NBER WORKING PAPER SERIES

# THE EFFECT OF SMOKING ON MENTAL HEALTH: EVIDENCE FROM A RANDOMIZED TRIAL

Katherine Meckel
Katherine P. Rittenhouse

Working Paper 29867
http://www.nber.org/papers/w29867

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
March 2022

We are grateful for helpful comments from Julie Cullen, Gordon Dahl, and Dan Sacks. This Manuscript was prepared using Lung Health Study Research Materials obtained from the NHLBI Biologic Specimen and Data Repository Information Coordinating Center and does not necessarily reflect the opinions or views of the LHS 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.

The Effect of Smoking on Mental Health: Evidence from a Randomized Trial
Katherine Meckel and Katherine P. Rittenhouse
NBER Working Paper No. 29867
March 2022
JEL No. I1,I12,I18


## ABSTRACT

This paper aims to identify the causal effects of smoking on mental health using data from the Lung Health Study, a randomized trial of smoking cessation treatment with five years of follow-up interviews. In the short-run, distress increases, likely reflecting the effects of nicotine withdrawal. Long-run effects on mental health are small overall, but mask heterogeneity by gender. For women, the cessation program leads to improved mental health, driven by decreases in insomnia and nervousness. Men do not experience these improvements, due in part to a small increase in severe disturbances.



Katherine Meckel
Department of Economics
Econ 210
University of California at San Diego
9500 Gilman Drive #0508
LaJolla, CA 92093
and NBER
kmeckel@ucsd.edu

Katherine P. Rittenhouse
Department of Economics
Econ 117
University of California San Diego
9500 Gilman Drive
La Jolla, CA 92093-0508
krittenh@ucsd.edu

# 1 Introduction

There is a well-documented correlation between smoking and mental illness. In the U.S., individuals with mental illness are two to three times more likely to be current smokers than individuals without mental illness and consume nearly half of the cigarettes sold nationwide (McClave et al. 2010; Lasser et al. 2000). Despite the strong relationship between smoking and mental health, the underlying causal mechanisms are not well-understood (Fluharty et al. 2016).

Prior work generally advances three hypotheses for the relationship between smoking and mental health: (1) smoking causes a decline in mental health; (2) poor mental health causes take up of smoking; and (3) smoking and mental illness are not causally related but coincide in the population due to third factors such as socioeconomic status or genetics. The first hypothesis is supported by evidence that, in some settings, nicotine may exacerbate symptoms of anxiety and depression. The second hypothesis posits that nicotine, delivered to the bloodstream during the act of smoking, provides *relief* from symptoms of depression and anxiety and is therefore used as a coping mechanism by individuals with untreated mood disorders.[1] Evidence for this theory includes the fact that smokers report using cigarettes to improve their mood as well as the fact that teenagers start smoking following traumatic events (Friedman 2020).[2] Note, however, that smokers who report that smoking improves their mood may be conflating these effects with relief from nicotine withdrawal.

In addition to the effects of nicotine on the nervous system, smoking may affect mental health by causing behavioral changes. For example, cigarettes may serve as a complement to other substances, such as alcohol, which could independently affect mood (Dee 1999).

[1] Nicotine is a psychoactive drug that can act as both a stimulant and depressant (Ashton et al. 1973). While nicotine can relieve the symptoms of depression and anxiety in some settings, in others it acts to exacerbate them (Picciotto, Brunzell, and Caldarone 2002).

[2] For example, participants in the Lung Health Study, which we analyze in this article, were asked whether they smoke to improve their mood. A majority replied "usually" or "always," as opposed to "sometimes" or "never," in response to the following prompts: "I light up a cigarette when I feel angry about something"; "When I get blue or want to take my mind off my cares and worries, I smoke cigarettes"; "When I feel uncomfortable or upset about something, I light up a cigarette".

Smoking may be a social activity, in which case quitting could reduce one's social network. The extensively-documented negative *physical* effects of smoking may also lead to declines in mental health (CDC 2014).

Given the many different ways in which smoking and mental illness may be related, it is crucial to use exogenous variation in smoking to study its effects on mental health. Simple comparisons of mental health between smokers and non-smokers may result in biased estimates due to factors that are correlated with both smoking status and mental state — e.g., a history of mental illness.

In this paper, we analyze data from a randomized controlled trial called the Lung Health Study (LHS). The LHS randomly assigned approximately 6,000 middle-aged smokers to receive an intensive anti-smoking intervention and followed them for the next five years. Random assignment ensures characteristics that are correlated with smoking status, such as baseline mental health, are unrelated to quitting behavior in the treatment group, allowing us to cleanly identify the causal effects of smoking cessation. As part of annual followup interviews, participants were asked several questions about their mental well-being, which we combine into a single "distress scale." These measures, along with data on prescription use of anxiolytics and anti-depressants, have never been studied as outcomes of the treatment. Importantly for a study of smoking and mental health, the long follow-up gives us the opportunity to differentiate between short term effects (i.e. due to nicotine withdrawal) and long term effects of cessation.

Another benefit of the LHS data is that smoking cessation is medically validated, mitigating concerns about measurement error that can arise when variables are self-reported. We find that assignment to the smoking cessation program causes a large increase in quitting, driven by sustained, rather than temporary, quits. The treatment effect on sustaining cessation across all five annual follow-up interviews is 17 percentage points, a 309% increase over the sustained quit rate in the control group.

Turning to mental health outcomes, we find that in the first annual interview, which

is conducted shortly after the end of the cessation program, there is a 13% increase in the distress scale. Reports of severe disturbances, while rare, also increase. These results likely reflect the effects of initial withdrawal from nicotine. To examine the long-run effect of smoking cessation on mental health, we calculate the average of our distress scale and prescription drug indicators over interview years 2 through 5. We find small and statistically insignificant effects of the cessation program on these outcomes.

Although we find little evidence of effects on long-run mental health in the full sample, prior evidence suggests there may be important heterogeneity by gender. Smoking behavior differs across men and women, perhaps due to differing effects of nicotine on the brain across gender. For example, smoking rates among men have historically exceeded those among women (Holford et al. 2014). Medical studies using neuro-imaging reveal that smoking activates male smokers' reward pathways more than those of women (Cosgrove et al. 2014). Other research suggests that this difference in satisfaction from smoking is tied to nicotine specifically, rather than other aspects of smoking. Perkins and Karelitz (2015) find that cigarettes with and without nicotine act similarly to alleviate the effects of withdrawal among female smokers, but only the cigarettes with nicotine provide relief to male smokers. Motivated by these facts, we study effects separately by gender.

Indeed, overall mental health effects mask considerable heterogeneity by gender. For women, assignment to the treatment leads to a 10% decrease in the distress scale, driven by sizable reductions in insomnia and nervousness. These results are consistent with hypothesis (2) above–that is, smoking causes declines in mental health. Conversely, for men, there is a small increase in distress which, although statistically indistinguishable from 0, is statistically distinguishable from the effect for women. In addition, the smoking cessation program leads to a small increase in severe disturbances for men, and possibly an increase in the use of anti-anxiety medication. These results are most consistent with hypothesis (1) above–that is, men may use smoking as self-medication for underlying mental health issues. Given the small and somewhat noisy effect sizes for men, we cannot rule out hypothesis (3), i.e. that

smoking and mental health are not causally related in this group.

Our results provide new evidence on the relationship between smoking and mental health that can be used to inform anti-tobacco policy. The LHS treatment involves a cessation program, making our results most relevant to policies that increase quitting among current smokers. For example, under the Affordable Care Act, health insurance expansions to low-income Americans increased utilization of smoking cessation aids and reduced cigarette consumption (Cotti, Nesson, and Tefft 2019; Maclean and Saloner 2019).[3] Our findings suggest that welfare benefits from such policies include long-run mental health gains, in particular for women. Our findings also suggest that mental health supports and anti-smoking policies may be complementary public health interventions, as anti-smoking efforts alone can have unintended adverse consequences on mental health outcomes, in particular for men.

However, our findings should be generalized to the broader policy context with some caution. The LHS sample is not demographically representative — for example, participants are more likely to be white and college-educated than the true population of smokers. (Courtemanche, Tchernis, and Ukert 2018). Second, participants signed up to be part of a smoking cessation program, implying that they all have some interest in quitting, which is not true of all smokers.[4] Another concern is that the treatment program, which emphasized behavioral and cognitive strategies for quitting, could independently affect mood. While we cannot rule this out, we expect that such effects would dissipate in the years following the program.

The paper proceeds as follows. Section 2 provides background on the Lung Health Study. Section 3 describes our treatment of the data. Section 4 describes our empirical methods. Section 5 presents our results. Section 6 concludes.

[3] Anti-tobacco policy includes a range of measures such as taxes, smoke-free laws, advertising restrictions, and public education campaigns. A recent review concludes that taxes have little effect on adult smoking; that evidence on the effects of bans on cessation is mixed; and that there is relatively little evidence on the effects of advertising and education campaigns (DeCicca, Kenkel, and Lovenheim 2020).

[4] In 2015, 68% of smokers in the U.S. said they wanted to quit (Babb et al. 2017).

## 2 Background

The Lung Health Study (LHS) was a clinical trial which randomly induced smoking cessation among its participants.[5] The study aimed to identify ways of delaying the onset of chronic obstructive pulmonary disease (COPD) among at-risk smokers. In particular, the authors were interested in the efficacy of pairing smoking cessation with regular, long-term use of a prescription inhaler.

Study participants included 5,887 smokers who were identified as at risk for COPD. The recruitment process occurred from October 1986-January 1989 at 10 clinical centers in the U.S. and Canada. To be eligible, participants had to be at risk for COPD, have no other serious illnesses or medical conditions, and have no plans to move away from the clinic area during the study. Note, the study sample is not representative of the general population of smokers. For example, participants are aged 35-65, are almost exclusively white, and live in urban areas (i.e. where the clinical centers were located).

Participants were randomly assigned to one of three equally-sized groups: two treatment arms and one control arm. The treatment groups underwent an intensive smoking cessation program in the first few months of 1989. In addition, one of the treatment groups received a bronchodilator (a prescription inhaler), whereas the other treatment group received a placebo inhaler. All participants were then followed for five years (1990-1994), returning once a year to the clinic for interviews and lung function testing. Participants in the treatment group were instructed to use their inhalers regularly during the five years.

The smoking cessation program combined several elements thought to promote quitting, including: (1) a physician's message regarding current lung impairment and disease risk; (2) a 10 week, 12-session group program emphasizing cognitive and behavioral strategies for cessation; (3) encouragement and support from family members; and (4) provision of nicotine gum for up to 6 months. The group sessions initially focused on quitting and later

[5] O'Hara et al. (1993) provide a detailed description of the LHS. Additional information can be found here: https://clinicaltrials.gov/ct2/show/NCT00000568.

emphasized relapse prevention, stress management, and tapering gum usage.

During the five years following the smoking cessation program, individuals in the treatment group returned to the clinics every 4 months. The purpose of these meetings was to promote inhaler usage and prevent smoking relapse. Extended intervention options were provided to individuals who relapsed, including restart and stay-quit support groups, and LHS physician visits.

Once a year, participants in both the treatment and control groups returned to the clinic for interviews that covered a wide variety of topics, including health outcomes and healthcare utilization over the past year.[6] Individuals also underwent pulmonary testing to validate cessation.[7] All measures of cessation we analyze are therefore medically validated. Attrition is relatively low, as 5,627 individuals (96%) remain in the sample in the final wave.

The smoking cessation program was quite successful: 22.0% of individuals in pooled treatment arms (i.e., with or without the prescription inhaler) quit smoking cigarettes and sustained cessation for all five years, compared to 5.5% of the control arm. Ultimately, however, the prescribed inhaler was found to have little added benefit for lung health relative to the smoking cessation program, and, as a result, many felt the study was a "failure" (Anthonisen et al. 1994).

Perhaps as a consequence of the "failure" of the trial, some of the measures collected by the investigators during the annual follow-up interviews have not yet been analyzed. Our paper constitutes the first analysis of treatment effects on mental health outcomes collected during the follow-up interviews.

[6]The first annual interview took place about 8 months after the initial, 4-month smoking cessation program ended ("annual" references time since randomization). Nicotine gum was then provided for up to 2 months. Questions asked about participants' mental health reference the individual's state of mind over the four months before the annual interview.

[7]Cessation is validated by measuring carbon monoxide in exhaled breaths and cotinine levels in saliva.

### 2.1 Prior Research Using the LHS to Estimate Causal Effects

In this section, we provide a brief overview of studies which use data from the LHS to estimate causal effects of smoking cessation.[8]

As discussed above, assignment to the smoking cessation program increased the likelihood of quitting smoking and improved lung function. These effects persisted five years (Kanner and Group 1996; Anthonisen et al. 1994) and 11 years (Anthonisen, Connett, and Murray 2002) after the initial cessation program. Assignment to the treatment also resulted in fewer respiratory symptoms (Kanner et al. 1999) and lower rates of lower respiratory illness (Kanner, Anthonisen, and Connett 2001) after five years. Anthonisen et al. (2002) and Anthonisen et al. (2005) examine effects on all-cause mortality, finding it is significantly lower in the treatment group 14.5 years after the smoking cessation program.

Several studies examine outcomes that, like mental health, were not a focus of the original study. For example, Murray, Istvan, and Voelker (1996) looks at effects on alcohol use at one year, finding no differences between the treatment and control group, despite substantial differences in smoking. Courtemanche, Tchernis, and Ukert (2018) use the LHS to estimate the relationship between smoking and body mass index, finding that quitting leads to an increase in BMI by 1.8 to 1.9 points, or 11-12 lbs for a male of average height. Lastly, Fletcher and Marksteiner (2017) study spousal health spillovers of smoking cessation, finding that spouses of individuals in the treatment group were also more likely to quit smoking.

Other works using the LHS data are primarily descriptive, reporting on the study's design and implementation or identifying predictors of various outcomes, such as lung health, or smoking cessation. A few additional studies use data from the two treatment arms only to

[8]To identify LHS studies that estimate causal effects of smoking cessation, we reviewed publications from three sources. First is a list of publications on the Study Record page for the LHS published by ClinicalTrials.gov, here: https://clinicaltrials.gov/ct2/show/NCT00000568. These publications were provided by study investigators or added from automatic searches of ClinicalTrials.gov's database. Second, we reviewed publications listed on BioLincc's study page for the LHS https://biolincc.nhlbi.nih.gov/publications/?studies.raw=Lung+Health+Study+%28LHS%29&acronym=LHS&sort=citation&page_size=100. This list includes research conducted by individuals who, like us, obtained LHS data through data use agreements with Biolincc. Third, we conducted a Google Scholar search for the phrases "Lung Health Study" and "causal".

estimate the causal effects of the bronchodilator.

## 3 Data

We first generate several different outcomes describing smoking behavior during the follow-up interviews. The first, "sustained quit," is an indicator equal to one if a participant is a medically validated quitter across all five follow-up interviews. For the second measure, "current quit," we use information on whether an individual has quit at the time of a given follow-up interview. Unlike those with a sustained quit, these individuals may have relapsed in the years since the cessation program, or may have quit sometime after the initial intervention. Specifically, we first create an indicator equal to one for a given individual-year if the the participant has quit smoking at the time of the interview. We then take the average of this indicator over interview years 2-5.[9]

Our third measure of smoking behavior is the average number of cigarettes smoked per day over the 12 months before a given follow-up interview. This outcome is self-reported and may be subject to measurement error. Participants are asked to provide the number of cigarettes smoked per day for each of the previous 12 months separately, and we calculate the mean of these 12 numbers. We then take the average value of this outcome over interview years 2-5.

Each measure of smoking behavior has distinct advantages and disadvantages. The two measures of cessation are medically validated, and thus less prone to measurement error. However, quitting only captures the extensive margin of smoking behavior and is measured at the time of the interview (potentially missing quits or relapses that occur between interviews). The number of cigarettes smoked over the past year instead captures the intensity of smoking. If the smoking cessation program causes some individuals to smoke less (but not quit), and

[9]We omit year 1 from all averages to avoid conflating the short term withdrawal symptoms with longer term mental health effects. Participants were provided with nicotine gum at the start of the smoking cessation program and expected to stop using it after 6 months (i.e., 2 months after the intervention ended). Nicotine withdrawal can impact mood for several months, and may thus have impacted responses at the first annual interview. See: https://www.insider.com/nicotine-withdrawal-symptoms. For completeness, we also show results separately for year 1.

smoking intensity affects mental health, it is important to consider intensive margin as well as extensive margin measures.[10]

Next, we generate several outcomes pertaining to mental health. The LHS asks about the interviewee's mental and physical state in each annual survey, via the following questions: "Indicate the extent to which you have been troubled in the last four months by any of the following. Please indicate Severe, Moderate, Mild, or Not at all." A list of 26 physical and mental conditions is provided (e.g., "Chest Discomfort," "Dry Mouth," "Excessive Salivation," etc.). The mental conditions are as follows: "Irritability," "Insomnia," "Mood Changes," "Nervousness," "Psychological Illness." We construct an overall measure of mental health by assigning each response a number from 0 through 3, where 0 corresponds to an answer of "Not at all" and 3 corresponds to an answer of "Severe", and summing the resulting scores across the five reported mental conditions for each respondent-year to create a "distress scale."[11] An increase in this distress scale indicates worsening mental health, while a decrease indicates an improvement in mental health. We also create a variable to isolate severe distress. Specifically, we first create an indicator variable equal to one for a respondent-year-condition if the interviewee states that they have been severely troubled by that condition, and zero otherwise. Next, for each respondent-year, we take the average of this indicator across the five mental health conditions. For example, if a respondent indicates in a given interview that they suffer from severe insomnia only, they would receive a value of 1/5 for that year.[12] Finally, we again take the average of the distress scale and severity

[10]In their study of the causal effects of smoking on obesity, Courtemanche, Tchernis, and Ukert (2018) use assignment to the cessation program in the LHS to instrument for cigarettes per day and another measure of smoking – exhaled CO. While exhaled CO does not suffer from reporting error because it is clinically measured, it only reflects smoking in the few days before the follow-up interview, so may not be reflective of usual behavior over the prior year (like the measure of cigarettes).

[11]The construction of this variable is similar to other measures of mental health such as the Kessler-6 and Kessler-10 Distress scores. These scores are calculated as the sum across a set of questions about emotional states with a five-level response scale.

[12]We also created an alternative version of this measure in which we first calculate z-scores for each "severe" indicator and then calculate the average of the z-scores across the five mental health conditions in each year (following Kling, Liebman, and Katz 2007). Doing so addresses the issue that some conditions are more likely to be reported as "severe" than others. Our findings using the index measure are highly similar to those using the averaged measure. Results available on request.

measure over interview years 2-5.

Next, we define indicators for prescription medications for mood disorders (anxiolytics and anti-depressants), and take the average of these indicators over years 2-5. At each annual interview, participants are asked to provide information on prescription drugs they have taken over the previous 12 months — first, within 11 medication categories related to lung and heart health and then by listing up to three additional drugs outside of these categories. Participants are instructed to bring in pill bottles or drug containers for medications they are taking for this purpose. The drug names are not cleaned and contain some inaccuracies such as spelling mistakes.

To match the drug names to their therapeutic categories, we performed the following steps. We first Googled each of the 1244 distinct drug names, as written.[13] For 629 of these, the search results in a sidebar Google automatically creates that lists the drug's medication class, which we record.[14] For the remaining 615 drugs for which Google does not produce the automatic description, we use a database of drug name-to-drug class matches that is provided as part of the Medical Expenditure Panel Survey (MEPS) for the years 2002-2016.[15] These two steps allow us to match 97% of the reported drug names to therapeutic categories, from which we can identify anti-depressants and anxiolytics.[16]

Between 5 and 8% of individuals per year do not answer the prescription drug and mental health questions, either due to attrition or failure to answer the question, and are assigned missing values for that year. In this case, we calculate the average for years 2

[13]A Google search is helpful because it autocorrects spelling errors and produces partial matches.

[14]E.g., a Google search for "Venlafaxine," an anti-depressant, produces a sidebar that describes it as a "Nerve pain medication and antidepressant," as shown here: https://www.google.com/search?q=venlafaxine.

[15]Data files are available here under "Prescribed Medication Files": https://meps.ahrq.gov/mepsweb/data_stats/download_data_files.jsp MEPS lists as the source for its therapeutic class assignment "Multum Lexicon variables from Cerner Multum, Inc." Matching for each drug is done by hand, as some modification of the handwritten LHS entry is typically needed to match it to the MEPS name, which may involve translation between generic and brand name, or vice versa.

[16]Specifically, anti-depressants are identified as follows: the Google sidebar description includes the words "ANTIDEPRESSANT" and/or "SELECTIVE SEROTONIN" (in reference to a selective serotonin re-uptake inhibitor), or the therapeutic class codes from the MEPS are as follows: 208, 209, 249 or 76. Anxiolytics are identified as follows: Google sidebar description includes the words: "ANXIOLYTIC", or the therapeutic codes from the MEPS are: 67, or 69.

through 5 using the remaining non-missing values. Our main analysis sample consists of individuals with non-missing values of the year 2 to 5 average distress scale and prescription drug outcomes. This sample includes 5,705 of the 5,887 individuals enrolled in the trial, or 97%. While prior studies using the LHS have established that the full sample (5,887) is balanced on observables, we verify that our main sample (5,705) is also balanced. In Table A1, we compare mean pre-treatment characteristics across the treatment and control groups. These characteristics include age, education, BMI, smoking behavior, and our mental health outcomes, measured during screening interviews.[17] Reassuringly, there is very little difference in means across the two samples and none are statistically significant.

Table A1 also reveals that the average study participant is 48 years of age and that men make up about 2/3 of the study population. On intake, participants smoke about 31 cigarettes (1.5 packs) per day, and started smoking around age 17 or 18. When asked to rate the extent to which irritability, insomnia, mood changes or nervousness have troubled them over the past 4 months from 0 (Not at all) to 3 (Severe), individuals indicate an average of 0.4 to 0.5. The average for psychological problems is approximately 0 (Not at all). Around 1 to 3% of individuals have taken anti-anxiety medications or anti-depressants in the past month.

## 4 Empirical Method

To estimate the effects of the smoking cessation program on smoking behavior and mental health, we estimate the following equation, for all individuals and separately for men and women:

$$Y_i = \alpha + \beta \text{Treatment}_i + \epsilon_i \tag{1}$$

Where $Treatment_i$ is an indicator for whether individual $i$ is assigned to one of the two

[17] During the screening interview, participants were only given the option to indicate "Not at all", "Mild", or "Severe" in response to the questions about mood. In this case, we assign values 0, 1.5, and 3 to these responses, respectively, so the scale is comparable to that in follow-up interviews.

treatment arms. Randomized assignment to the treatment group implies that $Treatment_i$ is independent of the error term, $\epsilon_i$, in which case $\beta$ measures the causal effect of the smoking intervention on outcome $Y_i$. We estimate robust standard errors to adjust for heterogeneity across individuals.

Our aim is to understand how quitting smoking affects mental health. Therefore, it may seem preferable to estimate an instrumental variables (IV) specification for our mental health outcomes. In an IV specification, we would instrument for smoking using assignment to the treatment group. We choose not to present IV estimates as our preferred specification because each of our smoking variables is an imperfect measure of smoking intensity. As discussed above, our measures of quitting are medically validated and thus highly accurate, but only capture the extensive margin of smoking. Our intensive margin measure of smoking — number of cigarettes smoked per day over the past year — is based on recall and likely contains measurement error. Thus, for enhanced transparency and interpretability, we choose to present the reduced form estimates of the effect of the treatment program on our measures of mental health.

Still, in order to compare the effects of quitting smoking on mental health between men and women, it is necessary to scale the reduced form coefficients to the change in smoking for each group. Therefore we also estimate the following two-stage least squares (2SLS) model:

$$\text{Cigarettes}_i = \gamma + \rho \text{Treatment}_i + \nu_i \tag{2}$$

$$M_i = \psi + \phi \text{Cigarettes}_i + \xi_i \tag{3}$$

where the second-stage outcome, $M_i$, is a mental health outcome for individual $i$. The endogenous regressor in Eq. 3, $\text{Cigarettes}_i$, is equal to cigarettes/day in year 1 or averaged over years 2-5, as indicated. The excluded instrument is the treatment group indicator, $\text{Treatment}_i$. We assume that assignment to the treatment group affects long-run mental

health only through its direct effects on smoking. Given this assumption, $\phi$ can be interpreted as the average effect on mental health of smoking an additional cigarette. We estimate the 2SLS model separately for men and women and test whether the resulting estimates of $\phi$ differ.

# 5 Results

## 5.1 Smoking Behavior

First, we estimate treatment effects on smoking outcomes recorded during the follow-up interviews.[18] Table 1 reports estimates of $\beta$ from Eq. 1, along with robust standard errors in parentheses and p-values in brackets. Panel A reports effects for the full sample and Panels B and C report effects for men and women, respectively. At the bottom of the table, we report the p-value from a test of whether the effects for men and women are equal.

In Column (1), we find that the smoking cessation program raises the probability of a sustained quit by 16.9 percentage points. This effect is a 307% increase with respect to the sustained quit rate in the control group of 5.5%. The treatment effect is larger for men than women (337% vs. 262%), implying that men are more responsive to the cessation program.

In Column (2), we report effects on the current quit rate, averaged over years 2 to 5. The treatment effect in the pooled sample is 18.9 percentage points, or 110% of the control mean. The fact that the effect on sustained quitting is nearly as large as the effect on the current quit rate ($16.9/18.9 = 90\%$) implies that the treatment works primarily to increase sustained quits.[19] The treatment effects are similar in magnitude for women and men, implying that women are somewhat more likely to quit temporarily (relapse) in response to the treatment than men.

In Column (3), we report effects on the number of cigarettes per day, averaged over years

[18] These results replicate findings from prior LHS studies that also estimate treatment effects on smoking behavior. See section 2.1 for a review of this prior literature.

[19] Sustained quits are a subset of current quits in a given year. Therefore, the difference between the current and sustained quit rate gives us the share of individuals who are not smoking in that year but relapsed during another year.

2 to 5. Overall, the effect is -8.1 cigarettes, or a 38% decrease relative to the control mean. The treatment effect is larger for men than women (-8.6 vs. -7.0, respectively). Men smoke more cigarettes per day than women (22.5 vs. 19.8), however, so the percentage declines are more similar (39% vs. 35%).

Note that the magnitude of the decrease in cigarettes, 8.1, suggests that the treatment program reduces cigarette consumption among individuals who continue smoking. The average daily cigarette consumption in the control group is 21.5. The treatment program increases the likelihood of quitting by by 19 percentage points, which implies a decrease in daily cigarettes by 19%*21.5 = 4.1 due to quits alone. Therefore, given that the total decline is 8.1, the treatment program likely works to reduce nicotine intake through both extensive (quitting) and intensive (number of cigarettes smoked) margins.

### 5.2 Mental Health Outcomes

In Table 2, we report the results of estimating Eq. 1 on mental health and prescription drug indicators averaged over annual followup interviews in years two to five. As in Table 1, Panel A reports effects for the full sample and Panels B and C report effects for men and women, respectively.

At the bottom of Table 2, we report statistics derived from the 2SLS model (Eqs. 2 and 3). Specifically, we first estimate the 2SLS model separately by gender, setting $M_i$ equal to the outcome indicated in the column heading. We then calculate the difference between the second stage estimates of $\phi$ for men and women ("Diff, Men-Women: b/cigs"). "P-Value" is the p-value from a Chi-squared test of whether the difference in coefficients is equal to 0.[20]

In the full sample (Panel A), effects are small and imprecise, but mask heterogeneity by gender. Treatment effects on the distress scale are positive but insignificant for men and negative and statistically significant for women (Column 1, Panels B and C). Specifically, assignment to the treatment group reduces women's distress scale by 0.25, or 10% of the control mean, an estimate that is statistically significant at the 5% level. The difference

[20] Appendix Table A2 reports the full set of results from these 2SLS regressions.

between the 2SLS estimates for men and women, -0.04 per additional cigarette, is significant at the 5% level.

Figure 1 displays treatment effects on each component score of the distress scale, averaged over years 2 to 5, along with 95% confidence intervals. Appendix Table A3 reports the corresponding coefficients, standard errors and p-values. The effects for men are generally positive, but small in magnitude and statistically insignificant. For women, the smoking cessation program reduces the average insomnia and nervousness scores by 18% and 13% of the control group means, respectively. In addition, the difference between the 2SLS estimates for insomnia and nervousness for men and women are statistically significant at the 5% level.

In Column 2 of Table 2, we estimate effects on the indicator for severe distress, averaged over years 2 to 5. In this case, assignment to the treatment increases the likelihood that men report severe distress by 0.48 percentage points relative to the control group (37% of the control mean). The effect for women is negative but noisy, and the difference between the 2SLS effects for men and women is statistically significant at the 5% level.

In Columns 3 and 4 of Table 2, we estimate effects on the average likelihood individuals take anti-anxiety or anti-depressant drugs in years 2-5. For men, the likelihood of taking an anxi-anxiety drug increases by 0.7 percentage points, or 33%, but this effect is only marginally significant at the 10% level. For women, the coefficients are both negative (suggesting less prescription drug use), but imprecisely estimated, and the difference between the 2SLS effects for men and women is not statistically different from 0.

In sum, we find that assignment to the treatment improves mental health for women, in particular through reductions in insomnia and nervousness. We do not find evidence that these reductions are accomplished through the use of prescription drugs. Men experience different (worse) effects on mental health than women. While we do not find evidence of declines in their overall mental health score, our results suggest an increase in severe disturbances.

Finally, we estimate the short-run effects of assignment to the smoking cessation program on smoking and mental health. Table 3 reports results from estimating Equation 1 on our

outcomes measured during the first annual follow-up survey. As discussed above, the first annual follow-up occurred shortly after the conclusion of the smoking cessation program and therefore these effects may reflect nicotine withdrawal.

At the time of the first annual survey, 26.0% of the treatment sample has quit smoking, compared to 9.3% of the control group. The treatment sample smokes 12.4 fewer cigarettes per day over the past 12 months, or 47.9 percent fewer than the control group mean. The declines in smoking and number of cigarettes are somewhat larger for men than women. The fact that these treatment effects are larger in magnitude than those in Columns (1) and (2) of Table 1 reflects the fact that some individuals that quit initially due to the intervention do not sustain cessation.

As for the mental health effects, there are statistically significant increases in both distress measures in the pooled sample (Panel A). Effects are positive (indicating more distress) for both men and women, and the difference between the 2SLS effects for men and women is not statistically significant.[21] These results suggest that withdrawal causes distress in the short term that is different from longer-run effects. The coefficients on use of anxiolytics and anti-depressants are mostly small and uniformly statistically insignificant.

## 6 Conclusion

The causal relationship between smoking and mental health is not well-understood. Prior work identifies three primary hypotheses for this relationship: (1) smoking worsens mental health; (2) poor mental health increases likelihood of smoking; and (3) the two are not causally related, but coincide due to a third factor. We use previously un-analyzed variables from the Lung Health Study to examine the long-run mental health effects of smoking cessation, aiming to shed light on these potential causal pathways.

Previous work shows differences across gender in the physiological effects of smoking, motivating us to separately analyze men and women. We find that the long-run effects smoking cessation on mental health differs across gender. While women experience improvements in

[21] Appendix Table A4 reports the 2SLS estimates.

mental health, men do not. In fact, we find evidence of an *increase* in severe distress among men, although this increase affects a very small share of the sample. The effects for women are consistent with hypothesis (1), i.e. that smoking was causing worse mental health for this group. For men, the effects are mixed. In particular, some men facing severe mental health issues may select into smoking to help manage severe symptoms (i.e., in line with hypothesis (2)). For others, smoking cessation seems to have little to no effect on reported mental health, in line with hypothesis (3).

Our results suggest that, for some groups, policies which aim to reduce consumption of cigarettes may have unintended mental health consequences. Pairing such policies with mental health supports may reduce these consequences, potentially making making cessation efforts and mental health supports a complementary set of public health interventions.

## References


**Anthonisen, Nicholas R, John E Connett, Paul L Enright, and Jure Manfreda.** 2002. "Hospitalizations and Mortality in the Lung Health Study." *American Journal of Respiratory and Critical Care Medicine* 166 (3): 333–339.

**Anthonisen, Nicholas R, John E Connett, James P Kiley, Murray D Altose, William C Bailey, A Sonia Buist, William A Conway, Paul L Enright, Richard E Kanner, Peggy O'Hara,** et al. 1994. "Effects of Smoking Intervention and the Use of an Inhaled Anticholinergic Bronchodilator on the Rate of Decline of FEV1: the Lung Health Study." *JAMA* 272 (19): 1497–1505.

**Anthonisen, Nicholas R, John E Connett, and Robert P Murray.** 2002. "Smoking and Lung function of Lung Health Study Participants After 11 Years." *American Journal of Respiratory and Critical Care medicine* 166 (5): 675–679.

**Anthonisen, Nicholas R, Melissa A Skeans, Robert A Wise, Jure Manfreda, Richard E Kanner, John E Connett, and Lung Health Study Research Group.** 2005. "The Effects of a Smoking Cessation Intervention on 14.5-year Mortality: a Randomized Clinical Trial." *Annals of Internal Medicine* 142 (4): 233–239.

**Ashton, Heather, JE Millman, Rosemary Telford, and JW Thompson.** 1973. "Stimulant and Depressant Effects of Cigarette Smoking on Brain Activity in Man." *British Journal of Pharmacology* 48 (4): 715.

**Babb, Stephen, Ann Malarcher, Gillian Schauer, Kat Asman, and Ahmed Jamal.** 2017. "Quitting Smoking Among Adults—United States, 2000–2015." *Morbidity and Mortality Weekly Report* 65 (52): 1457–1464.

**Centers for Disease Control and Prevention, Office on Smoking and Health.** 2014. *The Health Consequences of Smoking: 50 Years of Progress. A Report of the Surgeon General.* Technical report. Atlanta, GA, January.

**Cosgrove, Kelly P, Shuo Wang, Su-Jin Kim, Erin McGovern, Nabeel Nabulsi, Hong Gao, David Labaree, Hemant D Tagare, Jenna M Sullivan, and Evan D Morris.** 2014. "Sex Differences in the Brain's Dopamine Signature of Cigarette Smoking." *Journal of Neuroscience* 34 (50): 16851–16855.

**Cotti, Chad, Erik Nesson, and Nathan Tefft.** 2019. "Impacts of the ACA Medicaid Expansion on Health Behaviors: Evidence from Household Panel Data." *Health Economics* 28 (2): 219–244.

**Courtemanche, Charles, Rusty Tchernis, and Benjamin Ukert.** 2018. "The Effect of Smoking on Obesity: Evidence from a Randomized Trial." *Journal of Health Economics* 57:31–44.

**DeCicca, Philip, Donald S Kenkel, and Michael F Lovenheim.** 2020. *The Economics of Tobacco Regulation: A Comprehensive Review.* Working Paper, Working Paper Series 26923. National Bureau of Economic Research, April.

**Dee, Thomas S.** 1999. "The Complementarity of Teen Smoking and Drinking." *Journal of Health Economics* 18 (6): 769–793.

**Fletcher, Jason, and Ryne Marksteiner.** 2017. "Causal Spousal Health Spillover Effects and Implications for Program Evaluation." *American Economic Journal: Economic Policy* 9 (4): 144–66.

**Fluharty, Meg, Amy E. Taylor, Meryem Grabski, and Marcus R. Munafa.** 2016. "The Association of Cigarette Smoking With Depression and Anxiety: A Systematic Review." *Nicotine & Tobacco Research* 19, no. 1 (May): 3–13.

**Friedman, Abigail S.** 2020. "Smoking to cope: Addictive behavior as a response to mental distress." *Journal of Health Economics* 72:102323.

**Holford, Theodore R, David T Levy, Lisa A McKay, Lauren Clarke, Ben Racine, Rafael Meza, Stephanie Land, Jihyoun Jeon, and Eric J Feuer.** 2014. "Patterns of Birth cohort–Specific Smoking Histories, 1965–2009." *American Journal of Preventive medicine* 46 (2): 31–37.

**Kanner, Richard E, Nicholas R Anthonisen, and John E Connett.** 2001. "Lower Respiratory Illnesses Promote FEV1 Decline in Current Smokers but not Ex-Smokers with Mild Chronic Obstructive Pulmonary Disease: Results from the Lung Health Study." *American Journal of Respiratory and Critical Care Medicine* 164 (3): 358–364.

**Kanner, Richard E, John E Connett, David E Williams, and A. Sonia Buist.** 1999. "Effects of Randomized Assignment to a Smoking Cessation Intervention and Changes in Smoking Habits on Respiratory Symptoms in Smokers with Early Chronic Obstructive Pulmonary Disease: the Lung Health Study." *The American Journal of Medicine* 106 (4): 410–416.

**Kanner, Richard E, and Lung Health Study Research Group.** 1996. "Early Intervention in Chronic Obstructive Pulmonary Disease: a Review of the Lung Health Study Results." *Medical Clinics* 80 (3): 523–547.

**Kling, Jeffrey R., Jeffrey B. Liebman, and Lawrence F. Katz.** 2007. "Experimental Analysis of Neighborhood Effects." *Econometrica* 75 (1): 83–119.

**Lasser, Karen, J Wesley Boyd, Steffie Woolhandler, David U Himmelstein, Danny McCormick, and David H Bor.** 2000. "Smoking and Mental illness: a Population-Based Prevalence Study." *JAMA* 284 (20): 2606–2610.

**Maclean, Johanna Catherine, and Brendan Saloner.** 2019. "The Effect of Public Insurance Expansions on Substance Use Disorder Treatment: Evidence from the Affordable Care Act." *Journal of Policy Analysis and Management* 38 (2): 366–393.

**McClave, Annette K, Lela R McKnight-Eily, Shane P Davis, and Shanta R Dube.** 2010. "Smoking Characteristics of Adults with Selected Lifetime Mental Illnesses: Results from the 2007 National Health Interview Survey." *American Journal of Public Health* 100 (12): 2464–2472.

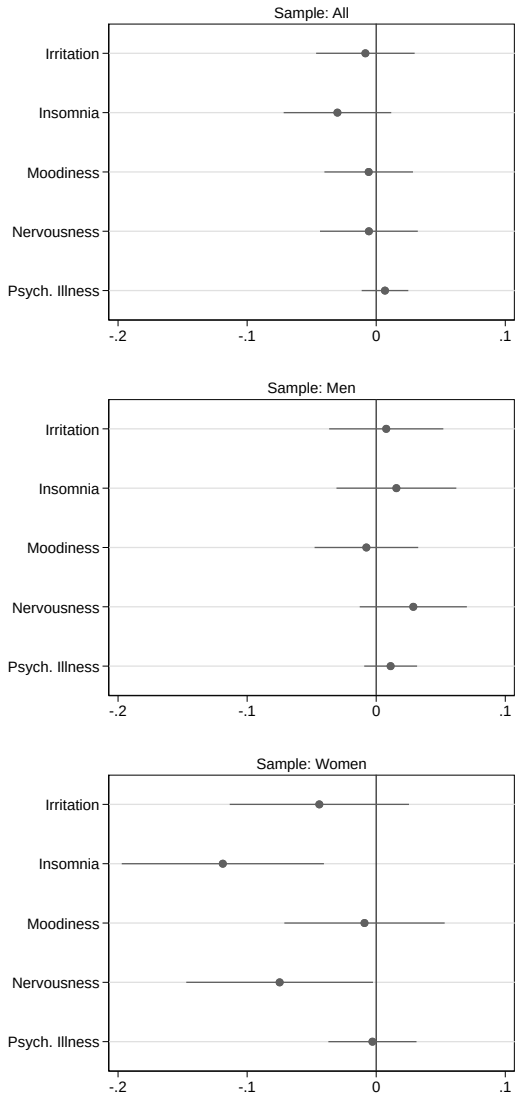
**Murray, Robert P, Joseph A Istvan, and Helen T Voelker.** 1996. "Does Cessation of Smoking Cause a Change in Alcohol consumption? Evidence from the Lung Health Study." *Substance Use & Misuse* 31 (2): 141–156.

**O'Hara, Peggy, Joseph Grill, Michael A Rigdon, John E Connett, Gary A Lauger, and Janet J Johnston.** 1993. "Design and Results of the Initial Intervention Program for the Lung Health Study." *Preventive Medicine* 22 (3): 304–315.

**Perkins, Kenneth A, and Joshua L Karelitz.** 2015. "Sex Differences in Acute Relief of Abstinence-Induced Withdrawal and Negative Affect Due to Nicotine Content in Cigarettes." *Nicotine & Tobacco Research* 17 (4): 443–448.

**Picciotto, Marina R, Darlene H Brunzell, and Barbara J Caldarone.** 2002. "Effect of Nicotine and Nicotinic Receptors on Anxiety and Depression." *Neuroreport* 13 (9): 1097–1106.

Figure 1: Long-Run Effects of Cessation Program on Distress Score Components




Notes: Displayed above are point estimates and 95% confidence intervals corresponding to $\beta$ from Eq. (1). The dependent variable is a numeric score corresponding to the severity of the indicated mental health condition, averaged over years 2 through 5. Participants are asked: "Indicate the extent to which you have been troubled in the last four months by any of the following [mental health conditions]. Please indicate Severe, Moderate, Mild, or Not at all." We translate these answers to a numeric score from 0 through 3, where 0 corresponds to "Not at all" and, 3 corresponds to "Severe." Thus an increase in the score indicates an increase in distress. Appendix Table 2 reports the point estimates, standard errors, and p-values from these regressions. Standard errors are adjusted for heteroskedasticity. See the notes to Table 1 for more information on the sample.

Table 1: Long-Run Effects of the Cessation Program on Smoking Outcomes

| | (1)<br>Sustained Quit | (2)<br>Current Quit | (3)<br>Cigs per Day |
|---|---|---|---|
| *Panel A: All* | | | |
| Treatment | 0.1689 | 0.1894 | -8.0687 |
| | (0.0086) | (0.0101) | (0.3674) |
| | [0.0000] | [0.0000] | [0.0000] |
| Obs. | 5,627 | 5,705 | 5,705 |
| Control Mean | 0.0546 | 0.1724 | 21.4838 |
| *Panel B: Men* | | | |
| Treatment | 0.1843 | 0.1922 | -8.6535 |
| | (0.0110) | (0.0129) | (0.4880) |
| | [0.0000] | [0.0000] | [0.0000] |
| Obs. | 3,519 | 3,575 | 3,575 |
| Control Mean | 0.0546 | 0.1779 | 22.4267 |
| *Panel C: Women* | | | |
| Treatment | 0.1438 | 0.1854 | -6.9925 |
| | (0.0137) | (0.0163) | (0.5374) |
| | [0.0000] | [0.0000] | [0.0000] |
| Obs. | 2,108 | 2,130 | 2,130 |
| Control Mean | 0.0547 | 0.1627 | 19.8210 |
| P-Value, Men-Women | 0.0214 | 0.7420 | 0.0221 |

Notes: The dataset is the Lung Health Study, limited to individuals with non-missing values of the dependent variable and the long-run distress score. Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate regression estimating Eq. 1. The dependent variable in column (1) is an indicator for whether the participant sustained cessation through all 5 follow-up interviews. The dependent variable in column (2) is an indicator for current cessation, averaged over interview years 2 through 5. Cessation is clinically validated. The dependent variable in column (3) is average cigarettes per day over the last year, averaged over interview years 2 through 5. At the bottom of the table, we report p-values from a Chi-squared test of whether the difference in coefficients is equal to 0.

Table 2: Long-Run Effects of Cessation Program on Mental Health

| | (1) Distress Scale | (2) Distress: Severe | (3) Anxiolytic | (4) Anti-Depressant |
|---|---|---|---|---|
| *Panel A: All* | | | | |
| Treatment | -0.0436 | 0.0016 | 0.0082 | -0.0024 |
| | (0.0671) | (0.0020) | (0.0072) | (0.0074) |
| | [0.5165] | [0.4187] | [0.2563] | [0.7514] |
| Obs. | 5,705 | 5,705 | 5,705 | 5,705 |
| Control Mean | 1.7719 | 0.0204 | 0.0681 | 0.0766 |
| *Panel B: Men* | | | | |
| Treatment | 0.0558 | 0.0048 | 0.0068 | -0.0026 |
| | (0.0762) | (0.0022) | (0.0039) | (0.0044) |
| | [0.4644] | [0.0259] | [0.0824] | [0.5557] |
| Obs. | 3,575 | 3,575 | 3,575 | 3,575 |
| Control Mean | 1.4151 | 0.0131 | 0.0206 | 0.0266 |
| *Panel C: Women* | | | | |
| Treatment | -0.2510 | -0.0046 | -0.0020 | -0.0055 |
| | (0.1231) | (0.0038) | (0.0071) | (0.0076) |
| | [0.0416] | [0.2307] | [0.7747] | [0.4714] |
| Obs. | 2,130 | 2,130 | 2,130 | 2,130 |
| Control Mean | 2.4010 | 0.0334 | 0.0448 | 0.0523 |
| Diff, Men-Women: b/cigs | -0.0423 | -0.0012 | -0.0011 | -0.0005 |
| P-Value | 0.0309 | 0.0438 | 0.3326 | 0.6897 |

Notes: Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate regression estimating Eq. 1. The dependent variable in Column (1) is equal to the sum of the distress scores across the five mental health conditions, averaged across years two through five. The dependent variable in Column (2) is equal to an indicator for severe distress, averaged across the five mental health conditions and interview years 2 through 5. The dependent variables for Columns (3) and (4) are equal to indicators for whether the participant took anti-anxiety drugs or anti-depressants over the past 12 months, averaged over interview years 2 through 5. We estimate IV regressions of the effects of smoking cigarettes on these outcomes (Eq. 3) separately for men and women. At the bottom of the table, we report the difference between these estimates and the p-value from a Chi-squared test of whether the difference in coefficients is equal to 0. See the notes to Table 1 for more information on the LHS sample.

Table 3: Short-Run Effects of Cessation Program on Smoking and Mental Health

| | (1) Quit | (2) Cigs/Day | (3) Distress Scale | (4) Distress: Severe | (5) Anxiolytic | (6) Anti-Depressant |
|---|---|---|---|---|---|---|
| *Panel A: All* | | | | | | |
| Treatment | 0.2604 | -12.3920 | 0.2113 | 0.0051 | 0.0010 | 0.0035 |
| | (0.0102) | (0.3649) | (0.0704) | (0.0025) | (0.0044) | (0.0038) |
| | [0.0000] | [0.0000] | [0.0027] | [0.0388] | [0.8272] | [0.3585] |
| Obs. | 5,705 | 5,484 | 5,474 | 5,497 | 5,497 | 5,497 |
| Control Mean | 0.0930 | 25.9182 | 1.6778 | 0.0181 | 0.0241 | 0.0164 |
| *Panel B: Men* | | | | | | |
| Treatment | 0.2735 | -13.7460 | 0.2286 | 0.0056 | 0.0013 | 0.0025 |
| | (0.0129) | (0.4766) | (0.0813) | (0.0026) | (0.0050) | (0.0039) |
| | [0.0000] | [0.0000] | [0.0050] | [0.0334] | [0.7942] | [0.5338] |
| Obs. | 3,575 | 3,418 | 3,410 | 3,427 | 3,427 | 3,427 |
| Control Mean | 0.0911 | 27.4547 | 1.3804 | 0.0117 | 0.0190 | 0.0112 |
| *Panel C: Women* | | | | | | |
| Treatment | 0.2386 | -10.0173 | 0.1470 | 0.0036 | -0.0002 | 0.0044 |
| | (0.0168) | (0.5450) | (0.1272) | (0.0049) | (0.0084) | (0.0076) |
| | [0.0000] | [0.0000] | [0.2482] | [0.4700] | [0.9813] | [0.5592] |
| Obs. | 2,130 | 2,066 | 2,064 | 2,070 | 2,070 | 2,070 |
| Control Mean | 0.0964 | 23.2489 | 2.1940 | 0.0291 | 0.0330 | 0.0255 |
| Diff, Men-Women: b/cigs | | | -0.0033 | 0.0000 | -0.0001 | 0.0003 |
| P-Value | 0.0987 | 0.0000 | 0.7927 | 0.9376 | 0.9026 | 0.7383 |

Notes: Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate regression estimating Eq. 1. The dependent variable in Column (1) is an indicator for whether the participant had quit smoking at the time of the first annual interview. The dependent variable in Column (2) is the average number of cigarettes smoked per day in the 12 months before the first interview. The dependent variable in Column (3) is the sum of the distress scores across the five mental health conditions, as reported in the first interview. The dependent variable in Column (4) is an indicator for severe distress, averaged across the five mental health conditions, from the first interview. The dependent variables for Columns (5) and (6) are indicators for whether the participant took anti-anxiety drugs or anti-depressants in the 12 months prior to the first interview. We estimate IV regressions of the effects of smoking cigarettes on these outcomes (Eq. 3) separately for men and women. At the bottom of the table, we report the difference between these estimates and the p-value from a test of whether they are equal. See the notes to Table 1 for more information on the LHS sample.

Table A1: Balance of Pre-Treatment Characteristics, Analysis Sample

| | Treatment | Control | Diff | P-Val |
|---|---|---|---|---|
| Age | 48.4460 | 48.4791 | 0.0332 | 0.8626 |
| Male | 0.6209 | 0.6381 | 0.0172 | 0.2059 |
| no HS Diploma | 0.1225 | 0.1199 | -0.0026 | 0.7779 |
| HS Diploma | 0.3025 | 0.2916 | -0.0109 | 0.3987 |
| College or Trade School | 0.5750 | 0.5885 | 0.0135 | 0.3323 |
| Body Mass Index | 25.5643 | 25.5679 | 0.0036 | 0.9740 |
| Cigs. per Day | 31.2933 | 30.9857 | -0.3075 | 0.3944 |
| Age, First Cigarette | 17.4303 | 17.5695 | 0.1391 | 0.1975 |
| Total Distress Score | 1.8915 | 1.8147 | -0.0768 | 0.2469 |
| Distress: Severe | 0.0233 | 0.0225 | -0.0008 | 0.7608 |
| Irritability, Past 4 Mos. | 0.5450 | 0.5206 | -0.0244 | 0.2814 |
| Insomnia, Past 4 Mos. | 0.3970 | 0.3899 | -0.0072 | 0.7403 |
| Mood Changes, Past 4 Mos. | 0.4049 | 0.3980 | -0.0069 | 0.7357 |
| Nervous, Past 4 Mos. | 0.4923 | 0.4596 | -0.0327 | 0.1392 |
| Psych. Problems, Past 4 Mos. | 0.0523 | 0.0468 | -0.0056 | 0.5037 |
| Anti-Depress., Past Month | 0.0147 | 0.0174 | 0.0027 | 0.4314 |
| Anxiolytic, Past Month | 0.0252 | 0.0243 | -0.0009 | 0.8402 |
| Observations | 3,812 | 1,893 | 5,705 | 5,705 |

Table A2: Long-Run Effects of Cessation Program on Mental Health, 2SLS Estimates

| | (1) Distress Scale | (2) Distress: Severe | (3) Anxiolytic | (4) Anti-Depressant |
|---|---|---|---|---|
| *Panel A: Men* | | | | |
| Avg. Cigarettes per Day, Years 2-5 | -0.0064 | -0.0006 | -0.0008 | 0.0003 |
| | (0.0089) | (0.0003) | (0.0005) | (0.0005) |
| | [0.4666] | [0.0285] | [0.0836] | [0.5560] |
| Obs. | 3,575 | 3,575 | 3,575 | 3,575 |
| Control Mean, Men | 1.4151 | 0.0131 | 0.0206 | 0.0266 |
| F-Statistic | 315 | 315 | 315 | 315 |
| *Panel B: Women* | | | | |
| Avg. Cigarettes per Day, Years 2-5 | 0.0359 | 0.0007 | 0.0003 | 0.0008 |
| | (0.0175) | (0.0005) | (0.0010) | (0.0011) |
| | [0.0403] | [0.2284] | [0.7749] | [0.4726] |
| Obs. | 2,130 | 2,130 | 2,130 | 2,130 |
| Control Mean, Women | 2.4010 | 0.0334 | 0.0448 | 0.0523 |
| F-Statistic | 169 | 169 | 169 | 169 |

Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate 2SLS regression estimating Eq. 3. The dependent variable in Column (1) is equal to the sum of the distress scores across the five mental health conditions, averaged across years two through five. The dependent variable in Column (2) is equal to an indicator for severe distress, averaged across the five mental health conditions and interview years 2 through 5. The dependent variables for Columns (3) and (4) are indicators for whether the participant took anti-anxiety drugs or anti-depressants over the past 12 months, averaged over interview years 2 through 5. At the bottom of the table, we report the F-Statistic from the first stage regression (Eq. 2). See the notes to Table 1 for more information on the LHS sample.

Table A3: Long-Run Effects of Cessation Program on on Distress Score Components

| | (1) Irritation | (2) Insomnia | (3) Moodiness | (4) Nervousness | (5) Psych. Illness |
|---|---|---|---|---|---|
| *Panel A: All* | | | | | |
| Treatment | -0.0084 | -0.0300 | -0.0058 | -0.0057 | 0.0068 |
| | (0.0195) | (0.0212) | (0.0175) | (0.0193) | (0.0092) |
| | [0.6674] | [0.1578] | [0.7398] | [0.7699] | [0.4598] |
| Obs. | 5,705 | 5,705 | 5,705 | 5,705 | 5,705 |
| Control Mean | 0.4697 | 0.4526 | 0.3589 | 0.4057 | 0.0850 |
| *Panel B: Men* | | | | | |
| Treatment | 0.0078 | 0.0157 | -0.0076 | 0.0287 | 0.0112 |
| | (0.0226) | (0.0237) | (0.0205) | (0.0212) | (0.0104) |
| | [0.7303] | [0.5086] | [0.7104] | [0.1744] | [0.2835] |
| Obs. | 3,575 | 3,575 | 3,575 | 3,575 | 3,575 |
| Control Mean | 0.3960 | 0.3411 | 0.3096 | 0.3037 | 0.0648 |
| *Panel C: Women* | | | | | |
| Treatment | -0.0440 | -0.1188 | -0.0091 | -0.0748 | -0.0029 |
| | (0.0354) | (0.0399) | (0.0317) | (0.0369) | (0.0174) |
| | [0.2137] | [0.0030] | [0.7747] | [0.0427] | [0.8674] |
| Obs. | 2,130 | 2,130 | 2,130 | 2,130 | 2,130 |
| Control Mean | 0.5998 | 0.6494 | 0.4457 | 0.5856 | 0.1204 |
| Diff, Men-Women: b/cigs | -0.0072 | -0.0188 | -0.0004 | -0.0140 | -0.0017 |
| P-Value | 0.2057 | 0.0030 | 0.9348 | 0.0153 | 0.5363 |

Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate regression estimating Eq. 1. The dependent variable in each column is the severity score for the given mental health condition, averaged across interview years 2 through 5. We estimate IV regressions of the effects of smoking cigarettes on these outcomes (Eq. 3) separately for men and women. At the bottom of the table, we report the difference between these estimates and the p-value from a test of whether they are equal. See the notes to Table 1 for more information on the LHS sample.

Table A4: Short-Run Effects of Cessation Program on Mental Health, 2SLS Estimates

| | (1) Distress Scale | (2) Distress: Severe | (3) Anxiolytic | (4) Anti-Depressant |
|---|---|---|---|---|
| *Panel A: Men* | | | | |
| Avg. Cigarettes per Day, Year 1 | -0.0161 | -0.0004 | -0.0001 | -0.0002 |
| | (0.0058) | (0.0002) | (0.0003) | (0.0003) |
| | [0.0054] | [0.0388] | [0.7941] | [0.5351] |
| Obs. | 3,575 | 3,575 | 3,575 | 3,575 |
| Control Mean, Men | 1.3804 | 0.0117 | 0.0190 | 0.0112 |
| F-Statistic | 71 | 71 | 71 | 71 |
| *Panel B: Women* | | | | |
| Avg. Cigarettes per Day, Year 1 | -0.0128 | -0.0003 | 0.0000 | -0.0004 |
| | (0.0112) | (0.0005) | (0.0008) | (0.0007) |
| | [0.2514] | [0.4736] | [0.9813] | [0.5608] |
| Obs. | 2,130 | 2,130 | 2,130 | 2,130 |
| Control Mean, Women | 2.1940 | 0.0291 | 0.0330 | 0.0255 |
| F-Statistic | 27 | 27 | 27 | 27 |

Each point estimate, heteroskedasticity-robust standard error (in parentheses) and p-value (in brackets) is from a separate 2SLS regression estimating Eq. 3. The dependent variable in Column (1) is the sum of the distress scores across the five mental health conditions, as reported in the first interview. The dependent variable in Column (2) is an indicator for severe distress, averaged across the five mental health conditions, from the first interview. The dependent variables for Columns (3) and (4) are indicators for whether the participant took anti-anxiety drugs or anti-depressants in the 12 months prior to the first interview. At the bottom of the table, we report the F-Statistic from the first stage regression (Eq. 2). See the notes to Table 1 for more information on the LHS sample.