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The Self-Medication Hypothesis: Evidence from Terrorism and Cigarette Accessibility

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Abstract

We use single equation and system instrumental variable models to explore if individuals smoke during times of stress (the *motivation effect*) and if they are successful in self-medicating short-term stress (the *self-medication effect*). Short-term stress is a powerful motivator of smoking, and the decision to smoke could trigger biological feedback that immediately reduces short-term stress. We use data on self-reported smoking and stress from 240,388 current and former smokers. We instrument short-term stress with temporal distance from September 11, 2001 (using date of interview). We instrument smoking with cigarette accessibility measures of cigarette price changes and distance to state borders. In the absence of accounting for endogeneity, we find that smoking is associated with increases in short-term stress. However, when we account for endogeneity we find no evidence of smoking affecting short-term stress. We do find a consistent positive effect of short-term stress on smoking.

JEL Classification Numbers: C26, C36, I19

Keywords: stress, smoking, self-medication

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I Introduction

Lowering rates of stress and smoking are important behavioral health priorities. High stress can result in serious health problems including insomnia, muscle pain, high blood pressure, a weakened immune system, heart disease, depression, obesity, and can exacerbate existing illnesses. Americans believe that persistent, high stress is unhealthy and consistently report stress levels that are higher than what they believe to be healthy (American Psychological Association, 2013). Meanwhile, cigarette use accounts for more than 480,000 deaths (including deaths from secondhand smoke), or one of every five deaths, in the United States each year (U.S. DHHS, 2014).

Theory and evidence suggest a linkage between stress and smoking. The ability of smoking to improve mood state in the short-term illustrates the self-medication hypothesis. This theory is rooted in neuroscience literature and finds that individuals are able to positively alter negative subjective beliefs through the use of tobacco or other substances. At least one economics study, Barnes and Smith (2009), has used the self-medication hypothesis to explain a contradiction to the rational addiction theory.[1] Biochemically, nicotine use increases dopamine levels, and this neurotransmitter is classically associated with altering mood state (Brody et al., 2004; Volkow et al., 2004). However, long-term exposure may cause fewer dopamine receptors that may necessitate the use of more nicotine to experience the same "high" (Doe et al., 2009).

This biochemical process can motivate nicotine use during times of high stress to the extent that individuals perceive smoking to be a method of stress reduction. Perceptions of smoking as a stress reduction device can be formed from past usage and advertisements. Smokers have cited stress reduction as their primary motive for smoking (McEwen et al., 2008). We will hitherto refer to the biochemical process of smoking on stress as the *self-*

[1]The authors found that future negative income shocks have a positive effect on current cigarette consumption. They argue that this effect is positive because individuals are self-medicating an expectation of a future income shock, in contrast to the decrease in smoking that would be predicted by the rational addiction theory assuming that cigarettes are normal goods.

medication effect and will refer to the motivation of individuals to use nicotine during times of high stress, likely for perceived or actual self-medication, as the *motivation effect*.

Several studies exploit plausibly exogenous variation in stress to examine what effect this has on cigarette smoking, or the *self-medication effect*. Siahpush and Carlin (2006) exploit longitudinal data to find that higher financial stress is associated with smokers being less likely to quit and former smokers being more likely to relapse one year later. A study by Barnes and Smith (2009) uses longitudinal data in an instrumental variable (IV) model, exploiting geographic variation in local labor market conditions as a source of exogenous variation, finding that a 1 percent increase in the probability of becoming unemployed causes an individual to be 2.4 percent more likely to continue smoking. Cotti, Dunn and Tefft (2014) finds that large negative stock market shocks are widely associated with increased cigarette consumption and purchases, independent of other macroeconomic labor conditions. Finally, Pesko (2014) finds that an increase in stress following the 9/11 terrorist attack, which persisted for one quarter before returning to baseline, accounted for one million former smokers relapsing back into smoking in the United States.[2]

Could former smokers rationally relapse to experience short-term stress reduction gains during periods of acute stress? The ideal way to answer this question would be to conduct a randomized controlled trial of former smokers that experience the same stressor, and randomly provide the treatment of smoking to only half of the respondents. The difference in stress levels between the two groups would be the causal effect of smoking on stress. While appealing from a research standpoint, we thankfully cannot conduct such an experiment due to ethical concerns. The next best approach to explore this question may be to conduct a natural experiment by studying variation in stress when people relapse back into smoking, using a plausibly exogenous component of smoking. This is what we attempt to do in this paper by using 9/11 as the exogenous shock to short-term stress and cigarette accessibility

[2]Pesko (2014) found no evidence that attempts to quit smoking decreased in the period after 9/11, suggesting that the increase in smoking was mostly from smoking relapse. This study did not explore the reverse impact that smoking had on stress, which is the goal of the current study.

variables as the exogenous shock to smoking.

Psychologists have long been interested in the relationship between smoking and stress (Parrott, 1995, 1998). Likely due to ethical concerns, psychological studies have only used variation in how stress changes when people try to quit smoking (rather than relapse back into smoking). These studies haven't attempted to use exogenous variation in smoking cessation and the samples in which estimates were derived are small convenience samples. Perhaps the strongest of these studies monitored self-perceived stress prior to quitting at 1, 3 and 6 months post-cessation for 260 subjects interested in quitting smoking. Subjects who failed to quit, or stopped for only a brief period, reported higher levels of stress at each time point, whereas those who remained abstinent for the whole 6-month period reported a steady decrease in stress over time (Cohen and Lichtenstein, 1990). While these results suggest the opposite of self-medication, it is unclear if smoking cessation caused lower stress, or if lower stress caused smoking cessation (or some combination of the two). We attempt to answer this question in our current study within the context of a nationally-representative sample of individuals by exploiting plausibly exogenous changes in smoking and stress. To explore the effect on stress, we also heavily rely on variation from smoking relapse rather than using non-experimental variation from quitting smoking.

In investigating the *motivation effect*, we find evidence from single equation IV models that short-term stress increases smoking. This suggests that individuals are motivated to smoke during times of high short-term stress to self-medicate higher stress. Was this self-medication strategy successful? To answer this question we first estimate the impact of smoking on short-term stress using a regression model. These results suggest that smoking actually *increases* short-term stress, and that, apparently, attempting to self-medicate by smoking is counter-productive. These results are at odds with the theory of self-medication and qualitative responses from smokers indicating that stress reduction is an important component in why they smoke. In exploring this contradiction further, we find that the positive *self-medication effect* is substantially attenuated, and becomes insignificant, when

we account for omitted variable bias using a two-stage least squares (2SLS) IV model and when we account for feedback from short-term stress onto smoking using a multi-equation, simultaneous IV model. In these specifications, smoking appears to have no effect on short-term stress. Therefore, while we do not find empirical evidence to support the self-medication hypothesis, our results do suggest that failure to account for endogeneity may result in a spurious positive estimate of the association of smoking on short-term stress.

This paper attempts to estimate the *motivation effect* and the *self-medication effect* using exogenous variation in terrorism and cigarette accessibility. The remainder of the paper is organized as follows. Section II discusses the data, Section III articulates our empirical strategy, Section IV presents the results and shows evidence that they are causal in nature, and Section V concludes.

II Data

We use survey data for the continental United States from the Behavioral Risk Factor Surveillance System (BRFSS). State health departments and the Centers for Disease Control and Prevention (CDC) collect these cross-sectional data on risky personal health behaviors via landline telephone surveys of individuals aged 18 years and older. The data are representative of the non-institutionalized population at national and state levels. State health departments collect the data throughout the course of the full year rather than in particular months. The data identify the respondents' state, county,[3] and date of response as well as a variety of socio-demographic controls including gender, race/ethnicity, age, education, employment/labor force participation, marital status, and income.

We use data from 1999, 2000, and 2001. We do not use data from 2002 because the impact of terrorism on stress declined quickly after 9/11 (Pesko, 2014) and because reports of stress became part of an optional module only completed by 23 states in 2002. We remove

[3]County was missing for 24.6% of respondents. Of these, 97% were suppressed due to the county having fewer than 10,000 residents.

observations with dates between 9/11/2001 and 10/1/2001 from our regression analyses to more cleanly separate pre-9/11 and post-9/11 effects for stress and smoking, both of which are answered over the past 30 days.

As a proxy for stress, survey respondents are asked a standard question of recent emotional and mental distress: "Now thinking about your mental health, which includes stress, depression, and problems with emotions, for how many days during the past 30 days was your mental health not good?" This measure of stress is deliberately broad and could reflect changes in perceived background risk, financial risk, and longevity expectations, among other things. These data are heavily skewed, with 64.8% of individuals reporting having 0 days of stress and 5.9% reporting having 30 days of stress. The remaining 29.3% report integer values between 1 and 29, and we observed that these integer values were bunched in multiples of 5. For example, 1.7% of respondents reported 4 days of stress, 0.4% reported 6 days of stress, and 3.8% reported 5 days of stress. For this reason, we binned these responses into a new ordinal variable taking the value of 0 for 0 days of stress, 1 for 1–5 days of stress, 2 for 6–10 days of stress, and onwards until 6 is used to represent 25–30 days of stress.

Individuals self-reporting their stress levels over the past 30 days may answer disproportionate to the stress they have experienced in the most recent days. We see evidence of this in the immediate aftermath of 9/11. In the five days before 9/11, individuals reported on average 3.52 days of stress. In five day intervals after 9/11 for the first 30 days, the peak stress level was reported during days 5-10 at 3.96 days of stress. *A priori*, we would have expected this peak to occur at 25-30 days of stress as that would maximize the number of post-9/11 days in which people could report elevated 9/11-induced stress. This provides evidence that the 30 day stress question is actually measuring stress levels over a shorter period of time than 30 days, which is helpful for exploring the effect that smoking has on short-term stress reduction.

For smoking, survey respondents are asked if they have smoked 100 or more cigarettes in their lifetime. If so, we include them in our analysis. These individuals are then asked

if they have smoked over the past 30 days. If the individual answers yes then we classify them as a current smoker, and if they answer no then they are classified as a former smoker. We restrict our sample to current and former smokers ("lifetime smokers") because of the bidirectional flows that occur between these groups compared to the unidirectional flow of newly initiated smokers entering into lifetime smoking.

Cigarette price data from the *Tax Burden on Tobacco* (Orzechowski and Walker, 2009) are used in the analysis. These data are collected annually through mail surveys of tobacco distributors, and provide a state-level weighted price average for a pack of 20 cigarettes from pack, carton, and machine sales of both brand and generic cigarettes. These prices include federal and state excise taxes, as well as any delayed price changes from the Master Settlement Agreement signed in November of 1998. We transform the annual data to monthly data using the date of cigarette excise tax changes, assuming a unitary pass-through rate from taxes to prices. All monetary values were deflated to 2001 dollars using the US consumer price index, city average.

[Insert Table 1]

Summary statistics for the population-weighted data are reported in Table 1. In the sample, 48% of the population are current smokers and 52% of the population are former smokers. On average, individuals experienced 3.83 days of stress over the past 30 days (the binned stress variable averages 0.86 on a scale of 0–6).

III Empirical Framework

In this paper, we attempt to unravel the causal relationships between stress and smoking through the *motivation effect* and *self-medication effect*. This is performed by using cross-sectional data on mental health and smoking status and exploiting exogenous variation from terrorism and cigarette accessibility. We also demonstrate how the interpretation changes

depending on estimating the equations without instrumentation, using single equation IV models, and using simultaneous IV models.

We first explore the associations between stress and smoking. To explore these associations, we estimate two separate equations with stress and smoking prevalence interchanged as dependent and independent variables.

$$\begin{aligned} smoke_{isctm} &= \alpha + \beta_1 stress_{istm} + \beta_2 cig\_access_{sct} + \beta_3 \phi_{istm} + \\ &\quad \beta_4 unemployment_{st} + \lambda_s + \lambda_m + \lambda_t + \epsilon_{isctm} \end{aligned} \tag{1}$$

$$\begin{aligned} stress_{istm} &= \gamma + \delta_1 smoke_{istm} + \delta_2 terrorism_{st} + \delta_3 \phi_{istm} + \\ &\quad \delta_4 unemployment_{st} + \theta_s + \theta_m + \theta_t + v_{istm} \end{aligned} \tag{2}$$

where $smoke_{isctm}$ is equal to 1 if individual i living in county c of state s at year t and month m has smoked in the past 30 days, or 0 if not. $stress_{istm}$ is equal to 0, ..., 6 depending on the "bin" of stress that individual i reports over the past 30 days. Equation (1) is estimated with probit and equation (2) with a regression model.

Each equation includes a unique set of identifying variables and various controls that could be correlated with both stress and smoking. ϕ_{istm} is a set of controls at the individual level: gender, race/ethnicity, household income, age, age squared, educational attainment, marital status, and employment status. The unemployment rate in state s at month t is also included because it may be correlated with both stress and smoking.[4] Finally, state, month, and year indicators allow us to exploit variation in smoking within each state, month, and year. Month indicators in particular remove seasonal effects of smoking. While we control for unobservable, time-invariant state-level characteristics in our base analysis, we also explore

[4]From equation (2), we observed a statistically-significant counter-cyclical association between the unemployment rate and stress. However, using (1), we did not observe an association between the unemployment rate and smoking. We also explored a possible wealth effect by including the daily closing value of the Dow Jones Industrial Average as a covariate in (1) and (2), and we found no evidence of an effect.

removing state indicators primarily to allow cross-state variation in cigarette accessibility. ϵ_{isctm} and ν_{istm} are error terms representing unobserved factors and random noise.

In equation (1), cig_access_{sct} is a unique set of cigarette accessibility identifying variables. Accessibility of cigarettes is influenced by opportunities to minimize prices of cigarettes, including purchasing cigarettes in border states that provide lower cigarette prices (Harding et al., 2012; Pesko et al., 2013; DeCicca et al., 2013). We include the county-level distance to the nearest state border to proxy opportunities to purchase cigarettes in border states,[5] which may provide a cheaper source of cigarettes. We find statistically-significant evidence that distance to a state border is inversely related to current smoking prevalence. Finally, we also used cigarette price changes in the state of residence from the prior month as a cigarette accessibility variable.

In equation (2), $terrorism_{st}$ is a unique set of variables for temporal distance from 9/11/2001, including post_t and days_post_911_t. The days after 9/11 variable allows the post-9/11 effect to respond linearly over time.[6] Previous research has suggested that increases in stress were strongly associated with 9/11, (Schlenger et al., 2002; Schuster et al., 2001; Pesko, 2014). In Figure 1, we show the mean levels of stress for the 23 states in which stress is collected in year 2002, which provides visual evidence of the pronounced increase in stress shortly after 9/11, and how quickly it returns to baseline.

[Insert Figure 1]

Several limitations are evident in equations (1) and (2). First, they make no attempt to correct for potential unobserved omitted variables which may influence both smoking and stress. Examples include measurement error, genetic factors, and social factors. Second, stress may be jointly determined with smoking through the *motivation effect* and *self-*

[5]Distance to the nearest state is measured from the center of the county. In cases in which county of residence was missing (24.6% of respondents), we used the average distance within the state for all respondents. In a later analysis we show that results were insensitive to excluding individuals with missing county information.

[6]We also explored a quadratic specification of temporal distance, but did not find the quadratic term significantly different from zero.

medication effect. If smoking reduces stress, then the coefficient on stress in equation (1) will be biased towards the null. Additionally, the coefficient on smoking in equation (2) will be biased towards the null if individuals smoke during periods of high stress.

A partial solution to this problem is to use a single equation IV model, using cigarette accessibility and terrorism as identifying restrictions. A single equation approach may purge the influence of omitted variable bias and measurement error from the analysis provided that suitable instruments are found. This is an improvement over not correcting for the endogenous regressor, but it does not fully capture the potential simultaneity between smoking and stress.

In order to use cigarette accessibility and terrorism as identifying restrictions, the instruments need to be sufficiently strong and independent of the outcomes except through the endogenous regressors. In both situations we believe that sufficient exogeneity of our instruments is a plausible assumption, and we provide evidence justifying this.[7] Our instruments provide various levels of strength depending on if we remove cross-state variation in our instruments by including state indicators. While the strength of the instruments changes depending on their specification, the measured influence of stress on smoking and vice versa is fairly consistent. This alleviates concerns that weak instrumentation is responsible for spurious measurement.

We also address the potential simultaneity between stress and smoking in a systems estimation context by estimating a generalized structural equation model (GSEM).[8] The standard structural equation model (SEM) approach considers one or several continuous responses in a linear regression context. The GSEM approach generalizes SEM by allowing for

[7]We explored the use of other instruments that did not meet the necessary exogeneity criteria for a valid instrument. For terrorism, additional variables that we tested were spatial distance from the terrorist attack epicenters to county of residence (hypothesizing that individuals living closer to the epicenters of New York City and Washington D.C. experienced disproportionate increases in stress) and state-level mean stress for all respondents except the individual being interviewed (hypothesizing spillover effects of stress uncorrelated with smoking). These variables were associated with smoking independent of stress, so we abandoned efforts to use these variables as instruments. For cigarette accessibility, we also tested if the cigarette price level in the state of residence and an average cigarette price level in surrounding states were independent of the error distribution. They were not, so we also abandoned efforts to use these variables as instruments.

[8]See Rabe-Hesketh, Skrondal and Pickles (2004) for the analytical background of GSEM methods.

binary, ordinal, count or multinomial responses in the estimation process. Estimation may employ linear regression, probit, ordered probit, Poisson, and others. Estimation in a systems context allows for correlations among equations' errors to be modeled, and simultaneity to be considered in a full-information maximum likelihood context. Implementations of GSEM also allow for survey weights, robust and cluster-robust standard errors. Our standard errors are corrected for heteroskedasticity and clustering within states.[9] Estimation by GSEM is available in recent versions of Stata. We use the earlier implementation of these techniques, CMP, developed by Roodman (2011). Unlike Stata's GSEM, CMP supports the estimation of nonrecursive systems. An example of the use of this methodology in the health economics literature is Spearing, Connelly, Nghiem and Pobereskin (2012), who explore simultaneity between injury compensation and recovery time following whiplash injury.

IV Results

A Single Equation IV Results

We estimate equation (1) to provide an estimate of the association of stress on smoking, or the *motivation effect*, using single equation instrumental variable probit models. Column (1) excludes state indicator variables, while column (2) includes them. By excluding state indicators, this allows cross-state variation in our instruments rather than relying on only within-state variation in the instruments.

[Insert Table 2]

We find that the temporal distance measures are suitable instruments. Their strength in predicting stress varies depending on the inclusion of state indicator variables. While the individual coefficients for the post indicator variable and the days after 9/11 variable are positive and insignificant, the variables are jointly significant above the 95% level with an

[9] 466 unique geographic clusters are provided throughout the three years of data used in this analysis. These clusters are perfectly nested within states.

F-statistic of 4.15 for the first specification and 10.56 for the second. For the instruments to be valid, they must only affect smoking indirectly through affecting stress. To test this, we reestimate equation (1) including the temporal distance parameters as covariates. The terrorism parameters are jointly insignificant at the 5% level, suggesting that the instruments have no direct effect on smoking. We also perform an over-identification test using unweighted data.[10] We fail to reject the Amemiya–Lee–Newey over-identification test, which is equivalent to a Sargan test when the model is estimated with a two-step IV probit model (Baum et al., 2003, 2007). This suggests that the temporal distance measures are suitably exogenous to smoking.

The coefficient of the association of stress on smoking is 0.509 when state unobservable characteristics are excluded. It is attenuated to 0.308 when state indicator variables are included. These values are much larger than coefficients of 0.047 and 0.048, respectively, from ordinary binomial probit models which do not account for omitted variable bias, as reported at the foot of Table 2. This suggests that treating stress as an exogenous regressor rather than an endogenous regressor results in omitted variable bias that attenuates the effect of stress on smoking.

We reject the null hypothesis that the estimate for the correlation parameter, ρ, equals zero in the equation without state indicators. This means that the error terms in the structural equation and the reduced-form equation for the endogenous variable are meaningfully correlated, providing additional evidence that the single equation IV approach is preferable over the non-IV regression. However, we fail to reject $\rho = 0$ in the equation with state indicators.

In Table 3, we estimate equation (2) using 2SLS IV models. Column (1) excludes state indicator variables, while column (2) includes them. We attempt to provide an estimate of how smoking is associated with the 'binned' measure of stress, or the *self-medication* effect. We attempt to purge any omitted variable bias by instrumenting smoking with cigarette

[10]To the best of our knowledge, no over-identification test has been constructed for errors arising in the context of complex survey data.

price changes from the prior month and county-level distance to the nearest state border.

[Insert Table 3]

In specifications with and without state indicators, we find that the cigarette accessibility measures have no independent effect on stress except through smoking. This is demonstrated by their joint insignificance when added as predictors to equation (2), rendering them appropriately exogenous to be used as instruments. Using unweighted data, we also find evidence from a Sargan–Basmann test that the instruments are valid and that the structural equation is correctly specified. In terms of instrument strength, the instruments generate an F-statistic of 30.14 when state indicators are not included, and 3.99 when state indicators are included, both above the 95% level of confidence. The coefficients on the individual instruments are both negative and statistically significant, suggesting that greater distance to a state border and higher cigarette price changes are associated with lower rates of smoking.

In Table 3 we show that when we use these instruments, the resulting association of smoking on stress is insignificant, ranging from -0.102 to 0.029. In contrast, results from ordinary least squares regression models which treat smoking as exogenous suggest that smoking increases stress by roughly 0.20 points, as reported at the foot of the table.[11] Similar to results in Table 2 for the *motivation effect*, we again find evidence of substantial omitted variable bias. In this case, the bias drives a result contradicting the theory of self-medication, as our 2SLS results do not find support for the hypothesis that smoking reduces stress.

Full results for Tables 2 and 3 are available online.

B Generalized Structural Equation Model Results

In Table 4, we turn now to estimating the equations simultaneously to account for possible feedback between stress and smoking, in addition to accounting for omitted variable bias. We

[11] In an online appendix, we demonstrate that Table 3 results are not materially affected by removing individuals with unreported counties of residence.

continue to instrument using temporal distance terrorism variables and cigarette accessibility variables.

[Insert Table 4]

We use generalized structural equation modeling to estimate this system of equations. In columns 1 and 2, smoking is estimated using probit and stress using a regression model on the 'binned' stress measure. Coefficients can be directly compared with those reported in Table 2 (probit coefficients) and Table 3 (marginal effects). In columns 3 and 4 we explore the sensitivity of the results to estimating the binned stress measure using ordered probit rather than a regression model, combined with a probit estimator of smoking prevalence.

In all columns, the *motivation effect* coefficients are positive and statistically significant and the *self-medication effect* coefficients are insignificant. Estimates for the *motivation effect* and *self-medication effect* from columns 1 and 2 are similar to the IV model results in Table 2 and Table 3. This suggests that while omitted variable bias is a substantial concern, there is limited concern from feedback affecting an estimate of the causal relationship.

The estimate for the correlation parameter is statistically distinguishable from zero in the model without state indicators, suggesting that modeling the two effects as a system is structurally appropriate in this situation and an improvement from single equation models. However, the correlation parameter loses its significance when state indicators are added. In this situation, estimates of the *self-medication effect* are attenuated although they remain statistically significant.

One limitation of GSEM models is the limited number of diagnostic tests available. In particular, we are unaware of a method to test the exogeneity of the instruments in this framework. We report the test statistics available to us, in particular a joint test of the significance of the instruments.

V Conclusion

Individuals self-report that stress reduction is a primary reason why they smoke (McEwen et al., 2008), but the research to date has mostly suggested that smoking increases short-term stress (Parrott, 1995, 1998; Cohen and Lichtenstein, 1990). These studies have been limited methodologically by 1) using small convenience samples, 2) using only variation in cigarette quitting to explore the relationship that smoking has on short-term stress, 3) not leveraging an exogenous component to smoking to explore the relationship that smoking has on short-term stress. In this study, we attempt to address these methodological shortcomings to see if we newly find empirical evidence that smoking reduces short-term stress, which is suggested by theory and qualitative responses from smokers.

We found evidence of substantial omitted variable bias when the relationship between stress and smoking is estimated without using instrumentation. We did not find evidence of simultaneity biasing our estimates. Potential sources of omitted variable bias may include measurement error, genetic factors, peer effects, and other social factors that may affect both anxiety and smoking. Continuing to examine the sources of these omitted variables and how they impact the relationship between stress and smoking should be a priority for future research.

After correcting for omitted variable bias we can only conclude that smoking has no effect on short-term stress, rather than beneficial effects suggested by theory and qualitative responses, or deleterious effects suggested by the psychological literature. In the absence of accounting for omitted variable bias; however, we find results identical to the psychological literature: smoking increases short-term stress.

In the face of this empirical evidence, it is unclear why smokers self-report that smoking cigarettes reduces their stress. If incomplete information is to blame, this may present an opportunity for an educational campaign to provide individuals with accurate information that cigarettes do not reduce stress and to discuss alternative proven stress-reduction methods. This may encourage substitution of cigarettes for other forms of stress relief when confronted

with exogenous sources of stress, such as terrorist events or natural disasters.

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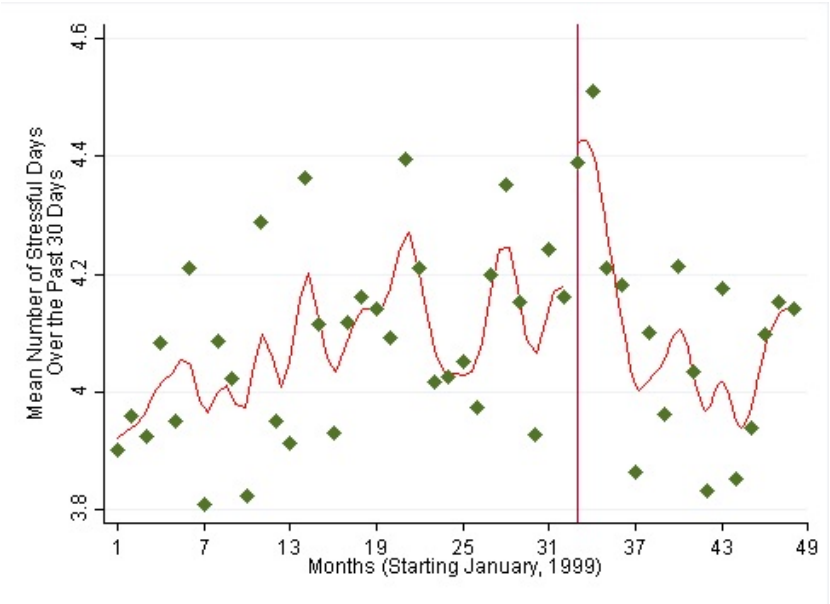

Figure 1: Stressful Days Over Past 30 Days Before and After 9/11 Among Current and Former Smokers

Notes: This graph shows local polynomial smoothed plots (bandwidth of 0.8) of the differences in the monthly mean of stressful days over the past 30 days starting in January, 1999 and ending in December, 2002, for 23 states that collect this information in 2002. All 51 states collect this information from 1999-2001, and so we use this larger sample in our main analysis.

Table 1: Sample Descriptive Statistics for Continental United States, 1999–2001

	Mean	Standard Deviation
Male (%)	0.542	-
Female (%)	0.458	-
White non-Hispanic (%)	0.781	-
Black non-Hispanic (%)	0.080	-
Asian non-Hispanic (%)	0.015	-
Native American non-Hispanic (%)	0.011	-
Hispanic (%)	0.092	-
Missing Race/Ethnicity (%)	0.022	-
Age	47.011	16.957
Junior High (%)	0.046	-
Some High School (%)	0.101	-
High School (%)	0.347	-
Some College (%)	0.277	-
College (%)	0.226	-
Missing Education (%)	0.003	-
Employed (%)	0.629	-
Unemployed (%)	0.045	-
Student (%)	0.026	-
Not Student, Not in Labor Force (%)	0.298	-
Missing Employed Status (%)	0.003	-
Married (%)	0.579	-
Divorced (%)	0.154	-
Widowed (%)	0.070	-
Unmarried and Other Marital Status (%)	0.195	-
Missing Marital Status (%)	0.002	-
Real Household Income (without imputation)	43,829	26,191
Real Household Income (with imputation)	43,226	25,222
State-Level Unemployment Rate (%)	4.310	0.949
Monthly Change in Real Price of Pack of Cigarettes (%)	0.013	0.030
Distance to Closest State (from Center of County)	65.815	67.845
Current Smoker (% of Lifetime Smokers) (Past 30 Days)	0.482	-
Stress (Days Mental Health Not Good over Past 30 Days)	3.829	8.104
Stress (binned)	0.865	1.636

Notes: N = 240,388. All estimates use survey weights. Prices are in 2001 dollars.

Table 2: "Motivation Effect" from Single Equation Instrumental Variable Probit Models

	(1)	(2)
Stress (binned) (95% CI)	0.509*** (0.289, 0.728)	0.308** (0.019, 0.597)
Instrument coefficients (from first stage model)		
Post-9/11	0.016 (-0.094, 0.125)	0.074 (-0.036, 0.184)
Days after 9/11	0.001 (-0.001, 0.002)	0.001 (-0.001, 0.002)
Strength of instruments		
p-value/F-statistic of excluded instruments for stress (H_0: instruments = 0)[a]	0.016, 4.15	0.000, 10.56
Endogeneity of regressor		
Correlation parameter, ρ (95% CI) (H_0: stress is exogenous)	-0.749 (-0.950, -0.115)	-0.141 (-0.764, 1.125)
Exogeneity of instruments		
p-value of joint test of exogeneity of instruments[b] (H_0: instruments exogenous)	0.066	0.308
p-value of Amemiya–Lee–Newey over-identification test[c] (H_0: instruments exogenous)	0.539	0.784
Unemployment rate	Yes	Yes
Socio-demographic controls	Yes	Yes
Month indicators	Yes	Yes
Year indicators	Yes	Yes
State indicators	No	Yes
Observations	240,388	240,388
Non-instrumented stress		
Stress probit coefficient (95% CI)	0.047*** (0.041, 0.052)	0.048*** (0.043, 0.054)

Notes: [a] Test statistic generated using survey data and regressing stress on all controls and instruments. [b] Estimated by regressing equation (1) with the distance variables included as covariates, and reporting the joint significance of the distance parameters. A failure to reject the null hypothesis signifies that temporal distance is not associated with smoking independent of stress. [c] Test statistic generated from instrumental variable probit regression using unweighted data. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 3: "Self-Medication Effect" from Single Equation Instrumental Variable 2SLS Models

	(1)	(2)
Smoking prevalence (95% CI)	0.029 (-0.855, 0.914)	-0.102 (-2.496, 2.292)
Instrument coefficients (from first stage model)		
Monthly price change in pack of cigarettes	-0.247** (-0.449, -0.045)	-0.189* (-0.392, 0.014)
Minimum distance to nearest state	-0.000*** (-0.000, -0.000)	-0.000** (-0.000, -0.000)
Validity of instruments		
p-value of Sargan–Basmann test[a] (H_0: instruments are valid and structural equation is correctly specified)	0.156	0.180
Exogeneity of instruments		
p-value of joint test of exogeneity of instruments[b] (H_0: instruments exogenous)	0.931	0.921
Strength of instruments		
p-value/F-statistic of excluded instruments for smoking (H_0: instruments = 0)	0.000, 30.14	0.019, 3.99
Unemployment rate	Yes	Yes
Socio-demographic controls	Yes	Yes
Month indicators	Yes	Yes
Year indicators	Yes	Yes
State indicators	No	Yes
Observations	240,388	240,388
Non-instrumented smoking		
Smoking prevalence coefficient (95% CI)	0.199*** (0.177, 0.222)	0.204*** (0.181, 0.227)

Notes: [a] Test statistic generated from instrumental variable 2SLS regression using unweighted data. [b] Estimated by regressing equation (2) with the cigarette accessibility variables included as covariates, and reporting the joint significance of the cigarette accessibility parameters. A failure to reject the null hypothesis signifies that cigarette accessibility is not associated with stress independent of smoking. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 4: Results from generalized structural equation models

	(1)	(2)	(3)	(4)
Stress (binned) coefficient	0.508***	0.308**	0.788***	0.389*
95% CI	(0.291, 0.725)	(0.019, 0.596)	(0.425, 1.151)	(-0.028, 0.807)
Smoking prevalence coefficient	0.013	-0.033	0.017	0.168
95% CI	(-0.419, 0.444)	(-0.907, 0.841)	(-0.327, 0.361)	(-0.634, 0.969)
Estimation for stress equation	Linear	Linear	Ordered Probit	Ordered Probit
Estimation for smoking equation	Probit	Probit	Probit	Probit
Strength of instruments				
p-value/F-statistic of excluded instruments (terrorism) for stress (H_0: instruments = 0)	0.024, 3.73	0.000, 7.90	0.071, 2.65	0.004, 5.51
p-value/F-statistic of excluded instruments (cigarette accessibility) for smoking (H_0: instruments = 0)	0.024, 3.73	0.025, 3.69	0.034, 3.38	0.033, 3.42
Endogeneity of regressors				
Correlation parameter, ρ (95% CI) (H_0: regressors are exogenous)	-0.752 (-0.953, -0.088)	-0.396 (-0.833, 0.345)	-0.746 (-0.957, -0.018)	0.461 (-0.904, 0.459)
Unemployment rate	Yes	Yes	Yes	Yes
Socio-demographic controls	Yes	Yes	Yes	Yes
Month indicators	Yes	Yes	Yes	Yes
Year indicators	Yes	Yes	Yes	Yes
State indicators	No	Yes	No	Yes
Observations	240,388	240,388	240,388	240,388
Non-instrumented, non-simultaneous results				
Stress probit coefficient	0.047***	0.048***	0.047***	0.048***
(95% CI)	(0.041, 0.052)	(0.043, 0.054)	(0.041, 0.052)	(0.043, 0.054)
Smoking prevalence coefficient	0.199***	0.204***	0.126***	0.134***
(95% CI)	(0.177, 0.222)	(0.181, 0.227)	(0.108 0.144)	(0.116 0.152)

Notes: *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table Online Appendix 1: Full Results from Single Equation Instrumental Variable Models

	(1)	(2)	(3)	(4)
Stress (binned)	0.5086***	0.3080**		
	(0.1103)	(0.1474)		
Current Smoker (% of Lifetime Smokers) (Past 30 Days)			0.0294	-0.1021
			(0.4514)	(1.2216)
State-Level Unemployment Rate	-0.0055	0.0196*	-0.0071	-0.0599***
	(0.0051)	(0.0103)	(0.0065)	(0.0125)
Female	-0.1148***	-0.0457	0.2789***	0.2794***
	(0.0406)	(0.0468)	(0.0123)	(0.0189)
Black non-Hispanic	0.0775***	0.0693***	-0.0737***	-0.0737**
	(0.0170)	(0.0190)	(0.0261)	(0.0348)
Asian non-Hispanic	0.1136***	0.1513***	-0.1448***	-0.1593**
	(0.0436)	(0.0534)	(0.0493)	(0.0628)
Native American non-Hispanic	-0.0093	0.068	0.2121***	0.2338**
	(0.0693)	(0.0665)	(0.0672)	(0.0913)
Hispanic	-0.0226	-0.0543	-0.1315***	-0.1569***
	(0.0471)	(0.0407)	(0.0348)	(0.0539)
Missing Race/Ethnicity	-0.0594	0.0339	0.2584***	0.2480***
	(0.0594)	(0.0584)	(0.0478)	(0.0671)
Age	-0.0063**	-0.0017	0.0168***	0.0162***
	(0.0030)	(0.0034)	(0.0023)	(0.0031)
Age Squared	0.0000	-0.0002**	-0.0004***	-0.0004***
	(0.0001)	(0.0001)	(0.0000)	(0.0001)
Some High School	0.0832**	0.1058***	-0.014	-0.0037
	(0.0366)	(0.0328)	(0.0411)	(0.0613)
High School	0.0519	0.0076	-0.1528***	-0.1453***
	(0.0354)	(0.0380)	(0.0329)	(0.0344)
Some College	-0.0551	-0.1184***	-0.1140***	-0.1142
	(0.0556)	(0.0446)	(0.0420)	(0.0711)
College	-0.1908	-0.3373***	-0.1904**	-0.2022
	(0.1181)	(0.0807)	(0.0742)	(0.1749)
Missing Education	-0.019	-0.0849	-0.1666	-0.1708
	(0.1014)	(0.1045)	(0.1330)	(0.1454)
Unemployed	-0.1775**	-0.0506	0.4749***	0.4742***
	(0.0770)	(0.0843)	(0.0369)	(0.0536)
Student	-0.1699***	-0.2009***	0.0847*	0.0758
	(0.0443)	(0.0350)	(0.0513)	(0.0920)
Not Student, Not in Labor Force	-0.2601***	-0.2028***	0.4033***	0.3980***
	(0.0286)	(0.0508)	(0.0209)	(0.0401)
Missing Employed Status	-0.1583*	-0.1323	0.1575	0.1522
	(0.0909)	(0.0960)	(0.1510)	(0.1558)
Divorced	0.0439	0.2043**	0.3360***	0.3458**
	(0.1079)	(0.0867)	(0.0552)	(0.1470)
Widowed	0.1444*	0.2559***	0.1289***	0.1365
	(0.0839)	(0.0565)	(0.0490)	(0.1220)
Unmarried and Other Marital Status	0.1156	0.2480***	0.1735***	0.1797
	(0.0869)	(0.0625)	(0.0510)	(0.1378)
Missing Marital Status	-0.0225	0.0138	0.0821	0.0847
	(0.0799)	(0.0853)	(0.1298)	(0.1318)
Real Household Income	-0.0012	-0.0033**	-0.0048***	-0.0053**
	(0.0017)	(0.0014)	(0.0009)	(0.0023)
Monthly Change in Real Price of Pack of Cigarettes	-0.5093*	-0.5516*		
	(0.3090)	(0.3100)		
Distance to Closest State (from Center of County)	-0.0004***	-0.0003**		
	(0.0002)	(0.0001)		
9/11 Terrorism Variable			0.0154	0.074
			(0.0565)	(0.0566)
Days After 9/11			0.0009	0.0008
			(0.0008)	(0.0008)
Constant	0.1946	0.3977	0.8909**	1.3595
	(0.3127)	(0.2709)	(0.4114)	(1.0401)
Month indicators	Yes	Yes	Yes	Yes
Year indicators	Yes	Yes	Yes	Yes
State indicators	No	Yes	No	Yes
Observations	240,388	240,388	240,388	240,388

Notes: Columns 1 and 2 present full results for Table 1. Columns 3 and 4 present full results for Table 2. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table Online Appendix 2: "Self-Medication Effect" from Single Equation Instrumental Variable 2SLS Models with Missing Counties Excluded

	(1)	(2)
Smoking prevalence (95% CI)	0.741 (-0.384, 1.866)	0.061 (-2.678, 2.800)
Instrument coefficients (from first stage model)		
Monthly price change in pack of cigarettes	-0.207* (-0.444, 0.030)	-0.157 (-0.396 0.081)
Minimum distance to nearest state	-0.000*** (-0.000, -0.000)	-0.000** (-0.000, -0.000)
Validity of instruments		
p-value of Sargan–Basmann test[a] (H_0: instruments are valid and structural equation is correctly specified)	0.265	0.339
Exogeneity of instruments		
p-value of joint test of exogeneity of instruments[b] (H_0: instruments exogenous)	0.549	0.977
Strength of instruments		
p-value/F-statistic of excluded instruments for smoking (H_0: instruments = 0)	0.000, 19.73	0.046, 3.07
Unemployment rate	Yes	Yes
Socio-demographic controls	Yes	Yes
Month indicators	Yes	Yes
Year indicators	Yes	Yes
State indicators	No	Yes
Observations	182,054	182,054
Non-instrumented smoking		
Smoking prevalence coefficient (95% CI)	0.197*** (0.169, 0.224)	0.201*** (0.173, 0.228)

Notes: [a] Test statistic generated from instrumental variable 2SLS regression using unweighted data. [b] Estimated by regressing equation (2) with the cigarette accessibility variables included as covariates, and reporting the joint significance of the cigarette accessibility parameters. A failure to reject the null hypothesis signifies that cigarette accessibility is not associated with stress independent of smoking. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.