Rational Self-Medication*

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Abstract

We develop a model of rational self-medication in which individuals use 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, arguably causal, reduction in alcohol consumption when SSRIs became available. Our findings illustrate how the effects of medical innovation operate, in part, through changes in behavior.

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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 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 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 drive 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 costly 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 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] SSRIs were not more effective than earlier antidepressants, but were far less harmful 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 long-run negative consequences) can also provide 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, i.e., we predict a negative cross shadow-price elasticity. 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 assumption

[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.

this implication is that alcohol cannot reduce symptoms that are eliminated by SSRIs.[3]

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.[4] 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, 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. 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 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. 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 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

[3]This means that there is a complementarity between alcohol use and depression symptoms, reminiscent of Becker & Murphy (1988), in which a key assumption driving addiction is a complementarity between the marginal utility of current and future consumption of addictive substances.

[4]A Google Scholar search for research with "self-medication" in the title yields 3,780,000 results.

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 clarify how an improvement in anti-depressant quality could lead to a reduction in alcohol use. The cost (in a lifetime utility sense) of antidepressants declined when SSRIs were introduced. SSRIs were as effective as earlier antidepressants at reducing symptoms of mental illness and improving mental health but had fewer adverse side effects. These cost changes amount to a reduction in the shadow price of antidepressants, so standard economic theory predicts that antidepressant usage would increase. Since antidepressants are effective, their use led 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 diminishes or eliminates this benefit, which should lead to a decline in the use of alcohol.

To formalize this cross-shadow-price elasticity, the model envisions agents as solving a two-period problem, where periods are denoted t and $t+1$. Our model is similar to Becker (2007), who distills the Grossman (1972) model into a two-period framework. An agent enters period t with state variable M_t, which is the stock of mental health and where lower M_t implies 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_t and antidepressant consumption d_t, which includes prices and non-pecuniary costs (e.g., hangovers in the case of alcohol or stigma and side effects in the case of antidepressants). They do not have preferences over mental health *per se*, but instead over symptoms of mental health S_t, which negatively affects period utility denoted $u(\cdot)$ and described in more detail below. Symptoms are a function of current period mental health M_t and choices a_t and 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. In the current period, alcohol improves symptoms, which is the "self-medication" effect, which we distinguish from the direct positive impact drinking can have on period utility. Finally, antidepressants can also reduce symptoms.

Agents are forward-looking and make choices to maximize their lifetime (two-period) utility. Formalizing the key dynamic tradeoffs requires specification of a law of motion for mental health. We assume that the stock of 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 improve long-run mental health. Finally, assume that agents die between periods t and $t+1$ with probability $\rho(a_t, d_t)$. We assume that alcohol and, depending on the state of technology, antidepressant use in period t increase the likelihood that agents do not survive to period $t+1$. Finally, if agents survive to period $t+1$, they receive a discounted payoff denoted $\phi(M_{t+1})$, which is an increasing function of its argument. In other words, agents have an incentive to survive to period-$t+1$ in good mental health.

Given this setup, agents choose a_t and d_t to solve the following two period problem.

$$\max_{a_t, d_t} \Big(u(S_t, a_t, d_t) + \beta(1 - \rho(a_t, d_t))\phi(M_{t+1}) \Big) \tag{3}$$

where we assume that S and d enter negatively and a enters positively into the period utility function $u(\cdot)$. Notice that alcohol can increase current period utility in two distinct ways: by reducing the symptoms of mental health conditions and by directly increasing utility. However, the model is entirely consistent with the idea that drinking is a behavior that is perhaps costly in the future because it reduces survival to period $t+1$ and also has long-run negative impacts on mental health, which reduces the period-$t+1$ payout $\phi(\cdot)$.

We use the model to make the following three points. First, we show conditions under which it is optimal to use antidepressants (i.e., $d^\star = 1$ versus $d^\star = 0$). Second, we characterize optimal alcohol usage. Third, 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 generates the cross shadow-price elasticity whereby technological improvement in available mental health treatments leads to a reduction in the use of inferior treatments, in this case, alcohol as self-medication.

To characterize optimal antidepressant usage, we first 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, where we suppress time subscripts and instead use a M' to indicate one-period-ahead mental health:

$$\begin{aligned} u(S(M, a^*, d=1), a^*, d=1) \quad &+ \quad \beta(1 - \rho(a^*, d=1))\phi(M'(M, a^*, d=1) \geq \\ u(S(M, a^{**}, d=0), a^{**}, d=0) \quad &+ \quad \beta(1 - \rho(a^{**}, d=0))\phi(M'(M, a^{**}, d=0)) \end{aligned} \tag{4}$$

To simplify the subsequent analysis, we make an assumption about period-t utility. Specifically, the costs of medication usage are additively separable from other utility components: $u(S, a, d) = \tilde{u}(S, a) - \delta(d_t)$.[5] Finally, to ease exposition, we remove arguments and instead use a subscript of 1 or 0 to denote the choice of using antidepressants or not, respectively. These terms also depend on alcohol consumption, which our discussion touches upon. The agent uses antidepressants if and only if:

$$\begin{aligned} \tilde{u}_1 - \delta_1 + \beta(1 - \rho_1)\phi_1 \quad &\geq \quad \tilde{u}_0 - \delta_0 + \beta(1 - \rho_0)\phi_0 \\ &\iff \\ (\delta_1 - \delta_0) \quad &\leq \quad (\tilde{u}_1 - \tilde{u}_0) + \beta[(1 - \rho_1)\phi_1 - (1 - \rho_0)\phi_0] \end{aligned} \tag{5}$$

[5] Additive separability implies that the marginal utility of alcohol is unaffected by SSRI usage. The assumption is imposed solely to simplify the exposition of our analysis of optimal SSRI usage. It does not affect key model implications we wish to empirically test.

The final inequality means that agents will choose antidepressants when costs are less than our equal to the benefits. The LHS of the inequality is the relative cost arising from taking antidepressants. The first term on the RHS is the utility gain from taking medication, which is primarily through a reduction in symptoms, but is likewise affected by optimal alcohol consumption, which is discussed below. The second term on the RHS requires some attention since it can be either positive (a net benefit) or negative (a net cost). It is the discounted period-$t+1$ payout weighted by the likelihood of surviving to period $t+1$. This component is weakly positive if and only if:

$$\begin{array}{ccc} (1-\rho_1)\phi_1 & \geq & (1-\rho_0)\phi_0 \\ & \iff & \\ \frac{1-\rho_1}{1-\rho_0} & \geq & \frac{\phi_0}{\phi_1} \end{array} \tag{6}$$

If antidepressants are safe, the likelihood of survival is roughly similar whether or not one takes antidepressants, which means that the inequality on the LHS is close to 1. The RHS is likewise close to 1 since antidepressants are not generally effective at improving underlying long-run mental health. Thus, the sign of the inequality will depend largely on marginal changes to alcohol consumption that accompany changes to antidepressant consumption, which we discuss below. The model suggests that in a setting where antidepressants are fairly safe (and ignoring optimal alcohol consumption that depends on antidepressant use, which we discuss below) the quantity $\beta[(1-\rho_1)\phi_1 - (1-\rho_0)\phi_0]$ is likely to be fairly small in magnitude, which means that the choice to use antidepressants will be driven largely by the current-period utility from symptom reductions and current-period costs (pecuniary and non-pecuniary). Contrast this with a context in which antidepressants are not safe. If so, the likelihood of survival when using antidepressants is lower, which means the fraction on the left is less than 1 and unlikely to be greater than the expression on the RHS of the inequality. In this case, the quantity $[(1-\rho_1)\phi_1 - (1-\rho_0)\phi_0]$ is negative, which means that it enters as an additional cost of antidepressant usage. The intuition is that antidepressant use may not have a substantial impact on long-run health and thus the payoff for reaching period-$t+1$ in good mental health, but it may affect the likelihood of survival until period-$t+1$. This feature generates a key testable implication of the model. Even absent any changes to the effectiveness of antidepressants, a change to the cost—in terms of survival here, but more broadly with respect to general health—should lead to increases in antidepressant usage as a key cost of doing so is eliminated.

Next, we characterize optimal alcohol consumption. Starting with equation (3), the first order condition is (again suppressing time subscripts) is:

$$\frac{\partial u}{\partial a} + \frac{\partial u}{\partial S}\frac{\partial S}{\partial a} = \beta\frac{\partial \rho}{\partial a}\phi(M') - \beta(1-\rho(a,d))\frac{\partial \phi}{\partial M'}\frac{\partial M'}{\partial a} \tag{7}$$

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 two marginal costs. The first cost is a lower likelihood of survival to period-$t+1$. The second cost is worse mental health in period $t+1$, which leads to a lower payment ϕ conditional on surviving to period-$t+1$. The equation summarizes the dynamic tradeoffs an agent faces when selecting whether or not to consume alcohol: doing so can be enjoyable and may also help to manage the symptoms of mental health conditions, but there are dynamic costs, including survival and long-run mental health.

Finally, we use our simple model to assess conditions under which an increase in antidepressant use—which the

model predicts should occur upon the arrival of SSRIs amounting to a lower shadow price of antidepressants—should lead to shifts in the consumption of alcohol. This is a crucial motivation for writing the model: to formalize the potential cross shadow-price elasticity of alcohol and antidepressant technological improvements. As we show, the cross shadow-price elasticity occurs when alcohol is a form of medication—albeit one that is dangerous and costly in the long-run—that users turn to when other options are unavailable.

Returning to equation (7), note that a shift in the quality of antidepressants should have no impact on most terms in the equation, leaving the first order condition intact. That is, there is little evidence that safer antidepressants through the introduction of SSRIs affected how much alcohol directly raises utility or the impact of alcohol on long-run mental health. Moreover, this technology shift should have no impact on the utility of symptoms or on the discount factor. There is evidence that the mixture of alcohol with antidepressants became safer following the introduction of SSRIs, which would suggest a rise in alcohol use. A decline in alcohol use could thus only occur with a reduction the impact of alcohol on symptoms. In other words, we expect alcohol use to decline following the introduction of SSRIs, particularly among users of SSRIs because:

$$\left.\frac{\partial S}{\partial a}\right|_{d^*=1} < \left.\frac{\partial S}{\partial a}\right|_{d^*=0} \tag{8}$$

When might this condition hold? The most intriguing possibility is that antidepressants are effective and can simply lower symptoms towards zero, in which case a potential benefit of alcohol vanishes. In this case, agents choose alcohol based solely on the enjoyment they gain from it and not because it is therapeutic. This interpretation relies on the idea that there is some lower bound on symptoms for depression and that this lower bound is reached with less alcohol (or no alcohol) once agents have chosen to use antidepressants.

To summarize, the model generates two key hypotheses. One, the introduction of SSRIs should lead to higher antidepressant use since it is a safer drug compared to earlier antidepressants. Two, users of SSRIs should exhibit reductions in alcohol consumption since SSRIs eliminate the self-medication benefit of alcohol. Put differently, if self-medication plays no role in agents' choice to use alcohol ($\frac{\partial S}{\partial a} = 0$ for $d^* \in \{0, 1\}$), we should see no cross shadow-price elasticity since agents can use SSRIs and continue to consume alcohol solely for the pleasure of doing so despite its long-term risks.

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.[6]

Participants range from 13 to 62 years of age at the first exam, which reflects the wide age variation in the Original

[6]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.

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 individuals for whom we have consistent information.[7] 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 first 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, present 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.[8] 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 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%

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

[8]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 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 suggests several interesting dynamics. For both men and women, conditional on drinking, those with depression (as measured by CES-D), drink more, primarily through more beer consumption for men. Alcohol consumption falls for both groups over time, but for women, antidepressant usage increases by more than double the rate of increase for men. While the CES-D score was recorded at exams three, six, seven, and nine, we are unable to separately identify a formal mental health production function. As a result, we focus on the baseline depression (exam three) as a source of variation to measure the impacts of antidepressants on alcohol consumption.

3.2 Econometrics

To test the hypothesis that consumption of risky goods 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—recognizing that nearly all of the observed antidepressant medication usage in our data occurs after the introduction of SSRIs. Specifically, we estimate:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \gamma d_{it} + \theta_t + \epsilon_{it}, \tag{9}$$

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 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. For alcohol variables on the extensive margin, Equation 9 takes the form of a linear probability model. On the intensive margin, conditional on positive drinks per week for a given alcohol beverage, we estimate the log number of drinks per week. A concern with Equation 9 is that time-varying unobserved factors may be correlated with both alcohol and antidepressant behavior, a point to which we return below. Standard errors are clustered at the individual level.

Table 2 presents estimates of γ, the parameter on the antidepressant use, for a variety of alcohol measures—total alcohol, beer, wine, and spirits—on both the extensive and intensive margins. These models were estimated by gender for two samples: 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 points (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.

Results from the full sample suggest significant quits from alcohol consumption associated with an antidepressant, however the connection between these reductions and self-medication are vague. Thus, we focus our attention on the group most likely to be self-medicating depression with alcohol prior to the introduction of SSRIs. For each dependent variable, the second column presents estimates of δ on the subsample of individuals with exam three CES-D scores at

or above 16. For both men and women, our results on the extensive margin are significantly larger in this subsample. 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%).

The identification argument in Equation 9 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 we cannot directly test this assumption, we can explore differential trends based on time invariant characteristics. 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. Equation 10 demonstrates this model, 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:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \gamma d_{it} + \theta_t + 1\{\text{Ever Uses Antidepressants}_i\}\tau_t + \epsilon_{it}. \tag{10}$$

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.

Finally, 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 that 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:

$$y_{it} = \mu_i + x_{it}^{'}\alpha + \theta_t + 1\{\text{Exam 3 CES-D}_i \geq 16\}\nu_t + \epsilon_{it}. \tag{11}$$

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 is an exam fixed effect, and ϵ_{it} is an i.i.d. 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 11 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 11 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 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 hypothesis, consistent with rational self-medication, that safer technology leads people to substitute towards less harmful substances.

While the empirical results provide evidence of rational self medication, we consider several issues. 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 9–11, 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.

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.

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 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."[9] Our work suggests such restrictions could backfire if individuals substitute towards more dangerous substances.

Finally, we 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, fully aware that the 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). "Despair" suggests a lack of hope, while self-medication suggests something different: heavy alcohol use or addiction may reflect an earlier, rational, and hopeful attempt to medicate away pain. 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.

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[9] See 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.

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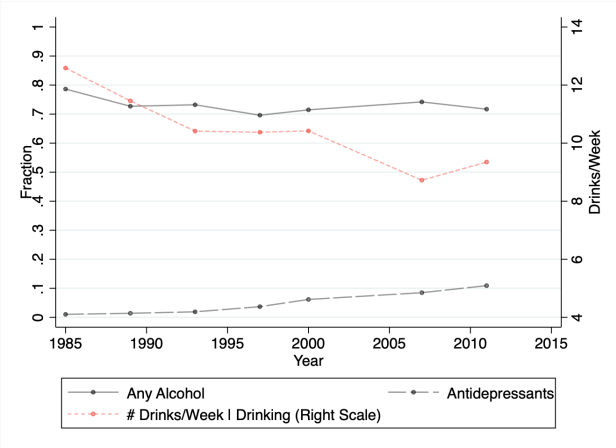
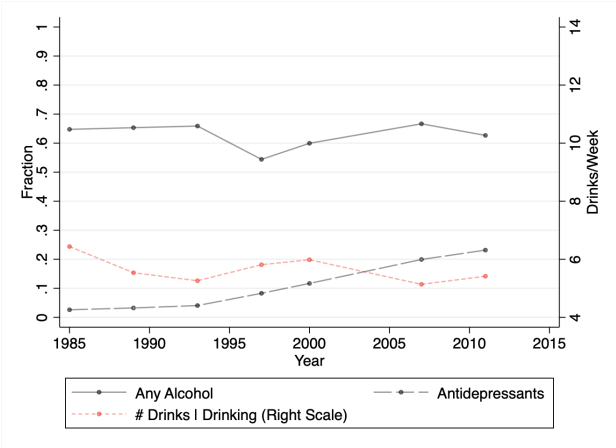
A Tables and Figures

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.

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.

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.