Economics and Human Biology 22 (2016) 94–102

Contents lists available at ScienceDirect

Economics and Human Biology

journal homepage: http://www.elsevier.com/locate/ehb

The self-medication hypothesis: Evidence from terrorism and cigarette accessibility

Michael F. Pesko [a,*], Christopher F. Baum [b,c]

[a] Department of Healthcare Policy and Research, Weill Cornell Medical College, Cornell University, United States
[b] Department of Economics and School of Social Work, Boston College, United States
[c] Department of Macroeconomics, DIW Berlin, Germany

ARTICLE INFO

Article history:
Received 4 November 2015
Received in revised form 9 February 2016
Accepted 6 March 2016
Available online 24 March 2016

JEL classification:
C26
C36
I19

Keywords:
Stress
Smoking
Self-medication

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.

1. 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).

[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.

* Corresponding author.
E-mail addresses: mip2037@med.cornell.edu (M.F. Pesko), kit.baum@bc.edu (C.F. Baum).

http://dx.doi.org/10.1016/j.ehb.2016.03.007
1570-677X/

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-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% increase in the probability of becoming unemployed causes an individual to be 2.4% more likely to continue smoking. Cotti et al. (2014) find that large negative stock market shocks are widely associated with increased cigarette consumption and purchases, independent of other macroeconomic labor conditions. Finally, Pesko (2014) find 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 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 have not 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 2 discusses the data, Section 3 articulates our empirical strategy, Section 4 presents the results and shows evidence that they are causal in nature, and Section 5 concludes.

2. Data

We use survey data for the continental United States from the Behavioral Risk Factor Surveillance System

[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.