The effect of smoking on obesity: Evidence from a randomized trial

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This paper aims to identify the causal effect of smoking on body mass index (BMI) using data from the Lung Health Study, a randomized trial of smoking cessation treatments. Since nicotine is a metabolic stimulant and appetite suppressant, quitting or reducing smoking could lead to weight gain. Using randomized treatment assignment to instrument for smoking, we estimate that quitting smoking leads to an average long-run weight gain of 1.8–1.9 BMI units, or 11–12 pounds at the average height. Semi-parametric models provide evidence of a diminishing marginal effect of smoking on BMI, while subsample regressions show that the impact is largest for younger individuals, those with no college degree, and those in the lowest quartile of baseline BMI.

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1. Introduction

The study of risky health behaviors has become a major part of the field of health economics, with the number of new papers on the topic growing exponentially since the 1980s (Cawley and Ruhm, 2011). While much of the literature on the causes of these behaviors focuses on traditional economic variables such as income, education, prices, and time preferences (Cawley and Ruhm, 2011), an increasing number of studies examine interactions between behaviors. Understanding the causal effects of different health behaviors on each other is necessary to fully understand the costs and benefits of associated public policies. However, endogeneity concerns complicate such investigations, which typically are either only associational or rely on potentially endogenous policy changes for identification. In contrast, we estimate the impact of smoking on weight by leveraging randomized variation from a clinical trial originally designed to track how lung health responds to smoking-related interventions.

A causal effect of smoking on weight is possible because nicotine is both an appetite suppressant and metabolic stimulant (Pinkowish, 1999). Such a relationship can be placed into the standard human capital model of health (e.g. Grossman, 1972), in which health stock depreciates over time but can be replenished by spending money or time on health investments or further depleted through disinvestments. Assuming an initial weight at or above the medical optimum, smoking and weight gain both represent disinvestments. Weight gain increases with calories consumed and decreases with calories burned, where the latter depends on both physical activity and basal metabolism. Nicotine’s appetite-suppressing properties could be seen as decreasing the marginal utility of calorie intake, while its stimulant properties increase basal

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metabolism. Both of these effects imply that exogenously-induced smoking cessation should lead to weight gain. On the other hand, quitting smoking may increase physical activity by improving lung capacity (Hedenstrom et al., 1986), thereby decreasing the marginal disutility from exercise. 2

Understanding the extent of the causal link between smoking and weight is important because of their roles as major causes of preventable illness and their inverse trends. The obesity rate 3 has steadily increased in the United States (US) over the past half-century, rising from 13% in the early 1960s to 35% in 2011–2012 (Flegal et al., 1998; Ogden et al., 2014). The rise in obesity has contributed significantly to increasing rates of diabetes, heart disease, and stroke (Mokdad et al., 2001; Manson et al., 1990; Rexrode et al., 1997). Estimates suggest that obesity-related diseases lead to 112,000 deaths per year (Flegal et al., 2005) and $190 billion in medical expenses (Cawley and Meyerhofer, 2012), about half of which are paid by public insurance (Finkelstein et al., 2009). In contrast, the percentage of adults who use tobacco in the US declined from 42% to 19% between 1965 and 2010 (U.S. Department of Health and Human Services, 2012). This decrease followed the 1964 Surgeon General’s Report, which concluded that smoking leads to adverse health conditions such as lung cancer and heart disease and raises mortality risk (U.S. Department of Health and Human Services, 1964). Nonetheless, tobacco is still responsible for one out of every five deaths in the United States and at least $30 billion per year in medical expenses (U.S. Department of Health and Human Services, 2014). If the effect of smoking on weight is sufficiently strong, the falling smoking rate could have meaningfully contributed to the rise in obesity.

A large public health literature documents that individuals tend to gain weight following smoking cessation. Two meta-analyses have found average weight gains of four and ten pounds, respectively (U.S. Department of Health and Human Services, 1990; Aubin et al., 2012). Evidence regarding longer-run effects is mixed. Some studies have found that much of the weight gain after quitting smoking is temporary (Chen et al., 1993; Mizoue et al., 1998), but others conclude that the effect remains sizeable five to ten years after cessation (Flegal et al., 1995; Travier et al., 2012; Williamson et al., 1991). These associational estimates could be susceptible to bias from unobservable characteristics, such as time preference and level of interest in one’s health, that influence both smoking cessation and weight trajectory.

The economics literature has attempted to move closer toward causality by leveraging variation from cigarette price or tax rate, obtaining mixed results. 2 Chou et al. (2004), Rashad et al. (2006), Baum (2009), and Sen et al. (2010) estimate positive relationships between cigarette costs and BMI, while Rashad (2006), Fang et al. (2009), Kasteridis and Yen (2012, 2014), and Amialchuk et al. (2016) find that smoking reduces BMI using cigarette price or tax as an instrument. However, Gruber and Frakes (2006), Courtemanche (2009), and Wehby and Courtemanche (2012) estimate the effect of cigarette costs on BMI to actually be negative, while Nonnemaker et al. (2005) and Courtemanche et al. (2016) find little evidence of an effect in either direction. A concern with this literature is whether cigarette costs are endogenous. Cigarette prices may depend on the demand for cigarettes, while high cigarette taxes may be more politically palatable in states where a relatively small percentage of the population smokes. 6

To our knowledge, the only paper that uses a randomized smoking cessation intervention to estimate the causal effect of smoking on weight is Eisenberg and Quinn (2006). EQ use summary statistics reported by O’Hara et al. (1998) on treatment and control groups’ average changes in weight and smoking status during the Lung Health Study to compute a Wald instrumental variables (IV) estimate. They find that sustained smoking cessation over a five-year period leads to a very large average weight gain of 9.7 kg (21.4 pounds), about two to five times the magnitude typically found in the associational literature. However, there is reason to suspect that EQ’s estimate is overstated. Their approach attributes the entire difference in weight changes between the treatment and control groups to sustained quitters, whereas the intervention could plausibly have also led to weight gains among those who quit smoking in some but not all follow-up periods or who reduced smoking intensity but never quit entirely. To the extent that part of the effect of the intervention on weight occurs among individuals besides those coded as sustained quitters, the Wald IV estimator will scale the difference in weight between the treatment and control groups by too small a denominator, leading to upward bias.

We add to the literature on the effect of smoking on weight by using the LHS microdata, which enables several contributions. First, the microdata allow us to construct smoking measures that account for delayed or temporary quitting as well as smoking intensity, thereby circumventing the above concern and enabling more credible estimation of causal effects. Our preferred results imply that quitting smoking leads to an average weight gain of 1.5–1.7 BMI units (10–11 pounds at the average height) after one year and 1.8–1.9 BMI units (11–12 pounds) after five years. Second, the microdata allow us to compute standard errors for IV estimates, which EQ were not able to do with aggregated summary statistics. Our third contribution is to leverage the availability of multiple smoking-related variables in the microdata to show that the results are similar regardless of whether a self-reported or clinical (carbon monoxide [CO] level) smoking measure is used. Finally, microdata enable the evaluation of heterogeneous effects across the smoking and weight distributions as well as by demographic characteristics. We estimate a semi-parametric instrumental variables model that allows the data to determine the functional form of the relationship between smoking and BMI. The results suggest a diminishing marginal effect, with additional smoking having little long-run impact on BMI beyond about a pack of cigarettes per day or a CO level of about 20 parts per million (ppm). We also find that on average those with no college degree, younger individu-

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2 The relationship between smoking and weight could also be examined using a static model of body weight (e.g. Philipson and Posner, 1999), a rational addiction model (e.g. Becker and Murphy, 1988), or models featuring irrationalities (e.g. Ruhm, 2012; Courtemanche et al., 2015a,b). However, the general discussion in this paragraph would remain the same.

3 Obesity is defined as having a body mass index (BMI) greater than 30, where BMI is equal to weight in kilograms divided by height in meters squared.

4 Economic causes of obesity may include falling monetary and time costs of food consumption attributable to technological innovations in food production, preservation, and distribution (Cutler et al., 2003; Lakdawalla et al., 2005; Courtemanche and Carden, 2011) as well as a rising opportunity cost of physical activity due to the increasingly sedentary nature of employment and the improvements in transportation infrastructure that enabled suburbanization (Lakdawalla et al., 2005; Zhao and Kaestner, 2010).

5 A related literature documents evidence of adolescents smoking in an effort to control their weight (Cawley et al., 2004; Rees and Saba, 2010; Cawley et al., 2016). Chou et al. (2004), Rashad et al. (2006), Baum (2009), and Sen et al. (2010) estimate positive relationships between cigarette costs and BMI, while Rashad (2006), Fang et al. (2009), Kasteridis and Yen (2012, 2014), and Amialchuk et al. (2016) find that smoking reduces BMI using cigarette price or tax as an instrument.

6 The discrepancies in observed results in the literature hinge largely on methodological issues such as whether cigarette prices or tax rates are used as the measure of cigarette costs, how time is modeled, and whether the difference between short-run and long-run effects is considered. See the debate between Chou et al. (2004) and Gruber and Frakes (2006) for further discussion of the first two issues and Courtemanche (2009) for a discussion of the third.

7 Fletcher (2013) considers different policy workplace smoking bans and finds evidence that smoking cessation induced by these bans increases BMI. Wehby et al. (2012) use genetic instruments and find that smoking may increase BMI at the lower end of the BMI distribution but decrease it at the higher end.
als, and those with baseline BMI levels in the lowest quartile of the distribution gain the most weight in response to smoking cessation.

2. Data

This section provides a brief introduction to the LHS, with an emphasis on the information most relevant for our paper. O’Hara et al. (1993, 1998) provide a more detailed discussion of the LHS, and further information is also available online at https://www.clinicaltrials.gov.

The purpose of the LHS was to observe changes in the severity of chronic obstructive pulmonary disease (COPD) among smokers. The study consisted of 5,887 smokers with initial ages between 35 and 59. Ten hospitals – in Baltimore (MD), Birmingham (AL), Cleveland (OH), Detroit (MI), Los Angeles (CA), Pittsburgh (PA), Portland (OR), Rochester (MN), Salt Lake City (UT) and Winnipeg (Canada) – each recruited about 600 participants and assigned 400 to treatment and 200 to control. Recruitment started in 1986 and ended in 1989. The clinical trial ended in 1994. To be eligible for selection, potential participants had to show signs of mild lung function impairment, have no history of certain medications, consume fewer than 25 drinks per week, and have no severe illnesses or chronic medical conditions. Each year all participants were extensively interviewed individually at a medical clinic near the residence of the participant (no more than 75 miles away from the participant’s permanent residence). The data therefore consist of the baseline period (1989) plus five annual follow-up periods (1990 through 1994). Attrition was relatively low, as 5297 individuals remained in the sample in the final wave.

Participants were randomly assigned into two treatment groups and one control group. Both treatment groups received a special intervention (SI) consisting of free nicotine gum, an intensive quit week, and frequent contact with support personnel with invitations to bring a spouse or relative to the meetings. The only difference between the two treatment groups was that, in addition to the SI, one group received an inhaled bronchodilator (SI-A) while the other received an inhaled placebo (SI-P). The intensive treatments were completed within the first 4 months of the study. Thereafter, treatment shifted to periodic maintenance programs for those who had successfully quit smoking, which included two weight management counseling sessions per year (O’Hara et al., 1993). The control group referred to as the usual care (UC) group received no intervention and members continued to use their own private sources for medical care.

Our BMI- and smoking-related variables were all recorded during the annual clinic visits. Weight and height were measured by medical staff, so our BMI measure is not susceptible to the concern about measurement error that is common in the economics of obesity literature (Courtemanche et al., 2015b). Nonetheless, Burkhauser and Cawley (2008) note that BMI is an imperfect measure of fatness compared to body fat percentage, so we cannot rule out the possibility that part of the weight gained following smoking cessation was due to increased muscle mass from beginning a strength training regimen. The data contain both self-reported and clinically measured smoking information. Respondents report whether they currently smoke and, if so, approximately how many cigarettes they smoke per day. The clinical measure is CO level, and the LHS considers an individual to be a non-smoker (i.e. medically verified quitter) if their CO level is under ten ppm.

Table 1 presents descriptive statistics for the three groups at the time of randomization. Average cigarette consumption was roughly 30 cigarettes per day, average CO level was about 26, and the average respondent was just slightly overweight. The summary statistics for all variables are very similar across the three groups, indicating the randomization was successful. For comparison purposes, the final column of Table 1 shows the summary statistics for white smokers ages 35–59 in the 1990 National Health Interview Survey. We see that LHS participants exhibit broadly similar observable characteristics to other smokers from the same time period with similar races and ages. Importantly, average baseline BMI in the LHS is very similar to that from the NHIS sample. Perhaps the most notable difference is that individuals in the LHS smoked an average of seven cigarettes per day more than those in the NHIS, which is not surprising since light smokers would not be expected to enroll in an intensive smoking cessation intervention.

Fig. 1 displays changes throughout the sample period in the average number of cigarettes smoked per day, objectively-verified smoking status, CO level, and BMI for each group. Sharp decreases in cigarette smoking, smoking status and CO level are evident for both treatment groups in the first year after the intervention. Smoking also declines for the control group during the sample period, but the reduction is much more modest and gradual than those of the treatment groups. The fact that we observe some drop in smoking for the control group is not surprising since all participants desired to quit smoking at the start of the study. Average BMI is trending

8 The data do not contain state identifiers so we are unable to control for state-level variables such as the cigarette tax rate. However, since the numbers of recruited participants in each group were equal across locations, the LHS’s randomization should prevent this omission from causing bias. Moreover, changes in cigarette taxes during the sample period were modest in the nine states containing LHS sites. The only tax increase larger than 13 cents per pack was MI’s increase from 25 to 75 cents, and this occurred at the very end of the sample period (May 1994) (Orzechowski and Walker, 2016).

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Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>SI-A Mean</th>
<th>SI-P Mean</th>
<th>UC Mean</th>
<th>NHIS Mean</th>
</tr>
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<td>Age</td>
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<td>48.55</td>
<td>48.24</td>
<td>45.02</td>
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<tr>
<td>(6.84)</td>
<td>(6.83)</td>
<td>(6.76)</td>
<td>(6.94)</td>
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<td>29.49</td>
<td>29.51</td>
<td>22.00</td>
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<tr>
<td>(14.08)</td>
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</tr>
<tr>
<td>Carbon monoxide</td>
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<td>26.70</td>
<td>25.98</td>
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</tr>
<tr>
<td>(13.47)</td>
<td>(12.67)</td>
<td>(12.69)</td>
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<td></td>
</tr>
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<td>BMI</td>
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<td>(3.83)</td>
<td>(4.63)</td>
<td></td>
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<td>0.30</td>
<td>0.29</td>
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<td>(0.45)</td>
<td>(0.46)</td>
<td>(0.45)</td>
<td>(0.50)</td>
<td></td>
</tr>
<tr>
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<td>0.35</td>
<td>0.20</td>
</tr>
<tr>
<td>(0.47)</td>
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<td>(0.47)</td>
<td>(0.40)</td>
<td></td>
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<tr>
<td>College degree</td>
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<td>0.22</td>
<td>0.24</td>
<td>0.15</td>
</tr>
<tr>
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<td>(0.42)</td>
<td>(0.42)</td>
<td>(0.36)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
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<td>0.64</td>
<td>0.64</td>
<td>0.54</td>
</tr>
<tr>
<td>(0.49)</td>
<td>(0.48)</td>
<td>(0.48)</td>
<td>(0.50)</td>
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</tr>
<tr>
<td>Married</td>
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<td>0.65</td>
<td>0.73</td>
</tr>
<tr>
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<td>(0.46)</td>
<td>(0.47)</td>
<td>(0.45)</td>
<td></td>
</tr>
<tr>
<td>Observations</td>
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<td>1962</td>
<td>1964</td>
<td>4074</td>
</tr>
</tbody>
</table>

Notes: * indicates that t-tests show the mean is different from the SI-P and UC means at the 5% level. SI-A and SI-P are the two treatment groups while UC is the control group; data for these columns come from the LHS. The ‘NHIS’ column shows summary statistics among 35–59 year old white smokers in the 1990 NHIS.
upward for all three groups, but the two treatment groups experience much sharper increases in BMI than the control group in the first year. The graph therefore provides preliminary evidence that the intervention was effective in reducing smoking and that smoking reduces BMI.

3. Econometric analyses

Our econometric objectives are to identify (1) the average short- and long-run causal effects of quitting smoking on weight gain, (2) how the effect of smoking on weight changes across the smoking distribution, and (3) how the effect of smoking on weight varies by demographic characteristics and baseline BMI. We begin by using parametric regressions to answer the first question. We then conduct semiparametric estimation allowing for a flexible relationship between smoking and weight to address the second question. Next, we answer the third question with subsample analyses. Finally, we address issues related to the generalizability of the results and show why our results differ from those of EQ.

3.1. Average effects

3.1.1. Short run

We begin by aiming to identify the average causal effect of quitting smoking on weight gain with a series of parametric regressions. Economists typically consider body weight to be a capital stock. Individuals start with an exogenous endowment of weight that changes over time due to depreciation as well as “investments” that take the form of caloric intake or expenditure. In the LHS, weight at the end of the first follow-up year can therefore be modeled as a function of weight at baseline and investments – such as smoking – in year one:

$$bmi_{i1} = \beta_0 + \beta_1 bmi_{i0} + \beta_2 S_i + \beta_3 X_i + \varepsilon_{i1}$$

where $bmi_{i1}$ is individual $i$’s BMI at the end of year 1, $bmi_{i0}$ is BMI at the beginning of the study (year 0), $S_i$ is smoking in year 1, $X_i$ is a vector of demographic controls that are assumed to be constant over time since they are only available for the baseline wave, and $\varepsilon_{i1}$ is period 1’s error term. $\beta_2$, the coefficient of interest, gives the short-run association between smoking and weight. We estimate the OLS model given by (1) as well as an IV model that uses the randomized treatment assignment to instrument for $S_i$. The first stage of the IV model is given by

$$S_{i1} = \gamma_0 + \gamma_1 bmi_{i0} + \gamma_2 si_{i1} + \gamma_3 si_{p1} + \gamma_4 X_i + \mu_{i1}$$

where $si_{i1}$ and $si_{p1}$ reflect whether the individual was assigned into the SI-A or SI-I treatment group, respectively. The second stage of the IV model is identical to (1) except it replaces $S_{i1}$ with the predicted value generated by (2). In the IV model, $\beta_2$ can be interpreted as the short-run local average treatment effect (LATE) of intervention-induced changes in smoking on BMI. We estimate linear models in both stages due to their relative ease of interpretation, their ability to produce reliable average effects (e.g. Angrist and Pischke, 2009), and the inherent difficulties with non-linear IV estimation (e.g. Terza et al., 2008).

We define $S_i$ in three different ways. The first smoking measure is a dummy equal to one if and only if individual $i$ was a medically validated non-smoker at the end of year one. We consider this to be a naive measure of smoking because it ignores variation in smoking intensity among smokers. This could lead to an overstatement of the average weight gain from quitting smoking estimated by IV models. When a quit dummy is used as the smoking measure, the IV estimator effectively scales the difference in BMI between the treatment and control groups by the difference in smoking cessation rates between the two groups. The validity of this estimator therefore hinges on the assumption that the randomized intervention only affected the BMIs of people who fully quit smoking. To the extent that the intervention also affected the BMIs of those who cut back but did not quit entirely, the difference in BMI will be scaled by too small a number and the IV estimate will be too large.

Our second smoking measure is therefore self-reported number of cigarettes smoked per day, with a value of zero assigned to those who reported quitting. This measure incorporates both reducing smoking and quitting entirely and therefore is not susceptible to...
the above criticism. In order to make the results using cigarettes per day comparable to \( \hat{\beta}_2 \) from the regressions using the smoking cessation dummy, we need to compute an implied average weight gain from quitting smoking. We do this by calculating the average weight that would be gained if all individuals in the sample switched from their baseline number of cigarettes to none. Specifically, we multiply the coefficient estimate on the cigarettes smoked per day variable by each individual’s number of cigarettes smoked at baseline, and then take the average across all individuals. Formally, this is \( \sum_{i=1}^{N} \beta_{i} \text{cigday}_{i} / N \), where \( \text{cigday} \) is cigarettes smoked per day and \( i \) indexes the \( N \) observations.\(^{10}\)

A key limitation with cigarettes per day is its self-reported nature. We therefore also utilize a third smoking variable that is both clinically measured and incorporates both the intensive and extensive margins of smoking: CO level. Using the CO regression estimates to compute the average weight gain from quitting smoking is somewhat more complicated than using cigarettes smoked per day since even non-smokers generally have a positive CO level. We therefore compute each individual’s predicted effect of quitting smoking as the effect of switching from her baseline CO level to the mean CO level for non-smokers, rather than to a CO level of zero. For the mean CO level of non-smokers, we use Deveci et al.’s (2004) estimate of 3.61 ppm; this is similar to the mean CO level of verified non-smokers in the follow-up waves of the LHS. The average effect of quitting smoking on weight across the entire sample is therefore given by \( \sum_{i=1}^{N} \beta_{i} (\text{CO}_{i} - 3.61) / N \) where \( \text{CO} \) is CO level in ppm. Note that CO levels are only available at baseline for 922 individuals, so our average effect is computed using only this portion of the sample (though our regressions still utilize the full sample). We doubt that this limitation is of consequence since reported numbers of cigarettes smoked per day at baseline are virtually identical for those with missing baseline CO levels and those with non-missing levels.

\[ \text{BMI}_{25} = \beta_0 + \beta_1 \text{BMI}_{0} + \sum_{t=1}^{5} \beta_{2t} \text{S}_{it} + \beta_{3} \text{X}_t + \epsilon_{2t} \]  

(3)

where \( \text{BMI}_{25} \) is individual \( i \)'s BMI at the end of year 5 and \( S_t \) is smoking in year \( t \). However, the need to utilize IV estimation prevents us from allowing separate coefficients for each of the five smoking variables, as this would require five instruments. To operationalize an IV model we need to compress the five years of smoking information into a single variable \( S_i \). The easiest way to do this is to take a simple average across the five years:

\[ S_i = S_{i1} + S_{i2} + S_{i3} + S_{i4} + S_{i5} \]

(4)

However, this approach assumes that smoking in each of the five periods has the same effect on weight. To the extent that weight is a depreciating capital stock, we might expect smoking in more recent years to have a larger effect on BMI than smoking in more distant years. We therefore also estimate models defining \( S \) as a weighted rather than simple average of quit status in the five follow-up years:

\[ S_i = \frac{S_{i1} + (1 - \delta)S_{i2} + (1 - \delta)^2 S_{i3} + (1 - \delta)^3 S_{i4} + (1 - \delta)^4 S_{i5}}{1 + (1 - \delta) + (1 - \delta)^2 + (1 - \delta)^3 + (1 - \delta)^4} \]

(5)

Since we do not have a sufficient number of instruments to credibly estimate the depreciation rate \( \delta \), we simply try several plausible values: 0.05, 0.1, 0.15, 0.2, 0.25, and 0.3. In all our regressions, the coefficient estimate on the baseline BMI variable will barely be below one, so we consider it probable that the “true” value of \( \delta \) is toward the low end of this range; i.e. there is little reason to consider values of \( \delta \) above 0.3.

### 3.1.3. Results

Table 2 reports the results of interest from the parametric regressions. Panel A shows the estimated effects of the treatment dummies on the smoking variables from the first stage of the IV
models, along with the F-statistic from a test of their joint significance. Panel B presents the OLS and IV estimates of the effects of the different smoking measures on BMI. The first three columns show the effect of year 1 smoking on year 1 BMI (short-run effect), with the first column using the binary quitting variable as the smoking measure, the second using cigarettes smoked per day, and the third using CO. The last three columns present the effects of the simple averages of these three smoking measures across years 1–5 on BMI in year 5 (long-run effect). The results using the weighted averages, available in Appendix Table A1, are similar to those obtained using simple averages.

The first-stage estimates in Panel A show that the treatment was effective in reducing smoking. In the short run, being assigned into the SI-A or SI-P groups increased the probability of quitting by 27–28 percentage points while decreasing cigarettes smoked per day by 11–12 and CO level by 8 ppm. In the long run, SI-A or SI-P assignment increased the fraction of the five follow-up years quit by 0.21 while decreasing average cigarettes per day by 9 and average CO by 6–7 ppm. The treatment variables are all highly significant in the first stage and the F-statistics from the test of their joint significance are easily large enough to conclude that they are sufficiently strong instruments. Also noteworthy is the fact that there is essentially no difference in the coefficient estimates for the two treatment variables; in other words, the inhaled bronchodilator given to the SI-A group did not influence smoking. This also means that, though our IV model is technically overidentified, the instruments are not sufficiently distinct to make an overidentification test informative or to consider instrumenting for two endogenous variables.

Turning to the estimated effects of smoking on weight in Panel B, the first column presents the short-run estimates using the quit dummy. The OLS regression estimates that quitting smoking increases BMI by 1.295 units, or 8.1 pounds at the US average height of 66.4 in.11 This is well within the range of estimates from the associational literature discussed in Section 1. The IV estimate is a larger 2.202 BMI units, and the Hausman test strongly rejects the consistency of OLS. This IV estimate equates to 13.8 pounds, which is larger than most estimates of the average short-run weight gain from quitting smoking from the associational literature.

The next two columns use the smoking measures that incorporate intensity: cigarettes per day and CO. In the IV specifications, we estimate that in the short run an additional cigarette smoked per day reduces BMI by 0.052 units while an additional ppm of CO reduces BMI by 0.077 units. The average effects of quitting smoking implied by these two regressions (shown in brackets) are 1.52 and 1.71 BMI units, which translate to 9.5 and 10.7 pounds at the mean height. These estimates are 31% and 22% smaller than the 13.8 pounds we obtained using the quit dummy. This is consistent with our prediction that neglecting to account for smoking intensity leads to an exaggerated IV estimate of the average weight gain from quitting smoking. Moreover, these implied average effects of quitting smoking both lie outside the 95% confidence interval from the short-run IV regression with the binary cessation measure, which has a lower bound of 1.91 BMI units. Nonetheless, this effect could arguably still be considered quite large, at it is toward the high end of the results from the associational literature.

The last three columns turn to the long-run estimates. The key result is that the long-run effects are slightly stronger than the short-run effects. This is an important result, as the issue of whether the effect diminishes over time has been a point of contention in the associational literature, as discussed in Section 1. In the IV specification using average quit status, quitting for all five follow-up years is estimated to increase BMI by 2.646 units, or 16.6 pounds. An additional cigarette smoked per day over the five years reduces BMI by 0.065 units, while an additional ppm of average CO reduces BMI by 0.082 units. These latter two estimates imply average weight gains from quitting smoking of 1.91 and 1.81 units of BMI, or 12.0 and 11.4 pounds. Again, these implied average effects are smaller than the lower bound of the 95% confidence interval from the corresponding regression using the binary smoking measure, which is 2.1 BMI units.

3.2. Semi-parametric estimation

An issue with the parametric regressions for cigrday and CO is that they assume that smoking intensity affects BMI linearly. This is a strong assumption, as it seems likely that there is either a non-linear dose-response effect of nicotine on metabolism/appetite or a non-linear effect of metabolism/appetite on weight-related behaviors. While it is not clear that this will bias estimates of the average weight gain from quitting smoking, such a restrictive functional form is likely to lead to systematically inappropriate predictions for at least some individuals. Moreover, given the complicated chain of biological and behavioral pathways through which smoking influences BMI, it is not obvious that the non-linearity could be captured through simple approaches such as logarithmic or quadratic specifications. We therefore next estimate a semi-parametric model that allows the data to determine the functional form of the relationship between smoking and BMI. Specifically, we implement Robinson’s (1988) semi-parametric double residual estimator with local smoothing. This approach allows us to model the expectation of the dependent variable at every point on the distribution of the independent variable, thereby enabling the prediction of the weight gained (or lost) from switching from any level of smoking to any other level.12

Semi-parametric IV models can be estimated using a control function approach (Blundell and Powell, 2004; Lee, 2007). The first stage takes the same form as Eq. (2). The second stage differs from Eq. (1) in two ways. First, it does not specify the functional form for the smoking measure. Second, rather than using the predicted value of the smoking variable from the first-stage regression, the second stage includes the residual from the first stage as a regressor.13 The second stage short-run regression can therefore be expressed as

$$bmi_{it} = \beta_0 + \beta_1 bmi_{i0} + f(S_i) + \beta_2 X_i + \beta_3 \tilde{\mu}_i + \epsilon_i$$

where $S$ is either cigrday or CO and $\tilde{\mu}_i$ is the first-stage residual. The second stage long-run regression is similar but replaces $bmi_{i1}$ with $bmi_{i0}$ and $S_i$ with the average smoking measures.

The estimation was conducted using the Stata program “semi” by Verardi and Debarsy (2012). The first step is to estimate $E(bmi|S_i), E(\mu_i|S_i)$ and $E(X_i|S_i)$, which are approximated by the predicted values $\hat{bmi}, \hat{\mu}_i$, and $\hat{X}_i$ by a kernel weighted local polynomial regression. The second step is to form the residuals $\tilde{\mu}_1 = bmi - \hat{bmi}$, $\tilde{\mu}_2 = X - \hat{X}_i$, $\tilde{\mu}_3 = \mu_i - \hat{\mu}_i$. Then the coefficients $\beta_0, \beta_1, \beta_2$, and $\beta_3$, representing the relationships between the independent variables and BMI are estimated by regressing $\tilde{\mu}_1$ on $\tilde{\mu}_2$ and $\tilde{\mu}_3$. The last step is to identify the relationship between cigarette consumption and BMI with a non-parametric regression of cigarette consumption on the predicted BMI residual, $bmi_{i1} - \hat{\beta}_0 - \hat{\beta}_1 bmi_{i0} - \hat{\beta}_2 X_i - \hat{\beta}_3 \tilde{\mu}_i$.

---

11 Average height is computed by taking a simple average of the male and female heights for adults ages 20 and older (69.1 and 63.7 in., respectively) during the time frame corresponding to the LHS (1988–1994). This information is available at https://stacks.cdc.gov/view/cdc/5301. We are not able to use LHS sample mean height because the LHS microdata suppress height and weight, reporting only BMI.

12 For simplicity, we round smoking values to the nearest integer; e.g. if someone averaged 21.2 cigarettes per day over the five follow-up waves we round this to 21.

13 For an overview of the control function approach to dealing with endogeneity, see Heckman (1979) and Heckman and Robb (1986).
This relationship is estimated at every level of cigarette smoking, allowing independent marginal effects. The idea behind this strategy is to estimate the non-parametric cigarette function by the residual variation that is unrelated to the parametric independent variables.

We calculate the average effect of quitting smoking on BMI using the semi-parametric estimates as follows. When using the cigarettes smoked per day variable, we first calculate the change in predicted weight from switching from the number of cigarettes smoked at baseline to zero. We then take the average of these predicted changes across all individuals in the sample. The process for the CO variable is similar; the only difference is that we compute the predicted effect of switching to the average CO level for non-smokers of 3.61 ppm, as opposed to zero.

In semi-parametric estimation, the confidence interval becomes very wide at extreme values where there are few observations. We therefore drop the top 1% of the smoking distribution, which means those who smoke more than 50 cigarettes per day on average across the five follow up years and those with average CO levels of over 50 ppm. We doubt that this restriction is consequential, since if we drop the same individuals in the parametric regressions the results (available upon request) remain similar.

Figs. 2 and 3 present the short-run semi-parametric IV results for cigday and CO, respectively. The graphs display both the point estimates for each integer level of smoking and the 95% confidence intervals. Fig. 2 shows that the short-run relationship between cigarettes smoked per day and BMI is highly nonlinear. Specifically, smoking has a diminishing marginal effect on BMI throughout most of the distribution, with the shape of the curve being approximately quadratic. Quitting smoking from levels of 10, 20, 30, and 40 cigarettes per day is predicted to lead to weight gains of 1.22, 1.58, 1.66, and 1.94 BMI units, respectively. Most of the effect of smoking on weight therefore appears to occur at levels below 20 cigarettes per day. Taken literally, this would suggest that heavy smokers could cut back to a pack a day without fear of substantial weight gain. Fig. 3 shows that the short-run effect of CO on BMI is less obviously non-linear than the effect of cigarettes per day. The curve is somewhat flat at very low levels of CO – specifically two to five ppm – but recall that even non-smokers often have non-zero CO so changes at such low levels probably do not reflect changes in smoking behavior. Starting at five ppm, the graph begins to take a quadratic shape, but unlike the graph for cigarettes per day we do not observe a complete leveling off until the far right tail of the distribution.

Figs. 4 and 5 turn to the long-run results using simple averages of the smoking measures; the graphs using weighted averages are very similar and are available upon request. Fig. 4 shows that the shape of the long-run relationship between cigarettes per day and BMI is roughly similar to the shape of the short-run relationship, as it is approximately quadratic and levels off at around 20 cigarettes per day. Fig. 5 displays a similar pattern of results for CO level. CO has a diminishing marginal effect on BMI, and most of the weight gain from reduced CO comes at levels below about 20 ppm. The long-run relationship between CO and BMI therefore flattens out more quickly than the short-run relationship.\textsuperscript{14}

\textsuperscript{14} Note that there is some evidence that additional CO actually leads to higher BMI at the far right tail of the distribution: CO levels of around 47–50 ppm. However,
The average effects of quitting smoking on BMI implied by these semi-parametric graphs are generally similar to those from the parametric specifications. Using cigarettes per day, the average effect of quitting is 1.67 BMI units in the short run and 1.93 in the long run, compared to 1.52 and 1.91 from the corresponding parametric regressions. For CO, the average effect is 1.80 in the short run and 1.99 in the long run, compared to the parametric regressions’ estimates of 1.33 and 1.81. The results presented in this section suggest that the marginal effect of smoking on weight is likely to be modest for levels of smoking above 20 cigarettes a day, which would be impossible to detect using linear specifications.

### 3.3. Subsample analyses

We next conduct subsample analyses to evaluate whether either knowledge or motivation to quit smoking can mitigate the resulting weight gain. We first examine the role of knowledge by stratifying by education, which could improve one’s ability to prevent weight gain by either directly providing information about nutrition or exercise or enhancing critical thinking skills (Kenkel et al., 2006). For motivation, we focus on two dimensions along which there is variation in the health consequences of weight gain, as individuals for whom the marginal effects of weight on health are the largest may be the most motivated to take steps to limit the amount of weight gained following smoking cessation. The first of these dimensions is age. Since the rates of obesity-related illnesses increase rapidly across our sample age range (Saad, 2011), the oldest individuals in our sample might reasonably be the most concerned about the health risks from gaining weight. The second such dimension is baseline BMI, as the impact of weight gain on health is highly nonlinear across the BMI distribution, with the severe health consequences being concentrated in the right tail (Flegal et al., 2013). Individuals who are already near or above the obesity threshold before quitting smoking might therefore be the most motivated to avoid further weight gain afterwards. On the other hand, such individuals could be genetically predisposed to a relatively large weight gain.

For education, we consider subsamples of those with no college education, some college, and a four-year college degree or greater. For age, we split the sample into three groups — those under 45, 45–54, and 55 and over at baseline — because there are no individuals under 35 or over 64 in the LHS. For baseline BMI, we split the sample into BMI quartiles: those with a baseline BMI of 22.7 BMI units or less, 22.7–25.2, 25.2–27.9, and 27.9 and above.

### Tables 3 and 4

<table>
<thead>
<tr>
<th>Education</th>
<th>Age</th>
<th>No college</th>
<th>Some college</th>
<th>College graduate</th>
<th>&lt;45</th>
<th>45–54</th>
<th>≥55</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average cigarettes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>−0.068***</td>
<td>−0.059***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(0.011)</td>
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<td>[1.98]</td>
<td>[1.73]</td>
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<tr>
<td>N</td>
<td></td>
<td>2082</td>
<td>1722</td>
<td>1162</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average CO</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>−0.088***</td>
<td>−0.071***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(0.016)</td>
<td>(0.019)</td>
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<td></td>
</tr>
<tr>
<td>[1.83]</td>
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</tr>
<tr>
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<td>1895</td>
<td>1555</td>
<td>1067</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Notes: Results are from IV regressions of year 5 BMI on the smoking variable. Heteroskedasticity-robust standard errors are in parentheses. *** and * indicate significance at the 1, 5, and 10 percent levels. The implied average effects of quitting smoking are in brackets. The controls for education, gender, marital status, age, and baseline BMI are included. Data come from the LHS.

### Table 4: Subsample results for baseline BMI (IV with year 5 BMI only).

<table>
<thead>
<tr>
<th>Lowest quartile (&lt;22.7)</th>
<th>Second quartile (22.7–25.2)</th>
<th>Third quartile (25.2–27.9)</th>
<th>Highest quartile (&gt;27.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average cigarettes</td>
<td>−0.063***</td>
<td>−0.059***</td>
<td>−0.054***</td>
</tr>
<tr>
<td>(0.013)</td>
<td>(0.014)</td>
<td>(0.016)</td>
<td></td>
</tr>
<tr>
<td>[1.83]</td>
<td>[1.75]</td>
<td>[1.65]</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>1278</td>
<td>1215</td>
<td>1211</td>
</tr>
<tr>
<td>Average CO</td>
<td>−0.080***</td>
<td>−0.075***</td>
<td>−0.069***</td>
</tr>
<tr>
<td>(0.022)</td>
<td>(0.024)</td>
<td>(0.021)</td>
<td></td>
</tr>
<tr>
<td>[1.71]</td>
<td>[1.60]</td>
<td>[1.56]</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>1173</td>
<td>1100</td>
<td>1097</td>
</tr>
</tbody>
</table>

Notes: Results are from IV regressions of year 5 BMI on the smoking variable. Heteroskedasticity-robust standard errors are in parentheses. *** and * indicate significance at the 1, 5, and 10 percent levels. The implied average effects of quitting smoking are in brackets. The controls for education, gender, marital status, age, and baseline BMI are included. Data come from the LHS.

This should be interpreted with caution as it is based on a very small number of individuals. Accordingly, the confidence intervals in this portion of the distribution are quite large.
27.9. It is noteworthy that the effect is so much stronger for the lowest quartile than the other groups: a 2 BMI unit weight gain following smoking cessation would only bring the heaviest person in the lowest quartile to a BMI of 24.7, which is still under the threshold for overweight. The population-wide health consequences from weight gain after quitting smoking are therefore likely smaller than would be implied by the full-sample estimates.15

In all, though, perhaps the most striking results from Tables 4 and 5 are that, while some heterogeneity appears to exist, the overall amount of heterogeneity is relatively small. Negative and highly significant effects of smoking on weight are evident for all subsamples, and most of the coefficient estimates are within each others’ confidence intervals. The smallest average effect of quitting smoking on BMI from any specification (55 and over, parametric, CO) is a still sizable 1.33. The lack of substantial heterogeneity in the effect within the sample provides some assurances that the results are generalizable outside the sample. The next section evaluates the generalizability issue in more detail.

3.4. External validity

We next perform checks related to external validity. One obvious concern about the generalizability of the results is that the LHS was conducted in the early 1990s, raising the question of the relevance for current policy debates. Another concern is that the LHS participants are not a random sample of smokers. Participants had to be sufficiently motivated to quit smoking to participate in an intensive intervention, have mild (but not major) lung function impairment, and live within reasonable proximity of the locations for follow-up visits. As discussed in the Data section, the end result was a sample that was almost exclusively white (97%) and exclusively middle-aged (starting age 35–59, ending age 40–64). While it is impossible to fully establish the external validity of our estimates, this section aims to at least somewhat alleviate these concerns.

First, we assess the consequences of the observable generalizability issues by conducting additional analyses with the National Health Interview Survey (NHIS), a large nationally representative survey conducted annually by the Centers for Disease Control and Prevention. The NHIS contains self-reported data on smoking, weight, and height, along with the same control variables used in our LHS analyses (except for baseline BMI, since the NHIS is not a panel). We use the NHIS to see if the association between cigarettes smoked per day and BMI varies by time period, race, and age. Obviously a causal analysis is not possible with the NHIS, but verifying that the association between smoking and weight is not particularly unique among the LHS population should provide at least some assurance that the causal effect is not likely to be unique either. We first estimate the association among the NHIS’ best available analog to the LHS sample: white 35–64 year olds in 1990–1994 (the years of the five LHS follow-up waves). We then evaluate whether this association has changed over time by estimating the same model among 35–64 year old whites in the five most recent NHIS waves currently available: 2009–2013. Next, we examine the issue of lack of representativeness by race by returning to the 1990–1994 NHIS waves and restricting the sample to 35–64 year old non-whites. Finally, we estimate the model for whites of an age outside of the 35–64 range (i.e. 18–34 year olds combined with those 65+) in order to evaluate the implications of the lack of representativeness by age.16

Table 5 reports the results. The first column shows that, in the sample most comparable to the LHS, each additional cigarette smoked per day is associated with a reduction in BMI of 0.038 units. This implies an average weight gain from quitting smoking of 0.8 BMI units. The second column shows that the association between cigarettes smoked per day and BMI is stronger in the 2009–2013 sample than the 1990–1994 sample (−0.061 compared to −0.038), but the average effects of quitting smoking are nonetheless fairly similar (0.91 BMI units compared to 0.8) which is similar to the short-run OLS estimate from the LHS. This is because the average number of cigarettes smoked among smokers has dropped over the past two decades. In other words, β2 may have grown over time but cigday has shrunk for the average smoker, leaving \(\frac{\sum_{i=1}^{N}(\beta_2 \cdot \text{cigday}_{i})}{N}\) roughly constant. Next, the third column provides evidence that the association between smoking and BMI for non-whites is stronger than for whites, but the implied average effects of quitting smoking are similar. Again, this is because on average non-white smokers consume fewer cigarettes than white smokers. The final column shows that the association between smoking and BMI among those who are not between the ages of 35 and 64 is virtually identical to the association among those who are in this age range. The average effect of quitting smoking is, however, slightly smaller among the non-35 to 64 sample due to a lower number of cigarettes smoked among smokers. In sum, though there is likely some heterogeneity across age, race, and time, these results provide at least some assurance that the lack of representativeness of the LHS is not driving our conclusions. Smoking is inversely associated with weight in all NHIS subsamples. The associations between cigarettes smoked per day and BMI all fall within a reasonably tight range of −0.038 to −0.061. The implied average effects of quitting smoking are all between 0.68 and 0.91 BMI units, which equate to 4.3–5.7 pounds.

It is harder to investigate the potential threats to external validity from unique unobservable characteristics of the LHS sample. A particular concern is that, since LHS participants had to have signs of diminished lung function and be willing to participate in an extensive intervention, they might be more highly motivated to quit smoking than the general population of smokers, which in turn could influence their subsequent weight gain. One possible proxy for motivation available in the LHS is an indicator variable for whether the individual ever quit smoking for at least one full day prior to the intervention, with the idea being that individuals who are highly motivated to quit smoking have likely tried to do so on their own. In Table 6, we report the IV results from stratifying the LHS sample by this prior quit attempt variable. The estimated short-run effects of smoking on weight are virtually identical for those with a prior quit attempt and those without one. The long-

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15 In unreported regressions (results available upon request), we also find that the effect of smoking on weight is somewhat larger for women than men but does not clearly differ by marital status.

16 The associations of the control variables with BMI are very different for the 18–34 year old age group and the 65+ age group. Therefore, in the regression combining 18–34 year olds with those 65 and older, we include as additional covariates the interactions of each control with an indicator for whether the individual is in the 18–34 portion of the sample or the 65+ portion.
run effects are somewhat larger for the group with no prior attempt, but the results are qualitatively similar. In sum, even if the LHS participants were generally more motivated to quit smoking than the typical smoker, the available evidence suggests that this is not consequential for our analysis of the impact of smoking on weight.

As a final argument for the external validity of our results, note that the effectiveness of the LHS intervention in helping participants quit smoking is broadly in line with that of similar interventions from other clinical trials. Kottke et al. (1988) analyze the results of 73 interventions from 39 earlier trials. While our estimated 27 percentage point effect of the LHS intervention on smoking after one year is toward the high end of the range of impacts, it is within the 95% confidence intervals of 25 of the interventions. Moreover, the LHS’ relative effectiveness appears to be attributable to the intensity of the intervention, as it included the features associated with the most successful trials: face-to-face meetings, multiple reinforcing sessions, and nicotine replacement gum. Indeed, the authors project that a multi-pronged program with these features could yield a 43% quit rate. Ultimately, then, there is no evidence that the LHS enrolled a particularly unusual group of smokers compared to other trials.

3.5. Reconciling our results with prior literature

The last section of our empirical analysis reconciles our results with those of EQ and provides direct evidence of the problems associated with using their smoking measure in an IV context. Recall that EQ used previously published LHS summary statistics from O’Hara et al. (1998) to estimate a very large 21.4 lb average weight gain from smoking cessation. We replicate this approach by computing a Wald IV estimate of the form

$$p_{WALD} = \frac{\bar{b}_{m1} - \bar{b}_{m0}}{\bar{q1} - \bar{q0}}$$

(7)

where subscript one indicates the treatment group (combination of the SI-A and SI-P groups) and zero the control group (UC). $\bar{b}_{m1}$ and $\bar{b}_{m0}$ are average BMIs among the treatment and control groups, respectively, at the end of the study period (year 5). $q_{1}$ represents EQ’s measure of quitting smoking, called “sustained quitting,” which is a dummy variable equal to one if and only if the individual was a medically verified non-smoker in all five follow-up waves. This is a very stringent measure, as anyone who smokes any amount in any of the five follow-up years is classified as a non-quitter. The validity of the Wald estimator hinges on the assumption that the intervention only affected the weight of individuals for whom $q_{1} = 1$. To the extent that the intervention also affected the weight of any individuals with $q_{1} = 0$, the denominator will effectively be too small. The observed difference in average weight between the treatment and control groups will therefore be scaled by too small a number, so the estimated effect of quitting smoking on weight will be overstated.

We suspect that the Wald estimator’s identifying assumption is violated since there are two types of individuals categorized by EQ as having $q_{1} = 0$ whose smoking behavior (and therefore weight) likely responded to the intervention to at least some extent. The first type consists of those who quit smoking for part but not all of the 5-year follow-up period. If, for instance, someone quit smoking for the first two years, relapsed in year three, and then quit again for years four and five, this person is not classified as a quitter by EQ, but it seems likely that they would have gained almost as much weight as someone who quit for all five years. 1114 people in the treatment group quit smoking in at least one follow up wave but were not sustained quitters. The second type contains those who reduced smoking but did not quit entirely. Among those in the treatment group who never quit in any of the five follow-up waves, average cigarettes smoked per day still fell from 31 to 22. There is no reason to suspect that the biological pathways through which smoking affects weight occur only along the extensive margin of smoking, so people who cut back on smoking would likely experience at least some amount of weight gain. Additionally, some people may be a blend of the two types; e.g. someone who responds to the intervention by gradually cutting back on smoking until successfully quitting at the end of the third year.

After replicating EQ’s results using the “sustained quitting” variable, we then examine the sensitivity of the results to the use of our more nuanced long-run smoking measures discussed earlier in Section 3.1.2. Our “average quitter” measure addresses the issue of people who quit in some but not all follow-up years. The average cigarettes per day and average CO variables also address the issue of cutting back but not quitting entirely.

Table 7 reports the results. The first column shows that, replicating EQ’s Wald estimator, we obtain an average estimated weight gain from quitting smoking of 3.196 BMI units, or 20.04 lbs, at the average height. This is very similar to the result obtained by EQ, differing slightly because EQ used weight as the dependent variable rather than BMI. (We are unable to directly use weight because the LHS microdata suppress height and weight and only provide BMI.) The second column shows that using simple average quitter rather than sustained quitter reduces the average estimated weight gain from quitting smoking by about 17% to 2.655 BMI units. In the last two columns, we see that using the simple averages of

### Table 6

<table>
<thead>
<tr>
<th>Cigarettes</th>
<th>Quit attempt prior to LHS</th>
<th>No quit attempt prior to LHS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Short run</td>
<td>Long run</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>0.052***</td>
<td>0.064***</td>
</tr>
<tr>
<td></td>
<td>(0.004)</td>
<td>(0.008)</td>
</tr>
<tr>
<td>N</td>
<td>152</td>
<td>184</td>
</tr>
<tr>
<td>CO</td>
<td>0.077***</td>
<td>0.079***</td>
</tr>
<tr>
<td></td>
<td>(0.006)</td>
<td>(0.012)</td>
</tr>
<tr>
<td>N</td>
<td>172</td>
<td>173</td>
</tr>
</tbody>
</table>

Notes: “Short Run” indicates IV regressions of year 1 BMI on the smoking variable. “Long Run” indicates IV regressions of year 5 BMI on the simple average of the smoking variable. Heteroskedasticity-robust standard errors are in parentheses. *** and ** indicate significance at the 1, 5, and 10 percent levels. The implied average effects of quitting smoking are in brackets. The controls for education, gender, marital status, age, and baseline BMI are included. Data come from the LHS.

### Table 7

<table>
<thead>
<tr>
<th>Sustained quit (EQ)</th>
<th>Average quit</th>
<th>Average cigarettes</th>
<th>Average CO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking variable</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3.196***</td>
<td>2.655***</td>
<td>0.063***</td>
<td>-0.072***</td>
</tr>
<tr>
<td>(0.736)</td>
<td>(0.605)</td>
<td>(0.015)</td>
<td>(0.021)</td>
</tr>
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<td>N</td>
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<td>5446</td>
<td>4966</td>
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<tr>
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<td></td>
<td></td>
<td>4517</td>
</tr>
</tbody>
</table>

Notes: Results are from IV regressions of year 5 BMI on the smoking variable. Heteroskedasticity-robust standard errors are in parentheses. *** and * indicate significance at the 1, 5, and 10 percent levels. For the non-binary smoking measures, the implied average effect of quitting smoking is in brackets. No control variables are included. Data come from the LHS.
cigarettes per day and CO attenuates this magnitude even further, to 1.84 and 1.58 BMI units, respectively. Ultimately, then, accounting for both temporary/delayed quitting and smoking intensity reduces the estimated average weight gain from smoking cessation by 42–51% relative to using the naive sustained quitter measure. Since the Wald estimates using our preferred smoking measures from Table 8 are quite similar to those from our preferred long-run specifications in Table 2, we conclude that the difference between our results and those of EQ is due to the different smoking measures rather than our use of a covariate-adjusted regression model in Table 2. This is not surprising given the randomized design. Note, however, that the standard errors are lower in Table 2, so including covariates is still beneficial in that it improves the precision of the estimates.

We now provide direct evidence that IV estimators featuring the sustained quitter smoking measure are problematic. We do this by estimating the effects of the treatment variables on the BMIs of individuals who were not sustained quitters, which essentially tests the exclusion restriction. Specifically, we estimate a reduced-form model of year 5 BMI on \( si_a \) and \( si_p \) for the subsample of individuals who were not medically validated quitters in at least one of the five follow-up waves. For purposes of comparability, we also run the same reduced-form regression for the full sample as well as subsamples consisting of those who made minimal changes to their smoking habits according to our average quitter and continuous measures.

Table 8 reports the results. The first column presents the reduced-form results for the full sample. The second column restricts the sample to non-sustained-quitters. The third column restricts the sample to those with a value of zero for the average quit measure, meaning that they did not quit smoking in any of the follow-up waves; i.e. any changes in their smoking were along the intensive margin. The fourth and fifth columns show the results obtained when we exclude those with greater than a 2.5% change in number of cigarettes smoked relative to baseline, while the fourth column does the same for CO. (Results are robust to the use of 10%, 15%, 20%, and 30% cutoffs.) Note that the sample sizes in the analyses based on CO are very small because, as discussed previously, much of the sample is missing baseline CO information, preventing the calculation of the percentage change.

The full-sample results show that, in both the short and long run, the reduced-form effects of the two treatment variables on BMI are between 0.54 and 0.61 before excluding any observations. Dropping sustained quitters reduces the magnitude of these effects, but significant effects of 0.25–0.26 remain. There is therefore clear evidence that the intervention affected BMI through a pathway besides sustained quitting. Further dropping those who quit smoking in some but not all follow-up waves does not do much to alleviate the problem, as the effects remain significant and are only slightly smaller at 0.21–0.25. In contrast, in the two regressions that exclude those with >25% changes in average cigarettes per day, the coefficient estimates for the treatment variables are small (between −0.014 and 0.038) and highly statistically insignificant. Excluding on the basis of changes in CO also leads to highly insignificant effects, with three being negative (the opposite direction of the full-sample relationship) and one positive, though the estimates are imprecise due to the small sample size. In sum, the exclusion restriction appears substantially more troublesome in models using binary smoking variables than in those using continuous measures.17

4. Conclusion

This paper aimed to provide the most credible answers to date to several questions related to the relationship between smoking and weight. First, what is the average short-run causal effect of quitting smoking on body weight? Our preferred estimates suggest that this effect is around 1.5–1.7 BMI units, or 10–11 pounds at the average height. Second, does the weight gain from quitting smoking disappear over time? The answer appears to be no, as the weight gain rises to around 11–12 pounds in the long run. Third, how does the impact of smoking on weight vary across the smoking distribution? We find evidence of a diminishing marginal effect, with additional smoking having little long-run impact beyond about a pack of cigarettes per day or a CO level of 20 ppm. Finally, how does the effect of smoking on weight vary by education, age, and baseline BMI? Our results suggest that, while quitting smoking leads to sizeable weight gain for all subsamples, the impacts are largest for those with no college degree, younger individuals, and those with low baseline BMIs.

Our estimated average effects of quitting smoking on weight fall within the range of estimates from the associational public health literature, albeit toward the high end of the range. It is clear, though, that our estimates are markedly smaller than those of EQ despite the fact that they utilized the same randomized intervention. The fact that our results are closer to the associational estimates than to those of EQ illustrates a broader methodological point about the dangers of using IV estimation uncritically even when the instrument is randomized as (as-good-as)-randomized. The randomization merely ensures the validity of the estimated reduced-form relationship between the instrument and outcome. Obtaining a reliable second-stage estimate requires the assumption that the endogenous variable is the only pathway through which the randomized instrument affects the outcome. This can be a difficult assumption to satisfy. As our paper shows, even if conceptually there is only one pathway through which the intervention can plausibly impact the outcome, careful measurement of that pathway is critical.

Our finding that the weight gain from smoking cessation increases slightly after several years also provides valuable new

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17 One potential concern with the analyses in this section is that, by selecting the sample based on changes in smoking that are endogenous to the treatment, we lose the ability to claim that unobservables are balanced across treatment and control groups. However, Appendix Tables 2–5 show that the summary statistics for the baseline values of the variables used in each subsample regression rarely exhibit statistically significant differences between the treatment and control groups. The fact that observable characteristics (particularly baseline BMI) are still reasonably well balanced across groups after imposing the sample restrictions increases our confidence that imbalance among unobservables is not meaningfully affecting the results.
information, as the issue of whether this weight gain is only temporary has been the subject of debate (Williamson et al., 1991; Chen et al., 1993; Flegal et al., 1995; Mizoue et al., 1998; Traver et al., 2012). Body weight accumulates over time (Philipson and Posner, 1999), and evidence suggests that BMI is often more responsive to exogenous shocks in the long run than the short run (e.g. Courtemanche, 2009, 2011; Goldman et al., 2011). The fact that we observe such a phenomenon here suggests that the effects of quitting smoking on appetite and metabolism persist over time and are not eventually offset by positive behavioral changes, such as beginning an exercise routine after lung capacity improves. Knowledge of this persistence can potentially help inform medical professionals and help patients realize the greatest possible health benefits when quitting.

Our results also have interesting implications for the economics literature on tobacco control policies. As discussed in the introduction, the literature on the effect of cigarette costs (prices or taxes) on BMI reaches conflicting conclusions, with some studies suggesting the effect is either very small or negative – implying that quitting or reducing smoking actually leads to weight loss. Is it possible that the causal effect of an aggressive smoking cessation program is to increase BMI while the causal effect of higher cigarette costs is either zero or negative? This seems conceivable for two reasons.

First, the LATE from a price-induced reduction in smoking may differ from the LATEs from smoking ban-induced or aggressive smoking cessation program-induced reductions in smoking. Different smokers could be affected by these different types of interventions; for instance, it seems reasonable to think marginal smokers would be the ones to respond to cigarette price increases whereas those with strong addictions would be the ones to volunteer for a comprehensive program. Perhaps those with strong addictions are relatively more likely to “quit at all costs”, even if it means gaining a substantial amount of weight. The results related to external validity from Section 3.4 provide some evidence against this hypothesis, but the possibility cannot be ruled out completely.

Second, perhaps cigarette prices/taxes affect BMI through pathways besides smoking behavior. In other words, people who quit smoking in response to higher cigarette prices may gain weight, but this could be counteracted by weight losses among those whose cigarette consumption is unchanged – a large share of the population given the price inelasticity of cigarettes (Chaloupka and Warner, 2000). Smokers who do not reduce their consumption when prices rise experience potentially sizeable negative income effects, which could lead to weight loss by reducing overall food consumption or frequency of eating out at restaurants. Moreover, cigarette taxes generate revenue for the state, which can be used to provide funding for nutrition education or health-related programs such as Medicaid. These, in turn, could reduce the BMIs of even non-smokers.

To close, we should emphasize that our results should not be interpreted as suggesting that individuals should be reluctant to quit smoking out of fear of gaining weight. The large body of epidemiological evidence that smoking is bad for health implies that any increase in obesity-related ailments after quitting smoking is far outweighed by the health improvements along other dimensions. Instead, our results fit with the broader literature that characterizes the rise in obesity as a side-effect of otherwise beneficial technological and societal changes such as improved food production, preservation, and distribution technologies, a shift toward white-collar employment, and the Interstate Highway System (Cutler et al., 2003; Lakdawalla et al., 2005; Zhao and Kaestner, 2010; Courtemanche and Carden, 2011). In such cases, the policy objective is not to undo these changes, but instead to target the mechanisms through which they lead to weight gain.

To that end, our findings should be interpreted as a call for further investigation into medical and policy interventions that can limit the weight gain from smoking cessation. Farley et al. (2012) provide a review of the literature on the effectiveness of various interventions in limiting the weight gain after quitting smoking. Some evidence suggests that the drugs dexfenfluramine, phenylpropanolamine, naltrexone, bupropion, and fluoxetine can reduce weight gain in the short run, though there is insufficient evidence to draw clear conclusions about whether the effects persist after the drugs are discontinued. Additionally, these drugs can have serious, potentially fatal side effects, and dexfenfluramine and phenylpropanolamine have been banned by the Food and Drug Administration. Weight management education alone does not seem to reduce weight gain and might actually hinder efforts to quit smoking, whereas weight management education combined with personalized support appears more successful. For all types of interventions reviewed by Farley et al. (2012), their ability to draw clear conclusions was hindered by a lack of available research and small sample sizes in the studies that do exist. Much more research is needed regarding which interventions can safely help limit weight gain following smoking cessation as well as how best to incentivize (e.g. more generous insurance coverage) interventions that prove effective.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jhealeco.2017.10.006.

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