Smoking, Taxes, and Suicides*

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February 7, 2019

Abstract

Government regulation of cigarettes has resulted in a large decrease in smoking over the last half century. I explore the hypothesis that this reduction has had the unintended consequence of leaving some individuals vulnerable to the effects of mental illness, as nicotine has psychological effects. Then, a welfare-improving policy would pair regulation with spending on mental health services.

I test this hypothesis in two ways. First, utilizing a randomized trial of a smoking cessation intervention, I show that quitting results in decreased mental health 1-5 years later, although only for men. Second, using variation in cigarette taxes at age 18 in one's state of birth, as well as peer smoking rates in youth, I show that smoking throughout adulthood is associated with fewer suicides in middle age but more deaths due to respiratory disease. Results are concentrated among men.

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1 Introduction

"*Smoke a Lucky to feel your level best! ...*
Picks you up when you are low... calms you down when you are tense."
— Lucky Strike ad, 1949

Since approximately 1964, when the Surgeon General's report revealed the important health costs of cigarettes, the U.S. has led a successful campaign to reduce smoking via legislation to restrict sales of tobacco (taxes and bans) and public education. As a result, the prevalence of smoking has declined greatly, from 42.4% of adults (18+) in 1965 to 18.1% in 2012, with especially large declines for men (51.9% to 19.0%) compared to women (33.9% to 17.3%).[1] It is estimated that reductions in smoking due to tobacco control policies have saved 8 million lives (5.3 million men and 2.7 million women) and resulted in other important health improvements, such as lowering the incidence of low birth weight and premature birth (Currie, Neidell, and Schmieder, 2009; Evans and Ringel, 1997; Holford, Meza, et al., 2014).

In this paper, I explore the hypothesis that the reduction in smoking has had the unintended consequence of leaving some individuals more vulnerable to the effects of mental illness. For these individuals, the nicotine in cigarettes serves as a form of "self-medication" for mood disorders, in lieu of a formal, or less harmful, treatment. My hypothesis is motivated by the well-documented correlation between smoking and mental illness (e.g., Lasser et al., 2000) as well as evidence from medical studies that nicotine has properties as an acute analgesic, anti-depressant and anti-anxiety agent (anxiolytic), although, in some settings, it may also aggravate these conditions (Ditre et al., 2016; McClernon et al., 2006; Picciotto, Brunzell, and Caldarone, 2002, respectively, among others).

If cigarettes are used as a form of "self-medication" for mood disorders, then the effect of quitting may be to reduce mental health and/or increase utilization of alternative treatments (e.g., prescription drugs) in the long term. I test this hypothesis using the Lung Health Study, a randomized controlled trial conducted in the early 1990s in which a group of current smokers aged 35 to 60 were either assigned receive to an intensive anti-smoking intervention, or to a control group, and then were followed for the next five years. Each year, they were medically tested for smoking cessation, and also asked a series of questions on mental health and prescription drug usage.

I first establish that the treatment was effective — the likelihood of quitting during the initial intervention and sustaining cessation through all five follow-up interviews is significantly higher among the treatment versus the control group (16.6 percentage points). I then show that, for men, assignment to the treatment group is associated with decreases in mental health (measured by an index compiling indicators for moodiness, nervousness, anxiety and depression) and increased use of anxiolytics across the five annual follow-up interviews. By contrast, effects are insignificant or indicative of mental health improvements for women. The effects are similar when I consider only the later follow-up years, in order to limit the immediate effects of withdrawal and recovery from nicotine.

[1] Source for these statistics: `https://www.surgeongeneral.gov/library/reports/50-years-of-progress/sgr50-chap-13.pdf`

These results speak to a specific scenario — the effects of quitting among smokers —, but my hypothesis also implies that mental health may vary across smokers and non-smokers. Therefore, in a separate analysis, I investigate whether variation in the likelihood of smoking throughout adulthood alters the distribution of mortality outcomes related to mental illness (suicides and drug poisonings) and smoking (heart disease, cancer, and respiratory disease). For this purpose, I combine individual-level data consisting of the universe of death certificates in the U.S. for 1990-2004 with a novel source of data on smoking behavior — Gallup Polls. Gallup polls are nationally representative surveys of public opinion conducted since the 1930s that consistently ask a question about smoking behavior. The fact that this question is uniform over time and also that many interviews also include data on demographic information (e.g., state of residence), make these data valuable in comparison to other longitudinal smoking series.

Previous research has established a correlation between smoking, suicide and drug overdoses, but causal mechanisms are not well-understood (Hughes, 2008). To identify the causal effect of smoking throughout adulthood on mortality outcomes, I exploit within-state variation in cigarette taxes in one's state of birth at age 18, when purchase is legal. In addition, as an alternate predictor of smoking, I construct the average youth smoking rate among one's peers at age 18, where peers are defined by birth cohort, Census Region of birth, race, and sex. My predictors are motivated by the fact that nearly all adult smokers initiate as teens, and that both tax rates and peer smoking have been found to predict smoking (e.g., Card and Giuliano, 2013; Carpenter and Cook, 2008; Gruber and Köszegi, 2001; Gruber and Zinman, 2001). To my knowledge, this is the first paper that links mortality outcomes to cigarette taxes encountered in youth.

My analysis samples are limited to native-born individuals aged approximately 35-60 ("middle age"), corresponding to cohorts for whom I observe both predictor and outcome variables (and also corresponding to the ages from the Lung Health Study). I estimate regressions that control for fixed differences across birth cohorts, birth states, demographic groups, and years. I first establish that both tax rates and youth smoking at age 18 are predictive of smoking behavior in middle age. For example, for taxes, an increase of \$0.01 in 1970\$ (and \$0.06 in 2016\$) reduces smoking in middle age by 0.87 percentage points (pp), or 2.01%, although this effect is somewhat imprecise. Effects are larger and statistically significant for men, while smaller and insignificant for women. For the youth smoking rate, a 10 pp increase increase adult smoking by 1.8 pp.

Next, I combine the death certificate data with population counts from the Census to construct death rates for suicides, poisonings, and smoking-related mortality. Estimating reduced form models, I find that that a \$0.01 increase in taxes at age 18 increases suicides in middle age by 0.0014 per thousand (0.88% of the mean). These results are robust to several modifications, including the addition of state of birth-specific time trends, replacing tax at 18 with average taxes over adolescence (15-18), removing demographic controls, etc. Findings from a regression of suicide on youth smoking among peers at age 18 implies a similarly sized results when scaled to effects on smoking. For both predictors, effects are larger and more significant among men than women.

As for drug poisonings, effects are noisier, but imply that higher taxes leads to more poisonings, consistent with substitution to other forms of "self-medication." Effects on the youth smoking rate imply an opposite signed effect, however, which may occur if peer smoking in youth is predictive of other risky behavior (e.g., drug use). As for smoking-related mortality, I find that a higher cigarette tax leads to fewer deaths due to respiratory disease (1.38% for \$0.01 in 1970\$). I do not find effects for cancer or heart disease, which is likely due to the fact that I focus on middle-age individuals and/or imperfect overlap between these outcomes and underlying smoking status.

The results of my analysis imply the importance of combining policy to regulate harmful substances such as cigarettes with efforts to address and treat the underlying mental disorders that they may be used to treat (via "self-medication"). This insight can be applied in the case of the current opioid epidemic, the response to which often focuses on restricting access and educating the public about costs.[2] My results also indicate that, in the case of tobacco regulation, such efforts may be particularly welfare-improving for men.

My findings are related to recent work demonstrating that suicides and drug overdoses have increased from 2000-2015 among middle-aged, white Americans of low educational status, especially for men (Case and Deaton, 2015, 2017). The timing of the initial decline in youth smoking (starting in approximately 1970), suggests an earlier increase in suicides (e.g., youths in the 1970 are in "middle age" starting around 1985, per my definition), however. Still, it seems plausible that those for whom cigarettes act as self-medication would tend to quit later than the average smoker (e.g., at a higher tax rate, due to a higher willingness to pay). Indeed, smoking remains significantly higher among those with poor mental health (Lasser et al., 2000). Data series containing both smoking and mental health behaviors for decades earlier than the 1990s is scarce, however.

The paper proceeds as follows. Section 2 describes the data. Section 3 discusses the empirical methods. Section 4 describes the results. Section 5 concludes.

2 Data

2.1 Lung Health Study

The Lung Health Study (LHS) was a clinical trial conducted from 1989-1990 in which a group of 5,887 current smokers aged 35-60 were randomly assigned to either an intensive smoking cessation intervention or to a control group that received no treatment (Anthonisen et al., 1994; Connett et al., 1993). Participants were then interviewed annually for the next 11 years in a clinic setting, which included medical testing for smoking cessation (via "salivary cotinine assay and exhaled CO measurement"). Interviews cover other health outcomes, including mental health status and usage of prescription medications.

The recruitment and screening process occurred from October 1986-January 1989 at 10 clinical centers in the following cities: Baltimore, Birmingham, Cleveland, Detroit, Los Angeles, Pittsburgh,

[2] E.g., see `https://www.dea.gov/press-releases/2018/07/11/department-justice-announces-regulatory-steps-addr`

Portland (OR), Rochester, Salt Lake City, and Winnipeg (500-600 participants per clinic).[3] To be eligible, participants had to be at risk for chronic obstructive pulmonary disease (COPD), have no other serious illnesses or medical conditions, and have no plans to move away from the clinic area during the study. Recruitment strategies included media campaigns, mailings, worksite and public site recruitment and HMO referrals.

Two-thirds of study participants (3,925) were assigned to the treatment group, which consists of two separate arms. The difference between the arms is that one group received an inhaled bronchodilator (in addition to the baseline treatment), while the other did not. For my purpose, I do not differentiate between these arms, as the bronchodilator was later found to have little effect on outcomes (Anthonisen et al., 1994). The control arm consists of 1/3 of sample, or 1,962 individuals. The smoking cessation intervention consists of the following: (1) a physician's message regarding current lung impairment and the risk of smoking in developing lung and cardiovascular disease; (2) a behavioral intervention program overseen by a specialist consisting of a "12 session group intervention program spread over a 10 week period combining general principles of cognitive and social learning theory with nicotine replacement in the form of nicotine gum" (to which the individual's spouse was invited); (3) clinic meetings every 4 months, incorporating individual meetings with intervention staff ("purpose is to assess and promote regimen adherence"); and (4) an extended intervention program for those who do not succeed quitting following the initial intervention ("program options include individual counseling, group meetings, physician visits, and restart groups").

I received a de-identified version of the data that exclude geographic identifiers and cover the screening interviews and the first 5 annual follow-ups. Attrition is low, which is fortunate for my purposes, with 5,556 participating in the study at the end of 5 years (6% attrition, half of which are deaths). To assess effects on smoking cessation, I create two outcome variables — (1) whether the individual quits during the intervention and sustains cessation (medically validated) for all five follow-up years and (2) whether they additionally do not report currently using nicotine gum, snuff or chewing tobacco in all five follow-ups.[4] If participants can substitute towards alternative nicotine sources, any effects on mental health I find may not be due to nicotine.

At each annual interview, participants are also 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 (i.e., not including psychiatric medications) and then by listing up to three additional drugs outside of these categories.[5] Participants are instructed to bring in pill bottles or drug containers for medications they are taking, for this purpose. The drug fields are not cleaned and

[3] "The participating clinics were selected by the [National Heart, Lung, and Blood Institute (NHLBI)] on the basis of expertise in pulmonary disease, adequacy of staff and facilities, and plausibility of their proposed plans for recruitment and follow-up" (Connett et al., 1993).

[4] In annual follow-ups 1-4, the LHS asks "Have you used [the substance] in the last 24 hours?" In fifth annual follow up, the question is 'Have you used [the substance] in the last 48 hours?"

[5] The categories are as follows: "MD-prescribed theophylline or xanthines", "Other MD-prescribed bronchodilators in tablet form," "Beta-blockers," "Calcium channel blockers," "Insulin," "Systemic or inhaled corticosteroids," "Nitroglycerin or other nitrates (for angina)," "Digitalis," "Anticoagulants," "Antiarrhymics," "Anticancer drugs (chemotherapy)."

contain spelling errors and other inaccuracies. In addition, some drugs taken in the early 1990s are expired today.

To match the self-reported drugs to their therapeutic categories (in order to identify usage of anti-depressants, anxiolytics, and painkillers), I take the following steps. I first Google each of the 1244 distinct drug names, as written. A Google search is helpful because it autocorrects spelling errors and produces partial matches. For 629 of these, the search results in a sidebar Google automatically creates that lists the drug's medication class, which I record.[6] For the remaining 615 drugs for which Google does not produce the automatic description, I utilize 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.[7] 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. At the end of this process, 38 drug entries from the LHS remain unmatched to therapeutic classes and are unused in my analysis. Appendix Section A lists the subsequent steps used to identify drug classes of interest.

The LHS also 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." For each mental condition, I create an indicator corresponding to cases in which the individual indicates "Severe," to focus on cases in which mental health outcomes may involve treatment or lead to "deaths of despair." About 5-8% of individuals per year do not answer the prescription drug and mental health questions, either due to attrition or failing to answer the question and are assigned missing values.[8]

For each interview year, I create a composite mental health index that combines these indicators. The index is constructed to have a mean of 0 and a standard deviation of 1, such that an increase in the index indicates better mental health. The creation of an index is suggested by the literature on multiple hypothesis testing (Anderson, 2008; Kling, Liebman, and Katz, 2007). If there are k indicators and Y_k is the k^{th}, then let μ_k be the mean and σ_k be the SD. The outcomes are normalized by subtracting the mean and dividing by the SD: $Y_k^* = \frac{Y_k - \mu_k}{\sigma_k}$. The summary index is then $Y^* = \sum_k \frac{Y_k^*}{K}$.[9]

In Appendix Table 1, I assess the successfulness of randomization by comparing baseline characteristics recorded during the screening interviews in 1988-9 across the treatment and control groups. These measures include demographic indicators (sex, age, and education), smoking mea-

[6] E.g., for "Venlafaxine," a popular anti-depressant, Google's sidebar lists "Nerve pain medication and antidepressant" and "It can treat depression, generalized anxiety disorder, panic disorder, and social anxiety disorder.", as shown here: `https://www.google.com/search?q=venlafaxine`.

[7] 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."

[8] LHS does not provide additional detail on the reason for missing values.

[9] Using inverse covariance weights, as suggested by Anderson (2008), produces similar results.

sures (cigarettes per day and age at first cigarette), as well as the baseline mental health index and prescription drug usage. The latter two measures are constructed using the same procedure as described above for the annual follow-up measures except that respondents were only asked to list two additional prescription drugs, rather than three, which may result in an artificially lower rate of usage, if a person takes several prescriptions.

Appendix Table 1 reveals that, prior to treatment, participants are about 48 years old, more likely to be male (63% overall), started smoking around age 17, and smoke about 31 cigarettes a day. Reports of "severe" irritability, insomnia, nervousness or mood changes over the previous 4 months occur for only about 1-4% of participants (per condition). About 2-3% of participants report utilizing anti-depressants and anxiolytics, respectively and 7% report using painkillers. The sample means are not statistically distinguishable across the treatment and control groups, suggesting that randomization was successful.

I then generate the following outcome variables from the annual follow-up interviews: the average of the mental health indicator across follow-up interviews 1-5 and 3-5, and an indicator for whether the individual ever reports taking an antidepressant, anxiolytic, or analgesic during follow-up years 1-5 or 3-5. I generate the "3-5 year" measures as an alternative to the "1-5 year" measures as a way of avoiding conflating the short term effects of quitting that may occur after the initial intervention (including withdrawal and recovery) with longer term effects on mental health.[10]

2.2 Gallup Poll Data — Smoking Behavior

My second source of data is Gallup polls, which are surveys of public opinion that have been conducted regularly since the late 1930s by the non-partisan American Institute of Public Opinion (AIPO).[11] Face-to-face or telephone interviews are conducted with a single adult in the sampled household and sampling is stratified to be nationally representative.[12] Farber et al. (2018) evaluate the extent to which the sampling methodology produces a representative sample of interviewees via comparison to contemporaneous Census counts — they find that samples are representative starting after 1950, before which the polls under-sample the South and, particularly, blacks in the South. Documentation provided by Gallup regarding the sampling weights is incomplete — for these reasons, I present results without weights, although adding weights does not measurably alter my results, as expected (unreported, available on request).[13]

Many questions asked by the Gallup polls are specific to the given time period (e.g., attitudes about the current president), but there are a subset of questions that are consistently asked across surveys over time. One of these is the following: "Have you, yourself, smoked any cigarettes in the

[10]Lung recovery occurs over the first 12 months while weight stabilization may take two years. Source: `https://www.verywellmind.com/after-the-last-cigarette-how-your-body-heals-2824388`.

[11]These data are made available via the Roper Center at Cornell: `https://ropercenter.cornell.edu/CFIDE/cf/action/catalog/`.

[12]In 1988, Gallup switched from door-to-door (face-to-face) to telephone interviews (Voss, Gelman, and King, 1995).

[13]Before the mid 1980s, Gallup relied on "times-at-home" weighting, in which respondents were asked how many days of the week they would have been home at the time they were interviewed. Starting around 1985, weights are calculated based on the distribution of demographic characteristics in the Census. Further detail is not made available.

past week?" Additional "core" questions I make use of are: state of residence, age, sex, and race (black vs. not). Restricting my focus to surveys that contain each of these questions and for which the sampling basis is nationally representative and focused on adults ("National Adult"), I gather 23 Gallup surveys, or 28,868 observations, from 6/1969 to 7/2011.[14] Of these observations, there are 27,410 that have non-missing answers to all of the variables of interest.

My use of the Gallup poll data to analyze smoking usage across time represents an important addition to our knowledge of smoking habits. The National Health Interview Survey (NHIS), which is often used to analyze historical trends (e.g., Holford, Levy, et al., 2014; Walque, 2010), asks about smoking status starting in 1965 (every few years at first and then annually) in a different way (regarding whether the individual has smoked 100 cigarettes in their lifetime, and then, if so, about smoking behaviors), and the wording has changed over time.[15] In addition, geographic identifiers are unavailable in the public-use NHIS data.

I first limit my sample to "adults" (as differentiated from "youths," defined below), which I define as aged 35+, corresponding with the Lung Health Study sample. I then construct and merge in two different predictors of lifetime smoking — the cigarette tax rate faced at age 18 and the average youth smoking rate among one's peers at 18. These predictors are motivated by previous research that finds that teen smoking is responsive to peer smoking and cigarette taxes and that there is a high rate of continuation of smoking from youth to adulthood (e.g., Card and Giuliano, 2013; Carpenter and Cook, 2008; Gruber and Köszegi, 2001; Gruber and Zinman, 2001, respectively). I return to this literature in more detail when discussing the magnitude of my estimates below.

For the tax rate at age 18, I obtain data on the sum of state and federal cigarette taxes per pack by state and year for 1970-2011 from Orzechowski and Walker (2011).[16] I deflate the series to 1970 dollars using a Consumer Price Index at the annual level.[17] My analysis below uses state fixed effects, so effects are identified off of within-state changes in tax rates. Importantly, a series starting in 1970 captures much of the within-state variation, as state adoption of cigarette taxes occurred through the 1960s.[18]

I then merge the tax rate onto the Gallup sample of adults aged 35+ by year at age 18 and state of *current* residence. Doing so assumes that one remains in one's state of residence from youth to adulthood, which introduces some error (although I do not need that assumption in the reduced form regressions below, as I observe birth state in the mortality outcomes data). Out of the 18,427 individuals in the full sample of adults 35+, I observe "age-18" tax rates for 3,651, interviewed from

[14] Dates are: 7/1969, 5/1971, 4/1972, 5/1974, 8/1977, 1/1978, 6/1981, 8/1984, 3/1987, 7/1988, 5/1989, 7/1990, 10/1991, 3/1994, 5/1996, 3/1997, 6/1997, 7/1997, 5/1999, 11/2000, 8/2007, 6/2009, 7/2011.

[15] Source: `https://www.cdc.gov/mmwr/preview/mmwrhtml/su6001a24.htm`.

[16] These data are available here: `https://chronicdata.cdc.gov/Policy/The-Tax-Burden-on-Tobacco-1970-2017/7nwe-3aj9`.

[17] Source: `https://fred.stlouisfed.org/series/CPIAUCSL#0`. Data series is annual "Consumer Price Index for All Urban Consumers: All Items (CPIAUCSL)." Index is set equal to 100 in 30.06.1970 (tobacco data starts in 1970). The aggregation method is "average."

[18] Source: `https://taxfoundation.org/when-did-your-state-adopt-its-cigarette-tax/`. The last state top adopt was North Carolina in 1969.

1987-2011, at ages 35-59. Taxes at age 18 correspond to years 1970-1987 in this sample.

Next, to construct the measure of youth smoking smoking among one's peers at age 18, I take the raw Gallup poll sample and keep individuals ("youths") aged 16-25 (4,021 out of 27,410 observations).[19] I then calculate average youth smoking rates per 5-year interval for the following demographic cells: Census region of residence, race (black vs. non-black), and sex.[20] I use regions rather than states and 5 year intervals rather than single years to ensure adequate sample size per cell.

Notably, there is significant variation in the youth smoking across and within demographic groups over time. For white youths, for example, the youth smoking rate falls 50% from 1970-85 (the fastest period of decline), from 0.48 to 0.24. Smoking is also more common among young men than young women (0.57 vs. 0.40 in 1970, for example). These rates are similar to those for high school seniors from alternative data that covers the same time period (e.g., Aguilar and Pampel, 2007, based on Monitoring the Future data.).

I merge the youth smoking rates by race/Census region/sex and the 5 year interval in which the adult was aged 18 to my sample of 35+ adults. In other words, for a white male aged 50 in 2000 living in New York (so 18 in 1978), I merge in the average smoking rate among white men aged 16-25 in the Middle Atlantic in 1975-1980. Doing so assumes that one remains in one's Census Region from youth to adulthood (although, again, in the deaths data, I will be able to observe Census Region of birth). Out of the 18,427 individuals in the full sample of adults 35+, 4,325 match to cells covered by the youth smoking means. These 4,325 adults for which the merge was successful were surveyed from 1987-2011 and are at ages 35-61.

Appendix Table 2 shows the summary statistics for the samples of middle aged adults (those for whom I observe a youth smoking rate in Column (1) and those for whom I observe the cigarette tax rate at age 18 in Column (2)).[21] The average age at the time of survey is 42.54 in Column (1) and 41.89 in Column (2). In both samples around 25% report smoking in the last week (25.50% and 25.80%). The average youth smoking rate faced at age 18 is 42.95% (Column (1)) and the average age-18 cigarette tax rate is \$0.15 in 1970 dollars, which translates to \$0.90 in 2016 dollars (Column (2)). Finally, the the average price per cigarette pack, which is also reported by Orzechowski and Walker (2011), is \$0.36 (\$2.23 in 2016\$).[22]

[19] For some surveys in my sample, the youngest age is 16, and for others, the youngest age is 18. Constructing the youth smoking rate for ages 18 to 25 (i.e., uniform across the underlying surveys) does not alter my results.

[20] Mapping of states to Census Regions: `https://www2.census.gov/geo/pdfs/maps-data/maps/reference/us_regdiv.pdf`

[21] Means are unweighted (weighting does not alter the means significantly, in line with findings from Farber et al. (2018)).

[22] Below, I use taxes as my independent variable to predict smoking behavior, following previous studies. Alternate specifications, in which I instrument for price changes with tax variation produce very similar results, which are available on request.

2.3 Mortality Rates

Lastly, I use individual-level data consisting of the the universe of U.S. death certificates for 1990-2004 from the National Center for Health Statistics (NCHS).[23] I use these data to assess the effects of lifetime smoking behavior on mortality outcomes related to mental health, substance abuse and smoking. The death certificates contain information on state of birth (importantly), black race, sex, and birth cohort. I restrict the sample to native-born U.S. residents, aged 35-65, leaving 7,075,381 observations (deaths), or 471,692 per year.

I first identify deaths due to suicide and accidental poisoning using the underlying cause of death variable, following definitions from Case and Deaton (2017).[24] I then utilize definitions from the Center for Disease Control (CDC) to identify deaths due to "diseases caused by smoking," which include: cancer (lip, oral cavity, pharynx, esophagus, stomach, pancreas, larynx, trachea/lung/bronchus, cervix uteri, urinary bladder, kidney and renal pelvis and acute myeloid leukemia), respiratory disease (pneumonia, influenza, bronchitis, emphysema, and chronic airways obstruction disease) and heart disease (ischemic, other, cerebrovascular, atherosclerosis, aortic aneurysm).[25] Importantly, these causes of death may be due to factors other than smoking (e.g., heart disease may be due to diet or genetics), as the death certificate does not identify whether the individual smoked or not (or identify smoking as a cause of death).

I then collapse the data to age-birth year-state of birth-race (black vs. non-black)-sex cells, taking sums of deaths of each category. [26] For example, I have a count of all suicides to white males aged 50 who are born in New York in 1950. To convert these sums into a rate per population, I use the 5% sample from the 1990 Census, which includes state of birth. I create a sum of population per age-birth year-state of birth-race (black vs. not)-sex, which involves dropping the foreign born. I then age forward these sums to cover the years of the deaths data (1990-2004).[27]

Next, I merge in cigarette taxes by year in which the individual was 18 in their state of birth (which assumes one remains in their state of birth at age 18, which seems reasonable). I then merge in the mean youth smoking rate by demographic group (sex/race/Census Region of residence) and

[23] I downloaded the data from the National Bureau of Economic Research, here: `https://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html`. Starting in 2005, the data exclude geographic identifiers.

[24] Codes from the International Statistical Classification of Diseases and Related Health Problems 9th and 10th Revision are as follows: suicide (ICD 9: 9500-9599, ICD 10: X60-84, Y87.0), poisonings (ICD 9: 8500-8589, ICD 10: X40-45, Y10-15, Y45, 47,49). The 10th edition is used starting in 1999.

[25] Source of smoking-attributable mortality definitions here: `https://www.cdc.gov/mmwr/preview/mmwrhtml/mm5745a3.htm`. These diseases are defined by the following codes: cancer (ICD9: 140-149, ICD10: C0-C14 (lip, oral cavity and pharynx); ICD9: 150, ICD10: C15 (esophagus), ICD9:151, ICD10: C16 (stomach), ICD9: 157, ICD10: C25 (pancreas), ICD9: 161, ICD10: 32 (larynx), ICD9: 162, ICD10: 33-34 (trachea/lung/bronchus), ICD9: 180, ICD10: 53 (cervix uteri), ICD9: 188, ICD10: 67 (urinary bladder), ICD9: 189, ICD10: 64-65 (kidney and renal pelvis), ICD9: 205.0, ICD10: 920 (acute myeloid leukemia)), respiratory disease (ICD9: 480-8, 490-2, 496, ICD10: J10-J18, J40-44) and heart disease (ICD9: 410-414, 391-398, 415-429, 430-438, 440, 459, ICD10: I20-25, I00-09, I26-51, I60-69, I70-1, I72-9.)

[26] Note that while the tax data are at the state-year level, the youth smoking rate additionally varies by demographic characteristics, hence the level of the collapse.

[27] This method gives me, for example, total white males aged 50 in 2000 (i.e., who were born in 1950) who were born in New York, as a denominator for the example above.

the 5-year period in which the individual was 18, from the Gallup poll survey data, as above. Of the 7,075,381 deaths in the underlying sample, 1,839,180 match to youth smoking means and 1,539,003 match to cigarette taxes. These individuals for which the merge is successful are aged 35-54 and 35-52, respectively. Taxes in the latter sample are from years 1970-1987 (same as for the Gallup sample above).

Appendix Table 3 presents summary statistics for these two samples (youth smoking sample in Column (1) and cigarette tax sample in Column (2)). Means are weighted by Census population per cell (denominator of death rates). Each observation is a year-birth cohort-birth state-black race-sex cell. The average age at death is 41.60 and 40.83, respectively. The suicide rate is 0.1526 suicides per 1,000 individuals and 0.1460 per 1,000 individuals, respectively. These rates are similar to other national estimates of suicide among middle-aged adults (e.g., the Substance Abuse and Mental Health Services Administration (SAMHSA) reports 0.132 per 1,000 for adults 45-64 in 1999).[28] By comparison, the poisoning rates are 0.1794 and 0.1953 per 1,000 and smoking-related deaths are more common at 1.0706 and 0.9592 (much of which is heart disease). Finally, the youth smoking rate and cigarette taxes at age 18 are 0.4763 and \$0.14 (1970\$, or 0.84 in 2016\$).

3 Empirical Methodology

3.1 Smoking Cessation and Mental Health: Lung Health Study

In order to assess the causal effects of the anti-smoking intervention on smoking behavior and mental health outcomes Y_i, I estimate the following specification.

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

$Treatment_i$ is an indicator for whether individual i is assigned to the treatment group. Randomized assignment to the treatment group implies that $Treatment_i$ is independent of the error term, ϵ_i, in which case β measures the causal effect of the smoking intervention on outcome Y_i.[29] I estimate robust standard errors to allow for the possibility of heteroskedasticity across individuals. I also estimate specifications separately for women and men, given evidence that medical interventions have different effects by sex (Currie, Jin, and Schnell, 2018; Garthwaite, 2012).

3.2 Effect of Smoking in Adulthood on Cause of Death

Next, I use the collapsed mortality and Gallup samples to investigate a separate, but related question: how do mortality outcomes in middle age vary across individuals who smoke throughout adulthood vs. those who do not? If nicotine is an important form of self-medication for mental disorders, then reductions in smoking over time may lead to an increase in deaths due to mental illness or alternative sources of self-medication (suicides and drug overdoses, respectively). Additionally, we would expect a decrease in mortality due to smoking risks (cancer, heart disease, and respiratory disease).

[28] Source: `https://www.samhsa.gov/data/sites/default/files/report_3370/ShortReport-3370.html`.

[29] Assuming $E(\epsilon_i) = 0$, then $E(Y_i|\text{Treatment}_i) = \alpha + \beta\text{Treatment}_i + E(\epsilon_i|\text{Treatment}_i) = \alpha + \beta\text{Treatment}_i + E(\epsilon_i) = \alpha + \beta\text{Treatment}_i$, so $E(Y_i|\text{Treatment}_i = 1) - E(Y_i|\text{Treatment}_i = 0) = \beta$.

A regression of mortality outcomes on smoking may yield biased estimates of underlying causal effects if selection into smoking is related to mental disorders (as per my hypothesis) or other other correlates of health (e.g,, income). Therefore, to identify causal effects, I utilize variation in cigarette taxes as a source of quasi-experimental variation in smoking incentives. As in related work, my models include state and year indicators to remove fixed differences across states and common trends over time (see Carpenter and Cook, 2008, for a review of studies using similar designs).[30] Then, the identifying assumption is that the remaining variation in tax rates is unrelated to factors that influence mortality outside of the direct effect on smoking behavior.

I first test whether cigarette taxes at age 18 predict smoking behavior in middle age, using the individual-level sample of Gallup adults aged 35 to 59. My estimating equation is as follows:

$$Smoke_{icdt} = \alpha + \beta \text{CigTax}_{(c+18)s} + \text{DemogFE}_d + \text{BirthCohortFE}_c + \text{YearFE}_t + \epsilon \tag{2}$$

in which i refers to an individual adult in birth cohort c (single year), year t, and demographic group d, where d is a unique combination of state of residence s, black race, and sex. $Smoke_{icdt}$ is yes/no to "Have you, yourself, smoked cigarettes in past week?" $\text{CigTax}_{(c+18)s}$ indicates the sum of state and federal taxes on a pack of cigarettes at age 18 $(c+18)$ in state s.[31] DemogFE_d represents fixed effects for demographic group d. Other controls include separate FE for birth cohort (single year) and year of survey, as indicated. ϵ is clustered at the state of residence level.

Then, I estimate the corresponding reduced form specification of taxes on mortality outcomes, as follows

$$Y_{cdt} = \delta + \gamma \text{CigTax}_{(c+18)s} + \text{DemogFE}_d + \text{BirthCohortFE}_c + \text{YearFE}_t + \nu \tag{3}$$

in which Y_{cdt} represents the share of individuals in a given birth cohort c (single year), age at year t and demographic group d (unique combination of birth state, black race and sex), that die of a given cause (e.g., respiratory disease). $\text{CigTax}_{(c+18)s}$ indicates the sum of state and federal taxes on a pack of cigarettes at age 18 in state of birth s. Other controls include fixed effects (FE) for demographic group, birth cohort, and year of death, as indicated. ν is clustered at the level of state of birth.

The coefficients of interest are β in Eq. 2 and and γ in Eq. 3, which measure the effect of a \$1.00 increase in taxes (1970\$) on adult smoking and death rates in middle age, respectively. Note that the demographic controls provide additional robustness against potential compositional effects (bias) that may occur if trends in population share by sex or race are correlated with the timing of tax changes. In fact, as discussed below, my results of estimating Eq. 3 are robust to excluding these controls, which is reassuring in that it supports the idea that the timing of tax changes is exogenous. These controls are important in the context of the youth smoking regressions (explained next), and

[30] As mentioned above, however, I am unaware of a study that uses long run mortality as an outcome.

[31] As noted above, this "first stage" regression assumes that individuals remain in their state of residence from age 18 through middle age, which will tend to introduce measurement error, which may attenuate estimates of β.

I retain them for uniformity.

I re-estimate the two specifications above, replacing cigarette taxes ($\text{CigTax}_{(c+18)s}$) with the youth smoking rate, which I denote $\text{YouthSmokeRate5yr}_{d(c+18)}$. $\text{YouthSmokeRate5yr}_{d(c+18)}$ is the youth smoking rate (ages 16-25) for demographic group d (Census Region of residence/black/sex) in the 5-year interval in which cohort c was 18 (e.g., 1980-85 for a $c = 1965$). Corresponding to above, Census Region refers to current residence in the smoking regressions and residence at birth in the mortality regressions. This analysis provides further evidence for the effects of smoking on mortality and also enables me to demonstrate a more direct link between youth smoking behavior and adult smoking

In this case, β and δ measure the relationship between the youth smoking rate in one's demographic group at age 18 and adult smoking and death rates in middle age. The fixed effects for demographic groups remove fixed differences in smoking and mortality that may be unrelated to the causal mechanism of interest. For example, if cigarettes are a normal good and income is protective of health, then a demographic group with higher income could have a higher youth smoking rate and be in better health in middle age. Then, the identifying variation comes from trends in youth smoking within a given demographic group (sex/race/Region) over time. However, it is still possible that there is remaining endogenous variation that my fixed effects would not capture — for example, if trends in peer smoking are correlated with other risky behaviors that affect health.

I estimate three variants of the above specifications as robustness exercises. First, I add state-specific linear time trends to control for the possibility of unobserved endogenous trends that vary across birth states. Second, I replace taxes at age 18 with "average taxes from 15-18," given that youth purchasing restrictions have historically not always been enforced. Third, I remove the demographic fixed effects (age and sex, not state of birth) to test the sensitivity of my estimates.

Each specification is estimated for the full sample and then separately by sex, given my findings from the Lung Health Study that smoking cessation leads to decreases in mental health among men only.

4 Results

4.1 Lung Health Study: Effects of Smoking Cessation on Long Term Mental Health

Using the Lung Health Study sample, I first estimate Eq. 1 in which Y_i is the likelihood of sustaining smoking cessation throughout all five years as well as abstaining from additional nicotine sources, separately for men and women. 1 presents the results. Each column corresponds to a separate regression and reports the coefficient estimate β, its standard error in parentheses and the associated p-value in brackets.

The intervention increases the likelihood of sustained cessation by 18.09% (from a baseline of 0 before the trial) among men and 14.22% among women. Recall that quitting is medically validated through saliva and lung function testing. In Columns (2) and (4), I examine the effect on additionally abstaining from alternative nicotine sources (nicotine patch, chewing tobacco and snuff). In this case,

the effects are a bit smaller, providing some evidence of substitution among those that quit, especially women. However, the effects are still relatively large and highly significant — the intervention causes 13.87% of men and 7.93% of women to abstain from all nicotine.

Next, in Tables 2-3, I present the results of estimating Eq. 1 in which Y_i is one of the mental health outcomes measured over the five annual follow-ups, separately for men and women. For men, the mental health index, averaged over the five years and then over years 3-5, decreases (indicating worse mental health). In addition, men report increased utilization of anxiolytics — for example, utilization of any anxiolytic during years 3-5 increases 1.56%, compared to baseline utilization rate of 2.05% reported during the the screening interviews. As for women, the effects are mostly opposite in sign, with a marginally significant increase in the mental health index in years 3-5 (indicating better mental health), and small or negative coefficients for anxiolytics and anti-depressants (insignificant). However, the coefficients on painkiller usage are positive, relatively large (3.26-3.37%), and marginally significant, which is of interest due to nicotine's reported analgesic properties. Figure 1 displays the coefficients and 95% confidence intervals for the "3-5" year measures, which reflect longer term mental health, and Appendix Figure 1 shows the "1-5" year coefficients.

4.2 Effects of Cigarette Taxes and Peer Smoking in Youth on Adult Smoking

In Table 4, Columns (1)-(3), I present the results of estimating Eq. 2, testing whether the cigarette tax rate at age 18 predicts adult smoking. Column (1) displays results for the full sample and Columns (2)-(3) show results by sex. Because an increase in tax should decrease smoking, the predicted direction of coefficients is negative. Correspondingly, the coefficient is negative (-0.7987, implying a decrease in smoking of -0.007987 for a \$0.01 increase in taxes in 1970\$, or \$0.06 in 2016\$) but only precise at the 10% level (p-value: 0.0915). Still, we might expect the precision to be reduced in comparison to a dataset in which we observe the full population and state of birth, rather than state of residence (as in the mortality dataset). In addition, the effect is larger and statistically significant for men (-1.6157), while smaller and insignificant for women (-0.0135).

It is not entirely straightforward to compare my cigarette tax estimates to those from the literature, as previous research focuses on estimating the *contemporaneous* effects of taxes on smoking behavior (rather than the effect of youth taxes on adult smoking). One important exception is Gruber and Köszegi (2001), who link pregnant women to the cigarette taxes they encountered as a teen in their birth state.[32] They find small, but precise declines (price elasticity of -0.1), indicating the persistence of tax effects. Their sample is comprised of women only, however, for whom I do not find effects.

The overall coefficient of -0.7987, converted to an elasticity, is actually similar to estimates of the contemporaneous demand elasticity among teens from studies using a similar design (state and year fixed effects) and outcome measure (smoking). Specifically, my estimate implies an elasticity to cigarette prices of -1.02, which is similar estimates of around -0.5 to -1.0 from other studies (e.g., Carpenter and Cook, 2008; Dee, 1999; Gruber and Köszegi, 2001; Gruber and Zinman, 2001; Ringel

[32] I am not aware of any additional studies that look at the effect of youth taxes on adult smoking.

and Evans, 2001, among others).[33][34] Note that an elasticity of -1.02 implies a 1.02% decrease in smoking for a 1% increase in price per pack.

Estimates of the contemporaneous cigarette tax elasticity among teens may be a relevant comparison given high continuation rates of smoking from youth to adulthood (over 0.5) Gruber and Köszegi (2001). It also seems plausible that my elasticity (based mostly off of 1970s tax changes) would be larger than existing estimates (largely based off of tax changes in the late 1980s and 1990s). Teen price sensitivity has trended downward over time as smoking prevalence has fallen, potentially reflecting compositional change in tastes and income among smokers (e.g., 1990s teen smokers tended to be more affluent) (Gruber and Köszegi, 2001; Hansen, Sabia, and Rees, 2017).

Next, in Table 4, Columns (4)-(6), I present the corresponding results using the youth smoking rate in one's demographic group at 18 to predict smoking in adulthood. I find that an increase in the youth smoking rate of 1 percentage point (2.3% of the sample mean of 42.95%) increases the likelihood an adult smokes by 0.0018 (0.69%). This effect is highly significant, providing further evidence that smoking in adolescence translates to smoking later in life. Again, effects are larger and more significant for men than women, although precision is reduced in the smaller samples. Translating the overall effect to the tax result yields the following: a *decrease* in the youth smoking rate of 4.5 percentage points (10.5% of sample mean) decreases adult smoking by the same amount (0.7987 percentage points) as an *increase* in taxes of \$0.01 in 1970\$ (7.14%).

Finally, to test the robustness of my results to additional controls, I add state-specific linear time trends in year of birth (Appendix Table 4). These controls can be used to remove pre-existing trends (e.g., in anti-smoking sentiment, as in DeCicca, Kenkel, and Mathios (2002)) that might be correlated with the timing of tax changes. The effects for the youth smoking rate are relatively unchanged, while the effects for taxes are actually larger in magnitude and more precise. The fact that state refers to current residence in the Gallup data, which may differ from state at age 18, complicates interpretation somewhat. I also present results below with and without time trends.

4.3 Effects of Smoking on Mortality Causes

In Tables 5 and 6-7 (Columns (1)-(3) in each table), I present the results of estimating the reduced form specification of adult mortality outcomes on cigarette taxes in one's state of birth at age 18 (Eq. 3), first for the full mortality sample and then separately by sex, respectively. The effect on suicides is positive and highly significant, indicating that a \$0.01 (1970\$, or \$0.06 in 2016\$) increase in taxes at age 18 increases suicides by 0.00144 per 1,000 individuals at ages 35-52. Translating to percentages, a 6.7% increase in taxes (\$0.01 compared to the sample mean tax rate of \$0.14) increases the suicide rate by 0.88%. As for poisonings, the coefficient is also positive, in line with substitution

[33] Other papers, studying smoking initiation, find smaller elasticities (e.g., DeCicca, Kenkel, and Mathios, 2002).

[34] Following (Evans, Ringel, and Stech, 1999) (and other papers in this literature), if price P is a function of taxes T, then the effect of taxes (T) on smoking (Q) is $\delta Q/\delta T = (\delta Q/\delta P)(\delta P/\delta T)$. A commonly used pass-through rate of taxes to prices in recent literature is 1.11 (Carpenter and Cook, 2008). Then, the elasticity of smoking with respect to price is $(\delta Q/\delta T)(P/Q)(1/1.11)$. The average price per pack in the sample is \$P=0.3648 (1970\$), as shown in Appendix Table 2, and the adult smoking rate is 0.2580.

from smoking to alternative "self-medication" sources, but insignificant, with a p-value of 0.1480.

As for the effect on smoking-related mortality, it is negative, corresponding to fewer deaths when cigarette taxes are higher, but statistically insignificant (p-value: 0.72). This result, which is perhaps surprising given that smoking is known to increase mortality, is likely explained by the fact that my sample of deaths is limited to ages 35 to 52, and the vast majority of deaths directly attributable to smoking occur at ages greater than 50 (Burns, 2003).[35] In addition, measurement error in identifying deaths caused by smoking may attenuate the results, as discussed above. In Appendix Table 5, I report the coefficients for the individual categories of "smoking-related" deaths. There is a negative and statistically significant decrease in deaths due to smoking-related respiratory disease, but not the other causes. In particular, a \$0.01 increase in taxes increases respiratory deaths by 0.00066 per thousand (1.38% of mean). It may be that middle-aged individuals with terminal respiratory disease are highly likely to be smokers (as opposed to middle aged individuals who dies of heart disease or cancer, for example).

In Tables 6 and 7 (Columns (1)-(3) in both), I replicate the effects by sex. Effects for suicides and poisonings are larger in magnitude and more significant among men than women, corresponding to the stronger first stage (Table 4). For suicides, for example a \$0.01 increase in cigarette taxes at 18 implies an increase of 0.93% for men versus 0.68% for women, as compared to the sample means, although the latter effect is noisy. For poisonings the positive effect is nearly significant at 10% for men. Effects on smoking-related mortality remain negative but insignificant.

In Appendix Table 7, I replace the tax at age 18 with average taxes for ages 15-18, as purchasing restrictions for minors were not always enforced in decades prior to the 1990s.[36] These results are shown in Appendix Table 7. Given that the average requires multiple years of tax data, the middle aged sample is restricted now to ages 35-49. The effect on suicides and poisonings are somewhat larger and both are now statistically significant at 5%. The coefficient on smoking related deaths is negative and larger in magnitude, but remains insignificant.

Next, in Table 5 and 6-7 (Columns (4)-(6) in each table), I present the corresponding reduced form results of mortality outcomes on the youth smoking rate at age 18 in one's birth state and demographic group. Recall I calculated above that a decrease in the youth smoking rate of 4.53 percentage points has the same effect on adult smoking (a decrease of 0.80 percentage points) as a \$0.01 increase in taxes. From Table 5, a 4.53 percentage point decrease in the youth smoking rate increases suicides by 0.0005 per thousand, or 0.32% (as compared to 0.88% for \$0.01 from the tax regressions). Effects on suicides are larger and more precise for men than women (0.45% vs 0.02%, respectively, for the effects corresponding to a 4.53 pp decrease in youth smoking).

As for poisonings, effects are positive but insignificant, implying an opposite signed effect from the cigarette tax results (i.e., a higher likelihood of smoking is associated with *more* poisonings). It

[35] Recall that the upper age limit of my deaths sample is determined by the overlap between the range of years for which I observe both death outcomes and predictor variables.

[36] In 1992, Congress enacted the Synar Amendment requiring states to enforce the laws prohibiting the sale or distribution of tobacco products to individuals under the age of 18. Source: `https://www.samhsa.gov/synar/about`.

may be that a higher youth smoking rate correlates with other risky behavior (e.g., drug use) outside of its direct effect on adult smoking, as mentioned above. There may also be differences between individuals for whom a marginal tax increase discourages smoking and those for whom peer behavior determines smoking. For smoking-related mortality, a decrease of 4.53 pp in the youth smoking rate decreases deaths by 0.0180 per 1,000 (2.31% of the mean).

Next, I explore the sensitivity of my results for cigarette taxes and the youth smoking rates to two additional variations. First, in **??**, I re-estimate the baseline results (Table 5), adding birth state-specific linear time trends in year of birth, which control against unobserved endogenous trends. The effects for suicides shown in Columns (1) and (4) are quite similar to the baseline results, and the coefficients on poisonings remain statistically insignificant (Columns (2) and (5)). For smoking-related deaths, the effect for the youth smoking rate is similar (Column (6)). However, the effect of taxes implies that an increase in cigarette taxes at 18 increases smoking related deaths in middle age, which is counterintuitive. Disaggregating the causes of smoking-related mortality, I find that the effect on respiratory disease, which likely overlaps most closely with deaths actually caused by smoking, is small (0.0036) and insignificant (p-value: 0.9165).[37] In addition, controlling for time trends may result in biased coefficients if treatment effects evolve dynamically, as seems likely for disease (Meer and West, 2016). Second, in **??**, I remove the controls for race and sex, and the effects for taxes are unchanged, which is reassuring in that it suggests that the timing of tax changes is exogenous (unrelated to shifts in population composition).

Finally, I provide two extensions of these analyses. First, in **??**, I report results of testing whether my predictors of smoking affect the combine mortality for all causes *except* suicide, poisonings and smoking-related mortality. There is no effect of taxes, but a positive and significant effect of the youth smoking rate, in line with the idea that youth smoking may be correlated with other health behaviors. Second, in Appendix Section B, I consider whether the effect of taxes on suicides may be explained mechanically (via "competing risks") due to the reduction in smoking deaths. Using a back of the envelope approach, I conclude that the competing risks explanation is unlikely because the effect on suicides is quite large compared to the population frequency.

In sum, there is consistent evidence higher peer smoking and cigarette taxes in youth are associated with a lower suicide rate in middle age for native-born individuals, concentrated among men, but effects on poisonings and smoking-related mortality are mixed.

5 Conclusion

Government regulation to restrict sales of cigarettes, via taxes and bans, alongside public education campaigns, have caused a precipitous decline in smoking over the last half century and, in doing so, saved millions of lives and resulted in numerous other benefits, including increases in infant and child health. In this paper, I explore the hypothesis that the reduction in smoking had the unintended consequence of leaving some individuals more vulnerable to the effects of mental illness. For these

[37] Disaggregated results available on request.

individuals, the nicotine in cigarettes serves as a form of "self-medication" for mood disorders, in lieu of a formal, or less harmful, treatment.

I provide two different empirical analyses to test this hypothesis. First, I use data collected during the Lung Health Study, which randomly assigned a smoking cessation treatment and then followed participants for five years. I then show that, for men, assignment to the treatment group is associated with decreases in mental health (measured by an index compiling indicators for moodiness, nervousness, anxiety and depression) and increased use of anxiolytics across the five annual follow-up interviews. By contrast, effects are insignificant or indicative of mental health improvements for women.

Second, I investigate whether variation in the propensity to smoke during adulthood changes the distribution of mortality outcomes associated with mental illness (suicides and poisonings) and smoking-related disease (cancer, respiratory disease, and heart disease). For this purpose, I combine data consisting of the universe of death certificates in the U.S. for 1990-2004 with Gallup polls, which are public opinion surveys conducted since the 1930s that contain a uniform question on smoking behavior. To identify the causal effects of smoking, I utilize cigarette tax rates at age 18 matched to state of birth, as well as average youth smoking rates among one's peers (defined by demographic group and cohort). The years and cohorts for which I observe both predictor and outcome variables limits my focus to individuals in middle age.

I first establish that cigarette tax rates and the youth smoking rate are predictive of smoking rates in middle age, although effects are larger and more significant among men for taxes. Next, estimating the reduced form specification of mortality on taxes, I find that that a \$0.01 (1970\$, or \$0.06 in 2016\$) increase in taxes at age 18 increases suicides in middle age by 0.0014 per thousand (0.88% of the mean). Findings from a regression of suicide on youth smoking among peers at age 18 implies a similarly sized results when scaled to effects on smoking. For both predictors, effects are larger and more significant among men than women.

The results of my analysis imply the importance of combining policy to regulate harmful substances such as cigarettes with efforts to address and treat the underlying mental or physiological disorders that they may be used for as "self-medication." This insight may be applied in the case of the current opioid epidemic. Recent work has found that states that legalized medical marijuana, which has analgesic properties, have lower rates of opioid usage in certain populations, providing additional evidence that regulation targeting a particular substance can have spillover effects on utilization of another (whether formal or informal) (Bradford et al., 2018).

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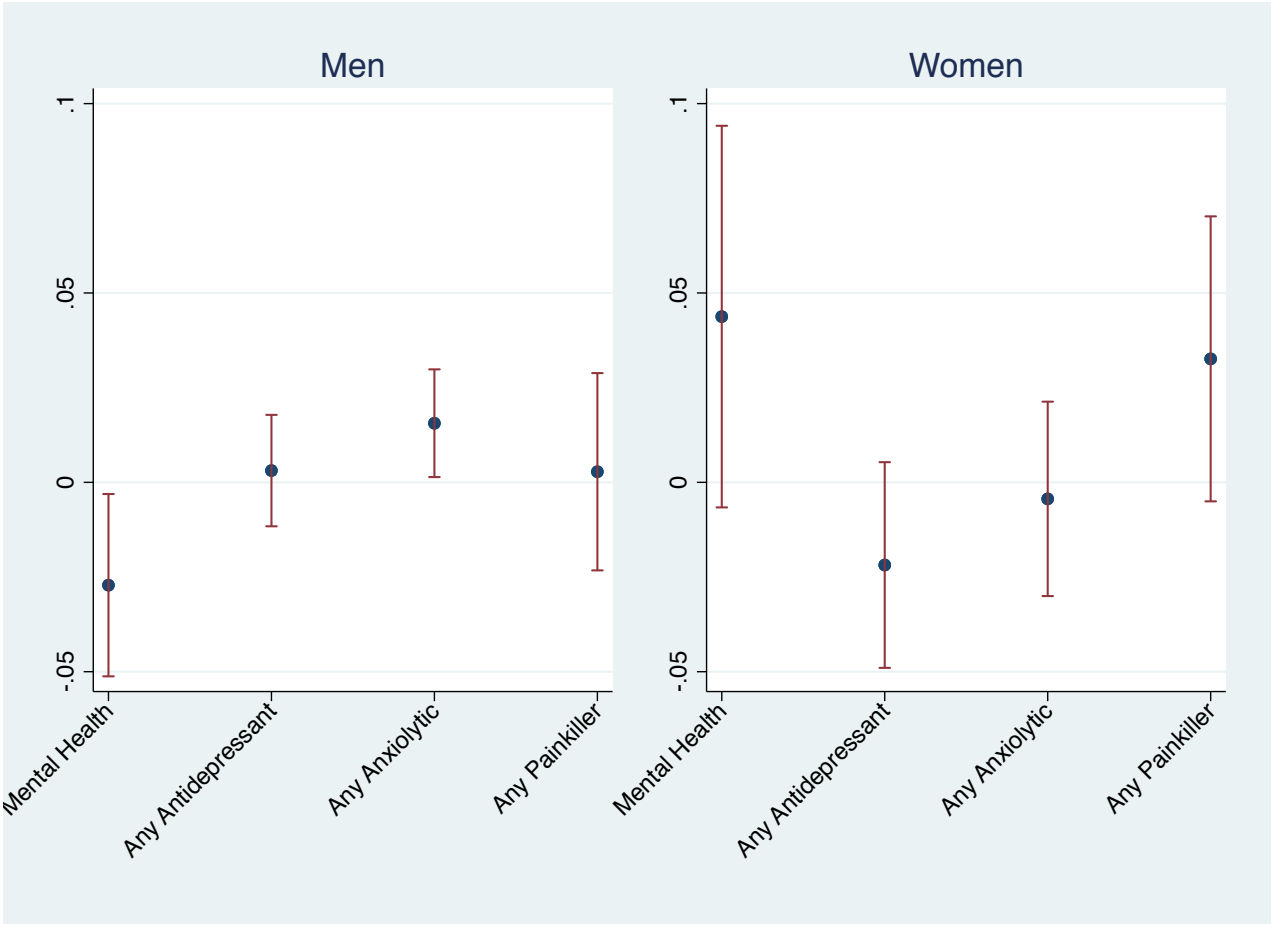
Figure 1: Mental Health Outcomes, 3-5 Yrs. after Anti-Smoking Intervention



Notes: This graph displays coefficients and confidence intervals corresponding to the estimates shown in Tables 2 and 3. Please see footnotes to Tables 2 and 3 for detail on the estimating strategy, sample construction, and variable definitions.

Figure 2: Smoke Cigarettes in Past Week, Ages 16-25, Gallup Polls

Table 1: Effect on Long Term Smoking, Lung Health Study

	Sustained Quit, All 5 Follow-Ups		...And No Add'l Nicotine	
	(1)	(2)	(3)	(4)
Treatment Group	0.1809	0.1422	0.1194	0.0716
	(0.0108)	(0.0135)	(0.0098)	(0.0116)
	[0.0000]	[0.0000]	[0.0000]	[0.0000]
Obs.	3,601	2,137	3,601	2,137
Sample	Men	Women	Men	Women

Notes: Each column reports the results of a separate regression, estimating Eq. 1. In addition to the coefficient estimate, the standard error is reported in parentheses, and the associated p-value is reported in brackets. For detail on sample construction, please see Tables 2-3. The sample is the Lung Health Study. "Sustained Quit..." indicates that the individual quit smoking cigarettes in the first year of the study and sustained cessation until year 5. Cessation was validated through medical testing. "No Add'l Nicotine" indicates that the individual additionally reports that they have not used nicotine gum, chewing tobacco or snuff in the last 24 (years 1-4) or 48 (year 5) hours. Standard errors are robust.

Table 2: Mental Health Outcomes after Anti-Smoking Intervention, Men, Ages 35-60

	Avg. Mental Hlth Index		Any Antidepressant		Any Anxiolytic		Any Painkiller	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment Group	-0.0306	-0.0271	0.0054	0.0031	0.0153	0.0156	0.0100	0.0028
	(0.0118)	(0.0123)	(0.0081)	(0.0075)	(0.0083)	(0.0073)	(0.0147)	(0.0133)
	[0.0095]	[0.0271]	[0.5078]	[0.6789]	[0.0660]	[0.0315]	[0.4964]	[0.8329]
Obs.	3,103	3,225	3,657	3,579	3,657	3,579	3,657	3,579
Outcome is for	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5

Notes: The sample is the Lung Health Study, restricted to male participants. Each observation is a participant. Each coefficient, standard error (parentheses), p-value (brackets) are from a separate regression, estimating Eq. 1. The mental health outcomes, described in the notes to Appendix Table 1, represent an average over years 1-5, or years 3-5 of the annual follow-up interviews after the anti-smoking intervention program. No additional controls are included. Robust standard errors are estimated.

Table 3: Mental Health Outcomes following Anti-Smoking Intervention, Women, Ages 35-60

	Avg. Mental Hlth Index		Any Antidepressant		Any Anxiolytic		Any Painkiller	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment Group	0.0122	0.0438	-0.0132	-0.0218	0.0018	-0.0044	0.0337	0.0326
	(0.0214)	(0.0257)	(0.0151)	(0.0139)	(0.0145)	(0.0131)	(0.0208)	(0.0192)
	[0.5676]	[0.0889]	[0.3824]	[0.1152]	[0.8997]	[0.7390]	[0.1060]	[0.0895]
Obs.	1,917	1,972	2,163	2,132	2,163	2,132	2,163	2,132
Outcome is for	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5	Yrs. 1-5	Yrs. 3-5

Notes: Please see notes to Table 2. The sample is the Lung Health Study, restricted to female participants.

Table 4: Predictors of Current Smoking in Adulthood

	(1)	(2)	(3)	(4)	(5)	(6)
Tax per Pack at 18	-0.7987	-1.6157	-0.0135			
(1970$)	(0.4639)	(0.7857)	(0.5662)			
	[0.0915]	[0.0452]	[0.9811]			
Youth Smoking Rate at 18				0.1765	0.1477	0.0702
				(0.0612)	(0.0894)	(0.0940)
				[0.0059]	[0.1050]	[0.4588]
N	3,651	1,821	1,830	4,325	2,145	2,180
Mean, Dep. Var.	0.2580	0.2674	0.2486	0.2550	0.2718	0.2385
Sample	All	Men	Women	All	Men	Women
Ages	35-59	35-59	35-59	35-61	35-61	35-61

Notes: Each column reports the coefficient, standard error (in parentheses), and p-value (in brackets) from a separate regression, estimating Eq. 2. The sample consists of individuals in the indicated age ranges surveyed in one of 15 nationally representative Gallup Polls conducted between 1987 and 2011. The outcome is whether the surveyed adult answers yes to the following question: "Have you, yourself, smoked cigarettes in past week?" "Youth smoking rate" refers to average current smoking for ages 16-25 per race (black vs non-black), Census Region, and sex, for the 5-year interval in which the individual is 18, calculated separately from 15 nationally representative Gallup polls, from 1969-1996. Cigarette taxes are the sum of state and federal taxes, in dollars (normalized to 1970$), merged into the sample by state of current residence and year in which the individual was 18 (1970-1987). These tax data are taken from from Orzechowski and Walker (2011), which lists annual tax per pack for each state from 1970-2011. All regressions include FE for year of birth, interview year, and the interaction of sex, black race, and state of residence. Standard errors are clustered at the state of residence level.

Table 5: Smoking and Mortality Rates (x1000)

	(1) Suicides	(2) Poisonings	(3) Smoking-Related	(4) Suicides	(5) Poisonings	(6) Smoking-Related
Tax per Pack at 18	0.1437	0.1366	-0.1300			
(1970$)	(0.0457)	(0.0930)	(0.3589)			
	[0.0028]	[0.1480]	[0.7188]			
Youth Smoking Rate				-0.0121	0.0182	0.3986
				(0.0034)	(0.0137)	(0.0347)
				[0.0009]	[0.1917]	[0.0000]
N (cells)	32,495	32,495	32,495	33,628	33,628	33,628
Sum of Weight (000s)	611,351	611,351	611,351	700,778	700,778	700,778
Mean, Dep. Var.	0.1635	0.1349	0.7029	0.1632	0.1277	0.7821
Ages	35-52	35-52	35-52	35-54	35-54	35-54

Notes: Each column reports the coefficient, standard error (in parentheses), and p-value (in brackets) from a separate regression, estimating Eq. 3. Each observation is an age-birth cohort-birth state-sex-black race cell. The outcome is the share of native-born individuals in that cell that die of the indicated cause. The numerator of this outcome measure is constructed from the Vital Statistics Multiple Cause of Death Data, 1990-2004, from the National Center for Health Statistics. The denominator (population per cell) is derived from the 1990 Census. For additional detail on the independent variables, please see Table 4 above. All regressions include FE for year of birth, death year and the interaction of sex, black race, and state of birth. Standard errors are clustered at the state of birth level. Observations are weighted by total Census person weights per cell (denominator of mortality rates).

Table 6: Smoking and Mortality Rates in Middle Age (x1000), Men

	(1) Suicides	(2) Poisonings	(3) Smoking-Related	(4) Suicides	(5) Poisonings	(6) Smoking-Related
Tax per Pack at 18	0.2388	0.2064	-0.1031			
(1970$)	(0.0929)	(0.1234)	(0.5378)			
	[0.0131]	[0.1007]	[0.8488]			
Youth Smoking Rate				-0.0252	0.0691	0.2153
				(0.0106)	(0.0383)	(0.1084)
				[0.0211]	[0.0773]	[0.0525]
N (cells)	16,208	16,208	16,208	17,029	17,029	17,029
Sum of Weight (000s)	303,059	303,059	303,059	347,308	347,308	347,308
Mean, Dep. Var.	0.2547	0.1902	0.9115	0.2537	0.1805	1.0329
Ages	35-52	35-52	35-52	35-54	35-54	35-54

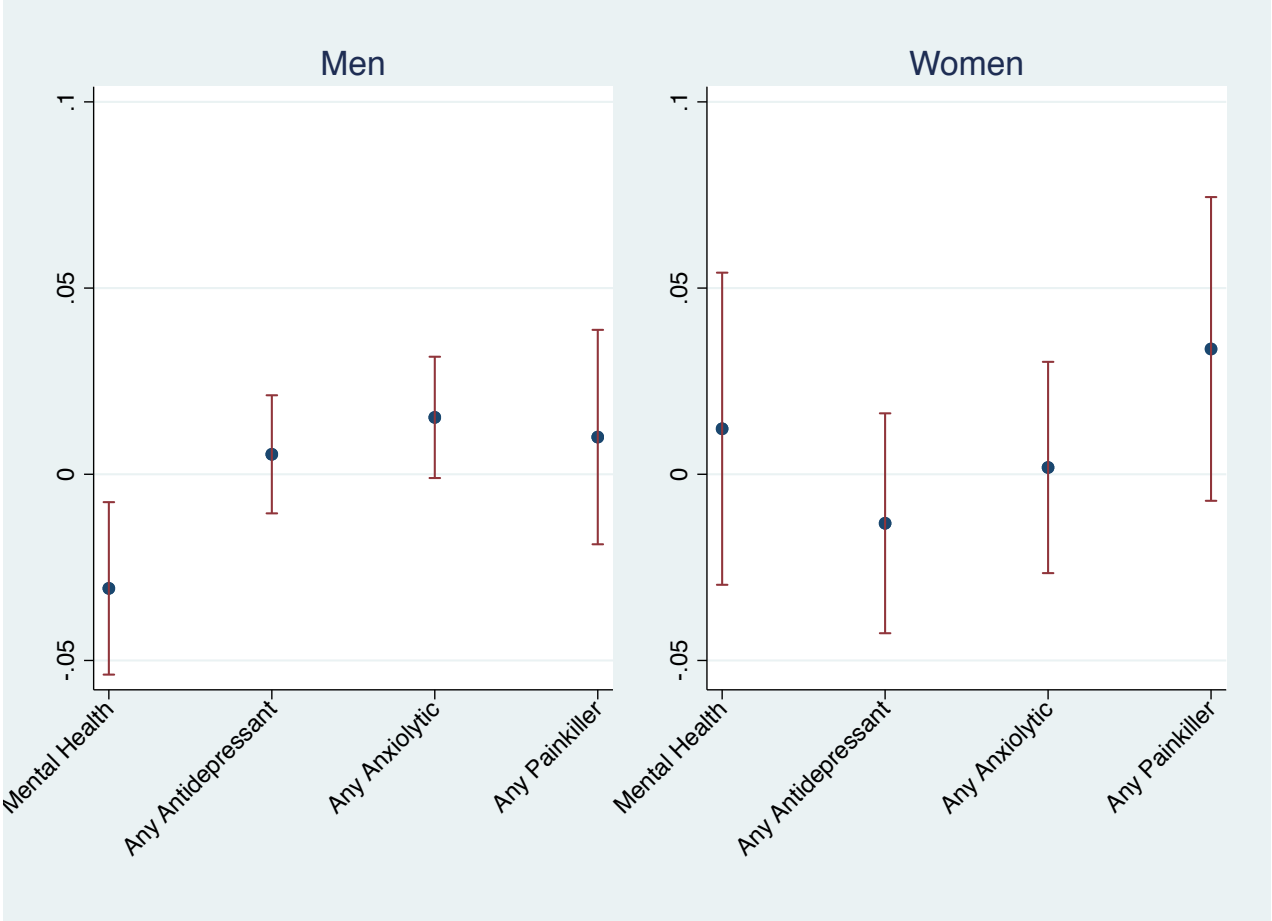
Notes: The table above replicates Table 6 for the subsample of men. Please see the notes to Table 5 for additional detail.

Table 7: Smoking and Mortality Rates in Middle Age (x1000), Women

	(1) Suicides	(2) Poisonings	(3) Smoking-Related	(4) Suicides	(5) Poisonings	(6) Smoking-Related
Tax per Pack at 18	0.0504	0.0681	-0.1647			
(1970$)	(0.0408)	(0.0787)	(0.2351)			
	[0.2219]	[0.3907]	[0.4869]			
Youth Smoking Rate				0.0003	0.0033	-0.0307
				(0.0027)	(0.0058)	(0.0412)
				[0.9268]	[0.5788]	[0.4597]
N (cells)	16,287	16,287	16,287	16,599	16,599	16,599
Sum of Weight (000s)	308,292	308,292	308,292	353,470	353,470	353,470
Mean, Dep. Var.	0.0738	0.0806	0.4979	0.0743	0.0758	0.5357
Ages	35-52	35-52	35-52	35-54	35-54	35-54

Notes: The table above replicates Table 5 for the subsample of women. Please see the notes to Table 5 for additional detail.

Appendix Figure 1: Mental Health, 1-5 Yrs. after Anti-Smoking Intervention



Please see notes to Tables 2 and 3.

Appendix Table 1: Balanced Sample Test: Baseline Characteristics, Lung Health Study

	Control	Treatment	P-Val
Age	48.4846	48.4369	0.8004
Male	0.6240	0.6385	0.2783
no HS Diploma	0.1239	0.1191	0.6008
HS Diploma	0.3018	0.2902	0.3595
College or Trade School	0.5743	0.5906	0.2315
Cigs. per Day	31.3599	31.0988	0.4623
Age, First Cigarette	17.4360	17.5535	0.2721
Irritability, Past 4 Mos.	0.0288	0.0290	0.9625
Insomnia, Past 4 Mos.	0.0347	0.0356	0.8479
Mood Changes, Past 4 Mos.	0.0206	0.0173	0.3833
Nervous, Past 4 Mos.	0.0275	0.0280	0.9168
Psych. Problems, Past 4 Mos.	0.0041	0.0015	0.1037
Mental Health Index	0.0040	-0.0079	0.4925
Anti-Depress., Past Month	0.0145	0.0173	0.4147
Anxiolytic, Past Month	0.0263	0.0234	0.5140
Painkiller, Past Month	0.0770	0.0662	0.1344
Observations	3923	1964	5887

Notes: Sample consists of all individuals in the Lung Health Study (one observation per person). Each outcome above is measured during one of three screening interviews that occurred before randomization. "no HS Diploma" corresponds to LHS codes: "less than or equal to 8th grade", "trade school instead of high school", and "some high school." "HS Diploma" corresponds to "High School Graduate." "College Degree or Trade School" indicates "trade school after high school or some college", "bachelor's degree," "some graduate education", or "graduate degree." Each "mood" indicator (e.g., "irritability") corresponds to the following question: "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," and is coded "1" if the respondent indicates "Severe." Prescription drug utilization is coded using answers to the following question: "Have you taken any other prescription drugs (not mentioned above) in the past 12 months." Self-reported answers are matched to therapeutic categories using Google searches and data from the Medical Expenditure Panel Survey (MEPS).

Appendix Table 2: Summary Statistics, Gallup Poll Sample

	Mean	Mean
Age	42.5429	41.8904
Black	0.0849	0.0893
Male	0.4960	0.4988
Lives: New England	0.0592	0.0619
Lives: Middle Atlantic	0.1533	0.1572
Lives: East North Central	0.1797	0.1786
Lives: West North Central	0.1662	0.1594
Lives: South Atlantic	0.0675	0.0663
Lives: East South Central	0.0606	0.0583
Lives: West South Central	0.0966	0.0981
Lives: Mountain	0.0770	0.0803
Lives: Pacific	0.1399	0.1400
Smoke, Last Week	0.2550	0.2580
Youth Smoking Rate at 18	0.4295	
Tax per Pack at 18 (1970$)		0.1504
Price per Pack (1970$)		0.3648
Observations	4325	3651

Notes: Please see notes to Table 4.

Appendix Table 3: Summary Statistics, Mortality Samples, 1990-2004

	Mean	Mean
Age	41.6041	40.8332
Black	0.4085	0.4821
Male	0.5064	0.4988
Born: New England	0.0799	0.1093
Born: Middle Atlantic	0.0696	0.0609
Born: East North Central	0.1160	0.1016
Born: West North Central	0.1489	0.1314
Born: South Atlantic	0.1713	0.1828
Born: East South Central	0.0928	0.0812
Born: West South Central	0.0880	0.0812
Born: Mountain	0.1211	0.1526
Born: Pacific	0.1125	0.0988
Suicide Rate per Pop. (x1000)	0.1526	0.1460
Poisoning Rate per Pop. (x1000)	0.1794	0.1953
Smoking-Related Deaths per Pop. (x1000)	1.0706	0.9592
Heart Disease Death Rate per Pop. (x1000)	0.7091	0.6442
Cancer Death Rate per Pop. (x1000)	0.2839	0.2379
Resp. Disease Death Rate per Pop. (x1000)	0.0777	0.0772
Youth Smoking Rate	0.4763	
Tax per Pack at 18 (1970$)		0.1447
Observations	33628	32495

Notes: Please see notes to Table 5.

Appendix Table 4: Predictors of Current Smoking in Middle Age, add Time Trends

	(1)	(2)	(3)	(4)	(5)	(6)
Tax per Pack at 18	-1.8787	-3.1035	-0.8268			
(1970$)	(0.6094)	(1.1134)	(0.9498)			
	[0.0034]	[0.0076]	[0.3883]			
Youth Smoking Rate at 18				0.2032	0.1273	0.1169
				(0.0615)	(0.1163)	(0.0963)
				[0.0018]	[0.2791]	[0.2308]
N	3,651	1,821	1,830	4,325	2,145	2,180
Mean, Dep. Var.	0.2580	0.2674	0.2486	0.2550	0.2718	0.2385
Sample	All	Men	Women	All	Men	Women
Ages	35-59	35-59	35-59	35-61	35-61	35-61

Notes: This table replicates Table 4, adding state of residence-specific linear trends in birth cohort year. Please see notes to Table 4 additional detail.

Appendix Table 5: Smoking-Related Mortality in Middle Age (x1000) and Taxes

	(1) Heart Disease	(2) Cancer	(3) Respiratory Disease
Tax per Pack at 18	-0.0829	0.0185	-0.0655
(1970$)	(0.2513)	(0.1097)	(0.0320)
	[0.7429]	[0.8670]	[0.0461]
N (cells)	32,495	32,495	32,495
Sum of Weight (000s)	611,351	611,351	611,351
Mean, Dep. Var.	0.4589	0.1931	0.0509
Ages	35-52	35-52	35-52

Notes: The table above replicates Table 5 (Columns (1)-(3)) by disaggregated causes of smoking-related mortality. Please see notes to Table 5 for additional detail.

Appendix Table 6: Smoking and Mortality Rates in Middle Age (x1000), add Time Trends

	(1) Suicides	(2) Poisonings	(3) Smoking-Related	(4) Suicides	(5) Poisonings	(6) Smoking-Related
Tax per Pack at 18	0.1452	0.0464	0.4499			
(1970$)	(0.0659)	(0.0664)	(0.1898)			
	[0.0321]	[0.4875]	[0.0217]			
Youth Smoking Rate				-0.0132	0.0117	0.4070
				(0.0034)	(0.0124)	(0.0367)
				[0.0003]	[0.3495]	[0.0000]
N (cells)	32,495	32,495	32,495	33,628	33,628	33,628
Sum of Weight (000s)	611,351	611,351	611,351	700,778	700,778	700,778
Mean, Dep. Var.	0.1635	0.1349	0.7029	0.1632	0.1277	0.7821
Ages	35-52	35-52	35-52	35-54	35-54	35-54

Notes: The table above replicates Table 5, adding state of birth-specific linear time trends in birth cohort year as controls. Please see notes to Table 5 for additional detail.

Appendix Table 7: Average Tax in Youth and Mortality Rates in Middle Age (x1000)

	(1) Suicides	(2) Poisonings	(3) Smoking-Related
Average Cig. Tax, Ages 15-18, 1970$	0.2366	0.2590	-0.3459
	(0.0536)	(0.1179)	(0.4193)
	[0.0001]	[0.0327]	[0.4134]
N (cells)	32,495	32,495	32,495
Sum of Weight (000s)	611,351	611,351	611,351
Mean, Dep. Var.	0.1635	0.1349	0.7029
Ages	35-52	35-52	35-52

Notes: The table above replicates Table 5, replacing taxes at 18 with average taxes at ages 15-18, based on the individual's birth cohort and state of birth, as reported on the death certificate. Please see notes to Table 5 for additional detail.

Appendix Table 8: Smoking and Mortality Rates, no Demog. Controls (x1000)

	(1)	(2)	(3)	(4)	(5)	(6)
	Other Deaths	Poisonings	Smoking-Related	Other Deaths	Poisonings	Smoking-Related
Tax per Pack at 18	0.1500	0.1334	-0.2014			
(1970$)	(0.0477)	(0.1010)	(0.3718)			
	[0.0028]	[0.1927]	[0.5905]			
Youth Smoking Rate				0.0862	0.1347	1.1052
				(0.0349)	(0.0286)	(0.0664)
				[0.0168]	[0.0000]	[0.0000]
N (cells)	32,495	32,495	32,495	33,628	33,628	33,628
Sum of Weight (000s)	611,351	611,351	611,351	700,778	700,778	700,778
Mean, Dep. Var.	0.1635	0.1349	0.7029	0.1632	0.1277	0.7821
Ages	35-52	35-52	35-52	35-54	35-54	35-54

Notes: This table replicates Table 5, removing controls for sex and black race. Please see notes to Table 5.

Appendix Section A Steps to Identify Drugs from LHS

I standardize the Google string results, removing special characters and spaces and putting all words in upper case letters. Effort was also made to check manually that these steps capture all of the relevant drugs.

- Anti-depressants: 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.
- Anxiolytic: Google sidebar description includes the words: "ANXIOLYTIC", or the therapeutic codes from the MEPS are: 67,69
- Analgesic/Painkiller: Google sidebar description includes the words: "NARCOTIC", "ANALGESIC", or "NONSTEROIDAL ANTI-INFLAMMATORY DRUG"

Appendix Section B Competing Risks

In this section, I consider whether decreases in smoking-related deaths in response to higher cigarette taxes at age 18 may explain the increase in suicides I find in Table 5 through a mechanical effect ("competing risks") because some people who would have died of smoking-related mortality causes now die of suicide. The result in Table 5 implies that a $1.00 increase in the cigarette tax rate decreases smoking-related deaths in middle age by -0.1300 per thousand individuals (95% confidence interval: -0.8482 to 0.5883). To be conservative, I assume the lower bound of the confidence interval, -0.8482, represents the true effect. Taking a back of the envelope approach, the suicide rate in middle age among these additional 0.8482 per thousand individuals in middle age should be approximately equal to 0.1635 per thousand (the sample mean suicide rate shown in Table 5). Then, the mechanical increase in suicides due to fewer smoking deaths should be the product of the two rates, or 0.00014 suicides per thousand, which is much smaller (about 1026x) than the coefficient I estimate of 0.1437 per thousand (95% CI: 0.0521, 0.2352). In other words, it would have to be that, of the individuals who did not die of smoking-related causes in middle age as a result of higher youth taxes, $\frac{0.1437}{0.8482} = 16.94\%$ of them ended up instead committing suicide by the end of middle age. Given that suicide is a rare event, this exercise suggests that the increase in suicides is not a mechanical effect due to the decrease in smoking deaths.