quartile of CES-D score, 81.4% consume alcohol at exam three versus only 73.5% for men with CES-D scores at or above 16. For women, this difference is 64% relative to 62.1%. However, conditional on drinking, those with baseline CES-D depression at exam three drink more at exam three. The number of drinks per week is roughly 1.0 to 1.3 drinks more for those with depression relative to those in the lowest quartile of the CES-D score for both men and women. We document a similar pattern for alcohol consumption in 1971, at roughly the instance of exam one, by exam three depression, demonstrating the dynamic and long-run nature of these behaviors and outcomes.

Figure 1 presents the evolution of antidepressant and alcohol behaviors by gender over the subsequent six exams following exam three.[6] Panel a of Figure 1 presents these trends for men. Antidepressant usage increases from 1% in 1985 to 11% by 2011. During this period, the share of the male sample who claim

[5]Similar samples from the FHS Offspring cohort are used by Kaestner et al. (2014) and Darden (2017).

[6]Each FHS exam occurred within a roughly four year window. For ease of presentation, and because the data do not have the exact exam date, we present trends where exam information occurs at the midpoint of the year range.

to drink any alcohol falls from 78.6% to 71.6%. Conditional on drinking alcohol, the number of drinks falls from over 12 drinks/week to roughly 9.5 drinks/week. The trends for women in panel b indicate antidepressant usage increases more significantly, from 2.6% to 23.6%. However, the share of female drinkers over this period is relatively flat, and conditional on drinking, the number of drinks per week falls from 6.4 to 5.4.

Evidence from Table 1 and Figure 1 provides suggestive evidence of the rational self-medication hypothesis. For both men and women, conditional on drinking, those with depression (as measured by CES-D), drink more; over the period in which the technology of antidepressants improves, antidepressant usage increases dramatically, particularly for women, while alcohol consumption falls for both sexes. In what follows, we use this variation to quantify the magnitude of substitution.

3.2 Econometrics

As we document above, antidepressant usage increased dramatically following the introduction of SSRIs. To test the hypothesis that consumption of alcohol should decline following an improvement in the choice set of treatment options, we begin by modeling alcohol consumption directly as a function of antidepressant usage. Specifically, we estimate:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \gamma d_{it} + \theta_t + \epsilon_{it}, \tag{11}$$

where y_{it} is alcohol variable y for person i in exam t, μ_i represents an individual specific effect, x_{it} are time-varying individual characteristics, θ_t are exam fixed effects, 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. The identification argument in Equation 11 such that γ may take a causal interpretation is that there is no *time-varying* unobserved heterogeneity that affects both the decision to take antidepressants and alcohol behavior. While the Framingham data are rich with time-varying observable health information (e.g., cardiovascular biomarkers, see Darden (2017)), we choose not to include this individual-level heterogeneity in X_{it} because they are endogenous with respect to the behaviors we study. Instead, in Equation 11, X_{it} captures age variation in alcohol consumption via five-year age binary variables. We view Equation 11 as the reduced-form of a model in which both alcohol and antidepressants affect both physical and mental health. To draw a connection with our theory of rational self-medication, we contrast estimates of γ from our entire sample with estimates of γ from sub-sample most likely to be self-medicating depression with alcohol *prior* to the introduction of SSRIs.

Table 2 presents estimates of γ, the parameter on antidepressant use, for a variety of alcohol measures, including total alcohol, beer, wine, and spirits consumption, on both the extensive and intensive margins. Standard errors are clustered at the individual level. For the extensive margin of alcohol, we estimate linear probability models; for the intensive margin, we estimate the log of standardized alcoholic units per week. For overall alcohol and for each alcohol type, we estimate Equation 11 separately by gender for the full sample and the subsample of respondents that were clinically depressed at the time of exam three

(see columns 4 and 8 of Table 1). Focusing first on the full sample results, for men, an antidepressant is associated with a statistically significant reduction on the extensive margin of alcohol consumption. At the mean in Table 1, the 7.5 percentage point (pp) effect equates to a 9.5% reduction in the fraction of drinkers. This effect is primarily driven by quits in beer (16.1%) and wine (15.2%). For women on the extensive margin, the effects are similar. An antidepressant is associated with 4.7 pp reduction in ever drinking, or a 7.24% reduction at the mean. For men, an antidepressant is associated with little significant (economic or statistical) change in the number of drinks per week. For women, we find some evidence of a reduction on the intensive margin—an antidepressant is associated with a 25.8% reduction in beers/week—however, the coefficients on wine and spirits consumption are positive and not statistically significant. In the subsample of respondents with clinical depression prior to SSRIs (i.e., exam three), for both men and women, our results on the extensive margin are significantly larger. For this group of men, an antidepressant is associated with a 16.4 pp reduction in drinking on the extensive margin, which is primarily driven by quitting beer (21 pp). Similarly, for women, the extensive margin result grows to a 7pp reduction in any drinking. On the other hand, the intensive margin results become less negative and in the case of total alcohol consumption for men, become positive (although not statistically significant). For women, the intensive margin results are suggestive of substitution away from beer (-61.9%) and towards wine (16.8%).[7]

The direction in which the results move for the subsample with baseline clinical depression provides evidence in favor of rational self-medication: when antidepressants improve, alcohol declines, and the decline in alcohol consumption is driven by the 11.2% of men and 18.2% of women with the most severe levels of baseline depression. One interpretation of these findings is that antidepressant usage obviated the need for alcohol in the management of depressive symptoms. However, a concern with Equation 11 is that time-varying unobserved factors may be correlated with both alcohol and antidepressant behavior. For example, a shock to mental health that causes both alcohol consumption and antidepressant usage would invalidate a causal interpretation of γ. While we cannot directly test for these types of shocks, we can explore the sensitivity of our estimates of γ to the inclusion of separate trends for relevant time-invariant observed heterogeneity. For example, time-varying unobserved heterogeneity in the group of individuals who *ever* are observed to use antidepressants may generate differential trends in alcohol consumption. By controlling for these trends, our estimates of γ may change. Equation 12 demonstrates this estimator, where the τ parameters capture exam-specific deviations from trend for the group of individuals who are observed to use an antidepressant at least once during the seven exams of observation:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \gamma d_{it} + \theta_t + 1\{\text{Ever Uses Antidepressants}_i\}\tau_t + \epsilon_{it}. \quad (12)$$

[7]We have re-estimated our main results from Table 2, while allowing the impact of an antidepressant on alcohol consumption to vary by both baseline depression and heavy alcohol consumption. From this exercise, we conclude that mental health is important in the effect of antidepressants on alcohol consumption, particularly for men. For women, the largest contributor of the effect of antidepressants on alcohol consumption is coming from women without depression but with a history of heavy drinking. Estimates are available upon request.

Our interest is in how the estimates of γ change with the inclusion of controls for differential trends. Table 3 presents estimates of γ in a similar fashion to our main results in Table 2. Generally, on the extensive margin, the inclusion of separate trends does not change our main results—antidepressants are associated with significant reductions in drinking on the extensive margin, especially in the baseline depression subsample. On the intensive margin, our results are qualitatively similar for both men and women relative to Table 2—antidepressants may decrease or increase the intensity of alcohol consumption, but these results are generally not statistically significant, with the exception of substitution for women away from beer.

To complement estimates of γ above, we estimate a difference-in-differences model in which we regress measures of alcohol on the extensive and intensive margins on FHS exam binary variables, where we allow trends in consumption to vary by exam three CES-D score. 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 who are more likely to use SSRIs. Because the effects in Table 2 are concentrated in those with depression, we now focus on how their trends deviate from the trends of those without depression. Formally, we estimate:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \theta_t + 1\{\text{Exam 3 CES-D}_i \geq 16\}\nu_t + \epsilon_{it}, \tag{13}$$

where y_{it} is alcohol variable y for person i in exam t, μ_i represents an individual specific effect, x_{it} are time-varying individual characteristics (i.e., five-year age bins), θ_t is an exam fixed effect, and ϵ_{it} is a standard error component. Here, we are interested in the ν parameters, which allow the trend in consumption to differ for those with a CES-D score above the clinical definition of depression. Recall the exam three score is a baseline metric of depression, prior to the improved technology. Deviations from the trend in alcohol consumption of those without baseline depression after exam three would provide suggestive evidence that SSRIs generated changes in alcohol consumption for those in worse mental health. We estimate Equation 13 on data from exams three through nine, where exam four represents the first exam taken after the introduction of SSRIs and trends are relative to exam three.

The top panel of Table 4 presents estimates of the ν parameters from Equation 13 on the extensive margin of any alcohol, beer, wine, and spirits, where we estimate a separate linear probability model for each variable. We find suggestive evidence that alcohol consumption decreases on the extensive margin for those with CES depression. For example, by exam eight, the incidence of any alcohol consumption among men with baseline depression declines by 12.7pp, roughly 17%, relative to those with better baseline CES depression scores. For both men and women, the estimates of ν are negative but only marginally statistically significant. The bottom panel presents estimates of ν for the natural log of each alcohol variable on the intensive margin. Again, the table presents suggestive evidence of declines in the amount of drinks per week conditional on drinking, but the results are imprecise.

Putting together results from Figure 1 and Tables 1–4, we find that those with depression prior to

the introduction of SSRIs (i.e., in exam three) have lower rates of alcohol use on the extensive margin and larger consumption of alcohol conditional on drinking, both contemporaneously at exam three and historically at exam one. These individuals are much more likely to eventually use antidepressants and the use of antidepressants is robustly associated with alcohol cessation. We interpret these results as broadly consistent with the rational self-medication hypothesis that safer technology may induce some people to substitute towards less harmful substances.

While the empirical results provide evidence of rational self-medication, we acknowledge three main issues that motivate the need for further research. First, even with forty years of longitudinal data on alcohol and antidepressant consumption, FHS lacks a consistently measured metric of mental health. Our preferred estimator would be a dynamic structural model in which both general health and mental health evolve each period as a function of alcohol and antidepressant behavior. However, because mental health is irregularly measured, we focus on reduced-form results generated from Equations 11–13, which demonstrate important heterogeneity by baseline depression. 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, 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 Food and Drug Administration's prescription 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, so it is unlikely that doctor recommendations drive the substitution patterns we identify. Ultimately, we view these results as suggestive evidence that rational self-medication has a role in the discussion surrounding the drivers of risky health behaviors.

4 Conclusion

We develop a theory that suggests how an indirect impact of medical innovation can be a reduction in incentives to rationally self-medicate with potentially addictive substances. We show that alcohol consumption decreased in the Framingham Heart Study following the introduction of SSRIs, particularly for those with CES-D depression. Our results are increasing in pre-SSRI depression, particularly on the extensive margin of alcohol consumption and in men.

Our theory has implications for health policy. 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 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."[8] Our work suggests such restrictions could backfire if individuals substitute towards more dangerous substances. Furthermore, our model demonstrates a parsimonious way to understand the behavioral implications of medical innovation.

We also speculate that our framework could provide insights into rational addiction. In the seminal paper on rational addiction, Becker & Murphy (1988) provide a theory of continued use of addictive substances despite long-run costs. However, the model is silent on why individuals would ever (rationally) commence a path towards addiction in the first place. A potential explanation is self-medication. People may begin using substances to relieve symptoms of illness, partially or perhaps even fully aware that their behavior could potentially lead to a Becker-Murphy type of addictive spiral. Notice that this idea goes against the characterization of the dramatic increase in mortality rates through addiction of white non-Hispanic men since 1998 as "Deaths of Despair" (Case & Deaton, 2015). Whereas "despair" technically suggests a lack of hope, self-medication suggests the opposite: problematically heavy alcohol use or addiction may reflect an earlier, rational, and hopeful attempt to medicate away pain.[9] If so, an appropriate policy response to people who use risky substances to self-medicate would be to develop treatments that are less addictive so that people can rationally substitute away from substances that may have negative long-term consequences.

Moreover, this insight could be extended to contexts beyond self-medication, including potentially addictive substances prescribed by doctors. Indeed, the opioid epidemic began as an attempt to prescribe a potentially addictive substance, Oxycontin, to manage pain. While thousands of individuals experienced struggles with addiction, an often overlooked point is that the vast majority did not. For example, Vowles et al. (2015) estimate that between 8–12% of prescription Oxycontin users were addicted. This proportion is of course deeply concerning, especially since the potential for addiction was not made clear to—indeed, was actively hidden from—doctors and patients. Never-the-less, it is not particularly difficult to imagine a fully-informed, rational, and forward-looking individual in severe chronic pain choosing to take this gamble, i.e., using a potentially addictive substance that can alleviate pain when doing so does not lead to dire consequences nine out of ten times. Whether models of paths to addiction based upon this insight could explain observed usage patterns and help in the evaluation of potential policies that might help to prevent addiction is a project we leave for future work. Factors such as education, work arrangements and other socio-demographic characteristics surely predict variation in how people approach this decision, which future research could also explore.

[8] See https://www.emro.who.int/mnh/news/alcohol-does-not-protect-against-covid-19-and-its-access-should-be-restricted-during-lock-down.html.

[9] 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.

References

Becker, Gary, & Murphy, Kevin. 1988. A Theory of Rational Addiction. Journal of Political Economy, **96**(4), 675–700.

Case, Anne, & Deaton, Angus. 2015. Rising Morbidity and Mortality in Midlife Among White Non-Hispanic Americans in the 21st Century. Proceedings of the National Academy of Sciences, **112**(49), 15078—-15083.

Clay, James M., & Parker, Matthew O. 2020. Alcohol use and misuse during the COVID-19 pandemic: a potential public health crisis? The Lancet, **5**.

Darden, Michael. 2017. A Dynamic Stochastic Model of Lifetime Smoking Behavior. Journal of Political Economy, **125**(4), 1465–1522.

Darden, Michael, Gilleskie, Donna, & Strumpf, Koleman. 2018. Smoking and Mortality: New Evidence from a Long Panel. International Economic Review, **59**(3), 1571–1619.

Dart, Richard C., Surratt, Hilary L., Cicero, Theodore J., & et al. 2015. Trends in Opioid Analgesic Abuse and Mortality in the United States. New England Journal of Medicine, **372**.

Grossman, Michael. 1972. On the Concept of Health Capital and the Demand for Health. Journal of Political Economy, **80**(2), 223–255.

Grossmann, Volker, & Strulik, Holger. 2021. Illicit drugs and the decline of the middle class. Journal of Economic Behavior and Organization, **183**, 718–743.

Hillhouse, Todd, & Porter, Joseph. 2015. A Brief History of the Development of Antidepressant Drugs: From Monoamines to Glutamate. Experimental Clinical Psychopharmacology, **23**(1), 1–21.

Kaestner, Robert, Darden, Michael, & Lakdawalla, Darius. 2014. Are Investments in Disease Prevention Complements? The Case of Statins and Health Behaviors. Journal of Health Economics, **36**, 151–163.

Khantzian, Edward. 1985. The Self-Medication Hypothesis of Addictive Disorders: Focus on Heroin and Cocaine Dependence. The American Journal of Psychiatry, **142**(11), 1259–1264.

Mahmood, SS., Levy, D., Vasan, RS., & TJ., Wang. 2014. The Framingham Heart Study and the Epidemiology of Cardiovascular Disease: a Historical Perspective. Lancet, **383**(9921), 999–1008.

Papageorge, Nicholas W. 2016. Why Medical Innovation is Valuable: Health, Human Capital, and the Labor Market. Quantitative Economics, **7**(3), 671–725.

Powell, David, Pacula, Rosalie Liccardo, & Jacobson, Mireille. 2018. Do Medical Marijuana Laws Reduce Addictions and Deaths Related to Pain Killers? Journal of Health Economics, **58**, 29–42.

Radoff, Lenore Sawyer. 1977. The CES-D Scale: A Self-Report Depression Scale for Research in the General Population. Applied Psychological Measurement, **1**(3), 385–401.

Twombly, Eric C, & Holtz, Kristen D. 2008. Teens and the Misuse of Prescription Drugs: Evidence-Based Recommendations to Curb a Growing Societal Problem. The Journal of Primary Prevention, **29**(6), 503–516.

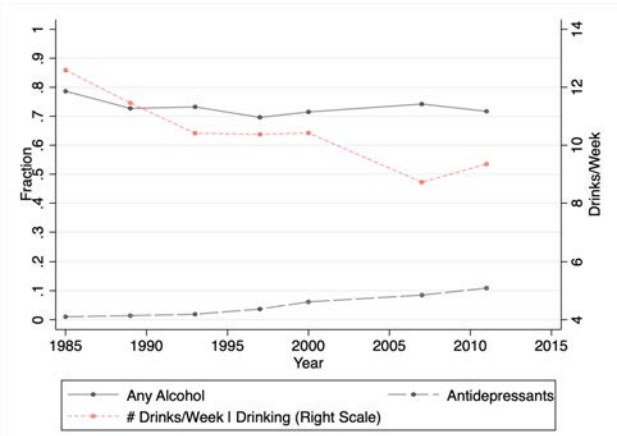
Vowles, Kevin E, McEntee, Mindy L, Julnes, Peter Siyahhan, Frohe, Tessa, Ney, John P, & Van Der Goes, David N. 2015. Rates of Opioid Misuse, Abuse, and Addiction in Chronic Pain: A Systematic Review and Data Synthesis. Pain, **156**(4), 569–576.

Weathermon, Ron, & Crabb, David. 1999. Alcohol and Medication Interactions. Alcohol Research and Health, **23**.

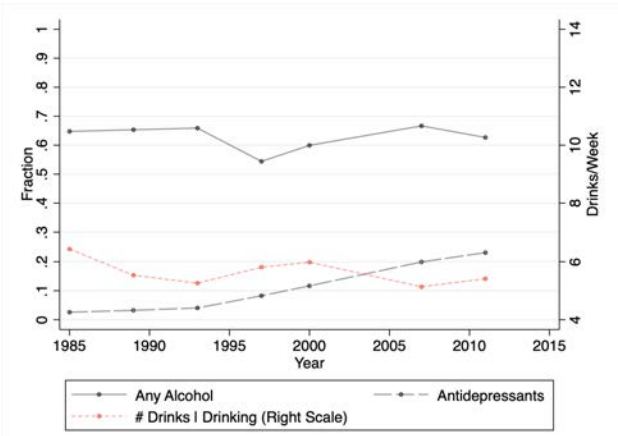
A Tables and Figures

Figure 1: Antidepressant and Alcohol Consumption Over Time.

a. Men



b. Women



Notes: Each figure presents the trends in antidepressant usage and alcohol consumption. Antidepressant usage and the extensive margin of alcohol consumption are relative to the left axis. Conditional on any alcohol consumption, the right axis captures the intensive margin of alcohol as standardized alcoholic drinks per week. The number of individual/exam observations are 8,053 and 8,983 for men and women, respectively.

Table 1: Baseline Characteristics by Gender and Baseline Depression

	Men, n=1,451				Women, n=1,535			
	Full Sample	CES-D 1st Quartile	CES-D 4th Quartile	CES-D ≥16	Full Sample	CES-D 1st Quartile	CES-D 4th Quartile	CES-D ≥16
CES-D Score	7.697	1.436	18.484	23.352	9.264	1.415	21.474	23.579
	(7.224)	(1.186)	(7.499)	(7.545)	(8.359)	(1.192)	(7.274)	(7.010)
Antidepressants	0.010	0.002	0.029	0.037	0.026	0.019	0.041	0.050
Ever Antidepressants	0.125	0.081	0.190	0.198	0.258	0.181	0.377	0.411
Exam 3-9								
Extensive Margin of Alcohol								
Any Alcohol	0.786	0.814	0.752	0.735	0.648	0.640	0.606	0.621
Beer	0.572	0.570	0.565	0.549	0.147	0.119	0.132	0.146
Wine	0.409	0.452	0.374	0.370	0.479	0.508	0.405	0.400
Spirits	0.436	0.441	0.413	0.389	0.369	0.353	0.380	0.400
Any Alcohol in 1971	0.890	0.908	0.881	0.858	0.837	0.821	0.826	0.821
Drinks per Week | Drinking								
Total Units	12.589	12.038	13.738	13.353	6.440	6.291	7.205	7.270
	(12.708)	(12.415)	(15.001)	(15.294)	(6.626)	(6.212)	(8.469)	(8.029)
12 Oz. Beers	9.312	8.823	10.011	10.292	4.310	4.140	5.271	5.732
	(11.195)	(11.023)	(12.770)	(12.785)	(5.660)	(5.632)	(7.704)	(8.222)
5 Oz. Wines	3.886	3.903	4.638	3.667	3.916	3.793	4.170	4.196
	(4.507)	(4.324)	(6.351)	(4.990)	(4.264)	(4.065)	(4.417)	(3.973)
1.5 Oz. Spirits	6.847	6.806	7.117	7.190	4.496	4.534	5.210	5.000
	(8.924)	(8.282)	(10.353)	(10.695)	(6.148)	(5.845)	(8.384)	(7.698)
Total 1971 Units	12.019	12.570	11.883	12.331	5.068	4.645	5.357	5.470
	(12.358)	(13.077)	(12.321)	(13.768)	(5.672)	(4.680)	(6.458)	(6.700)
Age/100	0.488	0.494	0.479	0.473	0.477	0.478	0.467	0.465
Education								
< High School	0.074	0.068	0.097	0.080	0.053	0.029	0.083	0.079
High School	0.269	0.230	0.310	0.333	0.328	0.274	0.386	0.393
Some College	0.373	0.377	0.371	0.340	0.418	0.468	0.358	0.361
College or More	0.171	0.200	0.145	0.167	0.087	0.107	0.058	0.054
Missing	0.113	0.125	0.077	0.08	0.115	0.122	0.116	0.114
n	1,451	456	310	162	1,535	419	363	280

Notes: With the exception of alcohol consumption in 1971, statistics are calculated from exam three, which took place between 1983 and 1987. The sample is constructed such that an individual must be present for exam three, after which an individual may leave the sample through death or attrition. The row for ever antidepressant usage reflects whether the person is ever observed to take an antidepressant through 2011. Depression is measured by the CES-D scale, which is broken into gender-specific quartiles in which the lowest quartile represents the best mental health. A CES-D score greater than 15 indicates clinical depression. Alcoholic drinks are measured per week.

Table 2: Main Estimates

	Total Alcohol		Beer		Wine		Spirits	
	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$
				Men				
Extensive Margin	-0.075	-0.164	-0.092	-0.210	-0.062	-0.070	-0.037	-0.061
	(0.031)	(0.069)	(0.030)	(0.076)	(0.027)	(0.055)	(0.026)	(0.052)
	[-0.135,-0.015]	[-0.301,-0.026]	[-0.152,-0.033]	[-0.360,-0.060]	[-0.115,-0.009]	[-0.177,0.038]	[-0.087,0.013]	[-0.164,0.041]
Intensive Margin	-0.040	0.087	-0.020	0.101	-0.050	0.108	-0.080	0.400
	(0.064)	(0.167)	(0.080)	(0.181)	(0.102)	(0.274)	(0.088)	(0.371)
	[-0.165,0.085]	[-0.244,0.418]	[-0.177,0.137]	[-0.257,0.460]	[-0.251,0.150]	[-0.436,0.653]	[-0.254,0.093]	[-0.336,1.137]
				Women				
Extensive Margin	-0.047	-0.070	-0.026	-0.066	-0.032	-0.011	-0.006	-0.012
	(0.019)	(0.035)	(0.015)	(0.027)	(0.019)	(0.038)	(0.017)	(0.032)
	[-0.084,-0.010]	[-0.301,-0.026]	[-0.054,0.003]	[-0.360,-0.060]	[-0.069,0.005]	[-0.177,0.038]	[-0.040,0.028]	[-0.164,0.041]
Intensive Margin	-0.041	-0.009	-0.258	-0.619	0.034	0.168	0.008	0.030
	(0.051)	(0.108)	(0.100)	(0.184)	(0.060)	(0.129)	(0.087)	(0.155)
	[-0.141,0.058]	[-0.222,0.204]	[-0.455,-0.062]	[-0.984,-0.253]	[-0.084,0.152]	[-0.086,0.422]	[-0.164,0.180]	[-0.277,0.337]

Notes: For each estimate of γ, we present the coefficient estimate, standard error, and 95% confidence interval. Estimates on the extensive margin come from linear probability models of any consumption of the relevant type of alcoholic beverage. Estimates on the intensive margin are of the natural log of the number of drinks of that beverage per week conditional on consuming positive drinks per week of that beverage. All models include individual fixed effects and controls for five-year age bins. The number of individual/exam observations in the full sample is 8,053 and 8,983 for men and women, respectively. In the subsample of those with exam three CES-D score at or greater than 16, the sample sizes are 868 and 1,628, respectively. All standard errors are clustered at the individual level. We follow the American Economic Association's guideline not to report statistical significance stars.

Table 3: Robustness: Estimates with Separate Trends

	Total Alcohol		Beer		Wine		Spirits	
	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$	Full Sample	$CES \geq 16$
				Men				
Extensive Margin	-0.088	-0.200	-0.093	-0.158	-0.049	-0.062	-0.030	-0.048
	(0.030)	(0.072)	(0.028)	(0.071)	(0.029)	(0.074)	(0.028)	(0.037)
	[-0.147,-0.030]	[-0.341,-0.059]	[-0.149,-0.038]	[-0.297,-0.018]	[-0.107,0.008]	[-0.208,0.084]	[-0.084,0.025]	[-0.121,0.025]
Intensive Margin	-0.047	0.001	-0.020	0.080	-0.062	-0.007	-0.100	0.077
	(0.078)	(0.158)	(0.079)	(0.205)	(0.112)	(0.337)	(0.095)	(0.258)
	[-0.200,0.105]	[-0.312,0.314]	[-0.174,0.134]	[-0.326,0.486]	[-0.282,0.159]	[-0.676,0.662]	[-0.287,0.087]	[-0.435,0.589]
				Women				
Extensive Margin	-0.042	-0.048	-0.022	-0.043	-0.023	0.015	-0.004	0.011
	(0.021)	(0.039)	(0.015)	(0.026)	(0.020)	(0.041)	(0.019)	(0.033)
	[-0.083,-0.000]	[-0.126,0.029]	[-0.053,0.008]	[-0.094,0.007]	[-0.063,0.018]	[-0.066,0.095]	[-0.041,0.033]	[-0.053,0.075]
Intensive Margin	0.074	0.093	-0.219	-0.603	0.114	0.207	0.064	0.150
	(0.052)	(0.117)	(0.105)	(0.205)	(0.065)	(0.156)	(0.099)	(0.201)
	[-0.028,0.176]	[-0.138,0.324]	[-0.425,-0.013]	[-1.011,-0.195]	[-0.013,0.240]	[-0.100,0.514]	[-0.131,0.259]	[-0.247,0.548]

Notes: For each estimate of γ, we present the coefficient estimate, standard error, and 95% confidence interval. Estimates on the extensive margin come from linear probability models of any consumption of the relevant type of alcoholic beverage. Estimate on the intensive margin are of the natural log of the number of drinks of that beverage per week conditional on consuming positive drinks per week of that beverage. All models include individual fixed effects and controls for five-year age bins. The number of individual/exam observations in the full sample is 8,053 and 8,983 for men and women, respectively. In the subsample of those with exam three CES-D score at or greater than 16, the sample sizes are 868 and 1,628, respectively. All standard errors are clustered at the individual level. We follow the American Economic Association's guideline not to report statistical significance stars.

Table 4: Estimates of Trend Deviations

	Extensive Margin							
	Men				Women			
	Alcohol	Beer	Wine	Spirits	Alcohol	Beer	Wine	Spirits
$1[CESD_{t=3} \geq 16]*$								
*1[Exam 4]	-0.015	-0.001	-0.039	-0.020	-0.008	-0.002	0.007	-0.029
	(0.037)	(0.039)	(0.043)	(0.039)	(0.034)	(0.023)	(0.033)	(0.033)
	[-0.087,0.057]	[-0.078,0.075]	[-0.123,0.045]	[-0.097,0.058]	[-0.074,0.058]	[-0.048,0.043]	[-0.058,0.072]	[-0.093,0.035]
*1[Exam 5]	0.005	-0.025	0.016	0.000	-0.023	-0.023	-0.031	-0.021
	(0.037)	(0.044)	(0.043)	(0.040)	(0.036)	(0.026)	(0.036)	(0.035)
	[-0.067,0.077]	[-0.111,0.060]	[-0.069,0.102]	[-0.079,0.079]	[-0.093,0.047]	[-0.075,0.028]	[-0.102,0.040]	[-0.090,0.048]
*1[Exam 6]	-0.035	0.025	-0.049	-0.037	-0.011	0.008	0.050	-0.058
	(0.044)	(0.044)	(0.044)	(0.042)	(0.036)	(0.026)	(0.037)	(0.036)
	[-0.121,0.052]	[-0.062,0.112]	[-0.135,0.038]	[-0.120,0.046]	[-0.081,0.059]	[-0.042,0.059]	[-0.022,0.122]	[-0.128,0.012]
*1[Exam 7]	-0.073	-0.000	-0.056	-0.038	-0.051	0.010	-0.035	-0.017
	(0.046)	(0.050)	(0.048)	(0.043)	(0.035)	(0.028)	(0.036)	(0.037)
	[-0.163,0.017]	[-0.097,0.097]	[-0.150,0.038]	[-0.123,0.047]	[-0.120,0.018]	[-0.044,0.064]	[-0.105,0.036]	[-0.089,0.055]
*1[Exam 8]	-0.127	-0.063	-0.143	-0.022	-0.050	-0.022	-0.036	-0.011
	(0.050)	(0.054)	(0.058)	(0.054)	(0.036)	(0.031)	(0.041)	(0.037)
	[-0.224,-0.029]	[-0.169,0.044]	[-0.256,-0.030]	[-0.128,0.083]	[-0.121,0.021]	[-0.083,0.039]	[-0.116,0.045]	[-0.084,0.062]
*1[Exam 9]	-0.044	-0.031	-0.051	-0.026	-0.071	0.024	-0.044	-0.039
	(0.055)	(0.059)	(0.059)	(0.054)	(0.040)	(0.033)	(0.042)	(0.043)
	[-0.152,0.063]	[-0.147,0.085]	[-0.168,0.065]	[-0.132,0.080]	[-0.150,0.007]	[-0.041,0.090]	[-0.126,0.039]	[-0.122,0.045]
	Intensive Margin							
	Men				Women			
	Alcohol	Beer	Wine	Spirits	Alcohol	Beer	Wine	Spirits
$1[CESD_{t=3} \geq 16]*$								
*1[Exam 4]	0.012	0.035	-0.088	-0.081	-0.047	0.165	-0.074	-0.014
	(0.090)	(0.096)	(0.162)	(0.145)	(0.074)	(0.142)	(0.107)	(0.101)
	[-0.165,0.189]	[-0.154,0.223]	[-0.406,0.231]	[-0.366,0.203]	[-0.192,0.098]	[-0.113,0.443]	[-0.283,0.135]	[-0.212,0.184]
*1[Exam 5]	-0.038	-0.003	-0.001	0.040	-0.148	-0.132	-0.158	0.022
	(0.096)	(0.115)	(0.139)	(0.147)	(0.079)	(0.208)	(0.104)	(0.117)
	[-0.226,0.151]	[-0.228,0.222]	[-0.273,0.272]	[-0.249,0.329]	[-0.303,0.008]	[-0.541,0.277]	[-0.363,0.047]	[-0.207,0.252]
*1[Exam 6]	-0.095	-0.028	-0.070	-0.137	-0.227	-0.517	-0.096	-0.138
	(0.092)	(0.112)	(0.163)	(0.185)	(0.091)	(0.182)	(0.114)	(0.153)
	[-0.276,0.086]	[-0.247,0.191]	[-0.390,0.251]	[-0.501,0.227]	[-0.405,-0.049]	[-0.875,-0.159]	[-0.319,0.127]	[-0.439,0.162]
*1[Exam 7]	-0.133	-0.077	-0.156	0.009	-0.174	-0.200	-0.094	-0.198
	(0.094)	(0.115)	(0.153)	(0.167)	(0.092)	(0.188)	(0.120)	(0.135)
	[-0.319,0.052]	[-0.302,0.148]	[-0.456,0.144]	[-0.319,0.338]	[-0.354,0.006]	[-0.569,0.168]	[-0.328,0.141]	[-0.462,0.067]
*1[Exam 8]	-0.131	0.008	-0.094	-0.193	-0.183	-0.347	-0.167	-0.117
	(0.114)	(0.137)	(0.166)	(0.142)	(0.095)	(0.208)	(0.123)	(0.153)
	[-0.354,0.093]	[-0.260,0.276]	[-0.420,0.232]	[-0.471,0.086]	[-0.370,0.004]	[-0.755,0.061]	[-0.409,0.075]	[-0.417,0.184]
*1[Exam 9]	-0.027	0.132	-0.039	-0.063	-0.099	-0.257	-0.175	0.004
	(0.134)	(0.157)	(0.176)	(0.235)	(0.098)	(0.213)	(0.136)	(0.142)
	[-0.291,0.236]	[-0.176,0.441]	[-0.384,0.306]	[-0.525,0.398]	[-0.291,0.094]	[-0.676,0.161]	[-0.441,0.091]	[-0.274,0.283]

Notes: We present the coefficient estimate, standard error, and 95% confidence interval. Estimates on the extensive margin come from linear probability models of any consumption of the relevant type of alcoholic beverage. Estimate on the intensive margin are of the natural log of the number of drinks of that beverage per week conditional on consuming positive drinks per week of that beverage. All models include individual fixed effects and controls for five-year age bins. The number of individual/exam observations in the full sample is 8,053 and 8,983 for men and women, respectively. In the subsample of those with exam three CES-D score greater than 15, the sample sizes are 868 and 1,628, respectively. All standard errors are clustered at the individual level. We follow the American Economic Association's guideline not to report statistical significance stars.