The Unintended Health Consequences of Cigarette and Alcohol Taxes

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THESIS

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I dedicate this thesis to the members of Darwinian Daycare, a true dynasty of free pub trivia in Chicago.

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List of Abbreviations

ACCRA: American Chamber of Commerce Researchers Association

BMI: Body Mass Index

BRFSS: Behavioral Risk Factor Surveillance System

FE: Fixed Effect

NCD: Noncommunicable Disease

Summary

Smoking and drinking account for more than 520,000 deaths annually in the United States, and have been a frequent target of public policy aimed at improving health outcomes. They are also two of the four major risk factors for the world's deadliest killer: non-communicable diseases. However, our understanding of how smoking and drinking are related to other risk factors for these diseases, such as obesity, is limited. The purpose of this dissertation is to address this lack of understanding. Using the Behavioral Risk Factor Surveillance System and state-level excise taxes on packs of cigarettes and gallons of beer, I estimate the impact of these taxes on Body Mass Index, a measure of body fat used to diagnose obesity, along with their impact on hypertension, diabetes, cholesterol, and arthritis, all obesity-related health conditions. The results indicate that higher taxes on cigarettes and alcohol leads to lower weights, a lower risk of diabetes, and in the case of cigarette taxes, a lower risk of obesity. Higher cigarette taxes lower the rates of hypertension, but higher alcohol taxes have the opposite effect, which is consistent with the j-shaped relationship found in the medical literature. These effects should be considered when changes to these tax rates are considered.

1 INTRODUCTION

1.1 PURPOSE

Worldwide more than 36 million people die from non-communicable diseases (NCDs) such as cardiovascular disease, cancer, respiratory diseases, and diabetes (WHO, 2013). These deaths represent 63% of all deaths worldwide, making NCDs the world's biggest killer. Where once NCDs were the worry of rich countries, now roughly 90% of those under the age of 60 killed by an NCD were living in the developing world. Increasingly, NCDs have been claiming the lives of those living in low- and middle-income countries. As such, the prevention of NCDs has become a major focus of health policy around the world.

Non-communicable diseases share four common risk factors in smoking, alcohol abuse, poor diet, and a lack of exercise. These primary risk factors lead to intermediate risk factors such as high blood pressure, high cholesterol, and being overweight or obese. Together these preventable risk factors account for nearly 40% of deaths worldwide (WHO, 2011). However, not much attention has been paid to how policies which seek to reduce one of these risk factors may affect the others. Policies aimed at reducing smoking or alcohol abuse may lead to changes in diet and exercise that reduce or enhance the effectiveness of the policy.

Nearly 90% of countries provide some funding for the prevention and control of NCDs, and funding has been on the rise over the past decade (WHO, 2011). Most of these efforts have focused on monitoring, restricting the use of tobacco and alcohol, taxing tobacco and alcohol, and raising awareness through mass media. Many of these interventions now being implemented in low- and middle-income countries were first introduced in high-income countries like the United States. An important contribution being made in the scientific literature has been the assessment of policies in high income countries which build an understanding of their effect and make their implementation in the developing world easier and more effective.

In the research I present here, I aim to make a contribution to this literature by exploring how people respond to taxes aimed at reducing cigarette and alcohol consumption, with a focus on obesity and obesity-related health outcomes. Obesity is the result of an imbalance between caloric energy consumed and caloric energy expended, and can be measured by the Body Mass Index (BMI), an estimate of a person's body fat percentage equal to their weight in kilograms divided by their height in meters squared. Cigarette smoking has long been associated with weight loss by acting as an appetite suppressant, but may also be impacting diet and exercise in a way that leads to weight gain. As a direct source of calories, alcohol consumption can directly contribute to an energy imbalance that results in obesity. These changes in weight may also be a factor in obesity-related health outcomes like hypertension, diabetes, and high cholesterol. Taxes on cigarettes and alcohol exogenously influence smoking and drinking decisions, and can be used to understand these relationships for the drinking and smoking population along with being an important point of study themselves.

The data I will use to estimate these impacts comes from the Behavioral Risk Factor Surveillance System (BRFSS) which is comprised of repeated cross-sections from 1984 to 2010 and contains self-reported weights, heights, other demographic information, and health behaviors. State-level excise taxes placed on packs of cigarettes are collected from *The Tax Burden on Tobacco*. State-level excise taxes placed on gallons of beer, acting as a proxy for all alcohol taxes, come from *The Brewers Almanac*. To estimate the effects of these taxes on body weight, obesity, and obesity-related health conditions, I use multiple regression, and where appropriate I estimate a logit model, on the premise that the taxes are exogenous influences on smoking and drinking decisions when controlling for state and year fixed effects.

While the primary focus of this research is to find policy-relevant reduced-form estimates, an instrumental variables approach can be applied to a structural model to find the impact of a behavior on an outcome. I estimate these models where appropriate and possible to find the impact of smoking and drinking on BMI

and health outcomes. In some cases, these estimations illuminate important groups moved by policy and also provide an opportunity to test the identifying assumptions of the research design.

In the chapter that follows, I examine the impact of cigarette taxes on body weight and obesity. Conventional wisdom has long held that smoking can be used as a method of weight control, and the medical literature suggests that quitting smoking leads to weight gain. However, using state-level excise taxes on cigarettes as an exogenous source of variation in smoking behavior and multiple regression analysis to estimate the model for a large sample of cross-sectional data over multiple decades, I find that a one dollar increase in the tax on cigarettes reduces smoking and leads to 0.08 point decline in BMI. For a person of average height, which translates to a loss of about 0.5 pounds.

To better understand this effect, I look separately at those who are of normal weight, overweight, and obese both by ordinary least squares and by quantile regression to find that the effect cigarette taxes have on weight is larger for those of higher weights. This, combined with evidence that those who smoke heavily are more likely to be obese than light smokers, suggests that the effect is being driven by obese smokers. Further, reducing ones smoking may have different effects on weight than quitting smoking, which may explain why these results run contrary to the conclusions in the medical literature.

I also look at the effect of smoking bans in restaurants, bars, and private workplaces on BMI. While none of these policies have a statistically significant impact on weight, all of the effects are negative. These policies also provide a second source of exogenous variation in smoking that I can use to over-identify the model and test whether or not one of these policies violates the exclusion restriction. I find no evidence to reject the identifying assumption that the tax or bans operate exclusively through changes smoking.

Lastly, I examine the mechanisms of the observed weight change using measures of fruit and vegetable consumption, exercise, and doctor's visits. I find a significant increase in the number of fruits and vegetables associated with an increase in the excise tax on cigarettes, but no significant changes in exercise or doctor's

visits. These results suggest that the primary mechanism of change is in diet. The reduction in smoking may be associated with more food consumption, but may also lead to a change in diet that reduces overall calorie intake, and results in weight loss.

Overall, chapter 2 provides strong evidence that increases in the cigarette tax lead to weight loss primarily for obese smokers. A one dollar increase in the tax is estimated to lead to a 0.5 percentage point reduction in obesity, implying 1.2 million people would move out of the obese classification. These effects should be considered when proposed tax increase are considered.

Chapter 3 examines the impact of alcohol taxes on body weight. Alcoholic beverages are a major source of caloric energy for Americans, providing 4.4% of total caloric intake (Block, 2004). However, evidence is mixed as to whether or not alcohol consumption is a contributing factor to obesity. Using state-level excise taxes placed on gallons of beer as an exogenous influence on drinking behavior, I find that a one dollar increase in the tax reduces consumption of alcoholic beverages and leads to a 0.08 point decline in BMI. If the one dollar tax were collected per 12 oz. of beer, these results suggest the average person would lose about 5 pounds.

The tax on beer can also be used to estimate a structural model that identifies the impact of drinking on BMI. Instrumenting for the average number of drinks consumed per day with the tax, I find that an additional drink per day leads to a 1.7 point increase in BMI. For a person of average height, this corresponds to about 10 pounds. Based on population estimates for resting metabolic rates, an increase in body weight of 10 pounds would require an additional 100 calories worth of consumption. This is in line with the average calorie counts of alcohol beverages, which ranges from about 97 calories to 153, and suggests that additional alcohol consumption is not offset by other dietary changes and is largely additive in calories.

As with cigarette taxes, there also appear to be differences in the magnitude of the effect across the BMI spectrum, with those classified as obese losing more weight in response to a tax increase than those of normal weight. These effects do not seem to be permanent, however. Lags of the beer tax produce significantly smaller effects the greater the lag. While alcohol does appear to play an important role in obesity, these taxes do not appear to be an effective policy tool in reducing obesity.

Chapter 4 examines the impact of both cigarette and alcohol taxes on obesity-related health conditions. These conditions include hypertension, diabetes, high cholesterol, and arthritis. The data for these conditions are self-reported instances of being told by a doctor that the respondent had one of these conditions. Like the previous chapters, these effects are estimated using state-level excise taxes on packs of cigarettes and gallons of beer as exogenous sources of variation in smoking and drinking.

Smoking is not directly related to hypertension, but cigarette taxes may impact hypertension rates through their effect on weight and obesity. The results indicate that this is the case, with a one dollar increase in cigarette taxes leading to 0.8 percentage point decline in the prevalence of hypertension. A similar effect is seen for diabetes, where smoking is a risk factor in its own right, and a one dollar increase in the cigarette tax leads to a 0.24 percentage point decline in prevalence. Together, these effects represent 523,000 fewer cases of hypertension, and 63,000 fewer people with diabetes.

Alcohol consumption has a more complex relationship with hypertension and diabetes. The medical literature suggests a j-shaped relationship, with 1-2 drinks per day having a protective effect against both conditions, but heavier drinking leading to an increased risk (Sesso, 2010). There may also be an impact due to changes in weight and a higher risk of obesity. The results suggest that higher alcohol taxes increase the risk of hypertension, with a one dollar increase leading to a 0.25 percentage point increase in prevalence. The same tax increase appears to reduce the prevalence of diabetes by 0.15 percentage points,

however. These effects correspond to about 170,000 more cases of hypertension, and 39,000 fewer cases of diabetes with a one dollar rise in the tax.

Neither the cigarette tax nor the beer tax had a significant effect on instances of high cholesterol or on arthritis. In the former case, this is contrary to expectations. Smoking and drinking are only loosely tied to cholesterol levels, but should be tied through the mechanism of weight change and corresponding changes in diet and exercise. In the case of arthritis, the lack of an effect is expected, and the presence of an effect would more likely be the result of problems with the research design. Smoking and drinking are only tied to relatively rare forms of arthritis, and would only be tied to the more common form, osteoarthritis, through a higher risk of obesity. But the effect of cigarette and beer taxes on weight are only suggestive of small changes to weight, which by themselves should not have a significant impact on cases of arthritis.

Across the three chapters, these results tell an important story. Cigarette smoking and alcohol consumption are tied to body weight and obesity in a significant way. Higher taxes on these goods lead to lower weights and in the case of a cigarettes, a reduced risk of obesity. These effects also impact other health outcomes. Higher taxes on cigarettes leads to fewer cases of hypertension and diabetes, the former being a condition only tied to smoking through its effect on diet and exercise. Higher taxes on alcohol leads to an increase in hypertension rates, confirming the j-shaped relationship found in the medical literature, but fewer cases of diabetes.

All of these results represent unintended consequences of those policies, which were conceived largely to address the externalities of second-hand smoke and alcohol abuse, prevent cancer and liver disease, or to simply raise revenue. Understanding those unintended consequences will be important for the many developing countries which seek to avoid the experience of the United States with regard to the detrimental health consequences of alcohol and tobacco consumption.

1.2 Analytical Framework

1.2.1 Body Weight and the Prices of Cigarettes and Alcohol

The primary research goal of this dissertation is to estimate the impact of smoking and alcohol taxes on body weight and other health outcomes. Since these taxes operate through the price of cigarettes and alcohol, it is important to lay out a model for how these prices impact weight and health. Weight is determined by a variety of factors which includes calories consumed, energy expended by activity, metabolism, and other genetic factors. To eliminate some of the differences between individuals in metabolism and genetics, I will measure weight using the Body Mass Index (BMI), which accounts for differences in height.

BMI is determined by a person's weight in kilograms divided by their height in meters squared. Keys et. al. (1972) gave the index its name and found that it was the best of several different measures in estimating a person's body fat percentage. It is the weight due to body fat that is of concern here, and so BMI will serve the purpose of this work well. Following the approach of Courtmanche (2009), a person's BMI (B) is determined by their intake of food calories (F) and their expenditure of calories either through activity (E) or metabolism (M). Therefore,

$$B = B(F, E, M) \tag{1}$$

where $\frac{dB}{dF} > 0$, $\frac{dB}{dE} < 0$, and $\frac{dB}{dM} < 0$.

Smoking is directly related to both food intake and energy expenditure. Conventional wisdom suggests that smoking reduces weight by suppressing the appetite, and acting as a stimulant which raises the metabolism. Indeed, these perceptions hold true in the medical literature (Jo, Talmage, & Role, 2002; Pinkowish, 1999). However, reduced smoking may also influence the consumer's diet and exercise behavior by making investments in improved health more attractive. Becker (2007) lays out a model for health which

concludes that reductions in the probability of dying from one disease raises the marginal benefit of investments that reduce the risk of dying from other diseases. In this case, a reduction in the risk of cancer or heart disease due to reduced smoking provokes increased investment in preventing the adverse health consequences associated with poor diet and a lack of exercise. This is demonstrated to some degree by Picone and Sloan (2003), who find that men who quit smoking also consume less alcohol. Cigarette smoking also reduces lung capacity, which raises the costs of exercise (Dept. of Health and Human Services, 2004).

Alcohol consumption directly contributes to food intake, as it is itself a source of caloric energy. However, there is some debate as to the degree to which changes in alcohol consumption result in changes in other food intake. Increased alcohol consumption may not lead to a change in total caloric intake through this offsetting (Wang et. al, 2010; Yeomans, 2010). Unlike cigarette consumption, however, alcohol consumption does not appear to be related to changes in exercise behavior or persistent changes in the metabolism (Raben et. al. 2003).

This evidence suggests that smoking should decrease exercise, increase the metabolism, but have an ambiguous effect on food intake. Reductions in smoking, then, will have an ambiguous effect on BMI. Alcohol consumption only influences food intake, though past evidence is mixed as to whether or not this effect is greater than or equal to 0. Putting this information into the model gives the following:

$$B = B\left(F\left(S(P_S), A(P_A)\right), E\left(S(P_S)\right), M\left(S(P_S)\right)\right) \tag{2}$$

Here I am assuming that the consumer's choice of cigarettes and alcohol depend on their respective prices P_S, and P_A, with income held constant. This offers the following result:

$$\frac{dB}{dP_S} = \frac{dB}{dF}\frac{dF}{dS}\frac{dS}{dP_S} + \frac{dB}{dE}\frac{dE}{dS}\frac{dS}{dP_S} + \frac{dB}{dM}\frac{dM}{dS}\frac{dS}{dP_S} \tag{3}$$

The previous discussion concludes that $\frac{dF}{dS}$ is indeterminate, $\frac{dE}{dS} < 0$, and $\frac{dM}{dS} > 0$. Assuming that $\frac{dS}{dP_S} < 0$, this implies that the overall effect of price changes for cigarettes is ambiguous, and therefore requires empirical estimation.

The relationship between alcohol prices and BMI is less ambiguous:

$$\frac{dB}{dP_A} = \frac{dB}{dF}\frac{dF}{dA}\frac{dA}{dP_A} \tag{4}$$

where the previous discussion concludes that $\frac{dF}{dA} \geq 0$. Assuming that $\frac{dS}{dP_A} < 0$, this implies that the relationship between alcohol prices and BMI should be negative or equal to zero, also making this a matter of empirical estimation.

1.2.2 Obesity-Related Health Outcomes and the Prices of Cigarettes and Alcohol

Non-communicable diseases such as hypertension, diabetes, and high cholesterol share common risk factors in smoking, alcohol abuse, poor diet, and a lack of exercise (WHO, 2011). The previous framework demonstrated that smoking and drinking can influence diet, and in the case of smoking could influence exercise as well. Thus, the relationship between smoking, drinking, and these non-communicable diseases may be more ambiguous than conventional wisdom implies. NCDs (N) are determined by food intake (F), exercise (E), smoking (S), and alcohol consumption (A).

$$N = N(F, E, S, A) \tag{5}$$

Hypertension is not directly tied to smoking (Dept. Health and Human Services, 2004). However, a more careful analysis reveals that smoking may influence hypertension through changes in diet and exercise. Similarly, alcohol has been shown to have a j-shaped relationship with blood pressure, with light-to-moderate drinking reducing risk (Sesso, 2010). These protective effects may be offset by alcohol's effect

on diet. These imply the following relationship between hypertension (H), and the four risk factors for NCDs:

$$H = H\left(F\left(S(P_S), A(P_A)\right), E\left(S(P_S)\right), S(P_S), A(P_A)\right) \tag{6}$$

where $\frac{dH}{dF} > 0$, $\frac{dH}{dE} < 0$, $\frac{dH}{dS} = 0$, and $\frac{dH}{dA}$ is indeterminate.

The relationship between hypertension and cigarette prices is then:

$$\frac{dH}{dP_S} = \frac{dH}{dF}\frac{dF}{dS}\frac{dS}{dP_S} + \frac{dH}{dE}\frac{dE}{dS}\frac{dS}{dP_S} + \frac{dH}{dS}\frac{dS}{dP_S} \tag{7}$$

Since the last term in this equation is zero, the relationship between cigarette prices and hypertension depends on the relationship between food intake and smoking. As noted in the previous section, this relationship is theoretically ambiguous.

Alcohol prices and hypertension are related in the following manner:

$$\frac{dH}{dP_A} = \frac{dH}{dF}\frac{dF}{dA}\frac{dA}{dP_A} + \frac{dH}{dA}\frac{dA}{dP_A} \tag{8}$$

Here the first term is less than or equal to 0, while the second term is ambiguous. Thus, the relationship between hypertension and alcohol prices will be determined by three factors: the relationship between food intake and alcohol, the relationship between hypertension and alcohol, and which of the two dominates in the case that they differ in their direction.

Similar procedures can be followed for the relationship between these prices and diabetes and high cholesterol. In both cases, however, smoking has a direct and negative relationship (Dept. Health and Human Services, 2010). This implies that the overall relationship not only depends on the direction of the relationship between food intake and smoking, but also whether or not that effect, if it is negative, is large

enough to dominate. If smoking leads to reduced food consumption, however, then the relationship between cigarette prices and these health outcomes are unambiguously negative.

In every case, though, empirical estimation is required to examine the relationship.

1.3 DATA

All three of the analyses to follow draw on the Behavioral Risk Factor Surveillance System (BRFSS). The BRFSS has been conducted by the Centers for Disease Control (CDC) annually since 1984 and contains repeated cross-sections of randomly selected individuals who respond to a telephone interview. These data contain self-reported measures of height and weight, allowing me to construct BMI and indicators of obesity, along with other demographic variables such as age, race, gender, marital status, education, income level, and employment status. The BRFSS also contains information about health-related behaviors such as smoking, exercise, and alcohol consumption in addition to information about health outcomes such as diagnosed hypertension, diabetes, arthritis, and high cholesterol. More detail on these variables will be discussed when relevant.

The CDC collects these data in conjunction with state health departments via telephone surveys, initially operating in 15 states in 1984, and eventually expanding to all 50 states and the District of Columbia by 1994. The initial 1984 wave of observations per state ranged from 604 in Tennessee to 1,501 in Minnesota, with an average of about 800 per state. The average number of observations grew to 1,800 across 45 states in 1990, to 3,500 across all 50 states and D.C. in 2000, and to 7,800 in 2010, the last year in the dataset used.

The BRFSS data are weighted based on national proportions of age, race, gender, and geographic region using a post-stratification method for years prior to 2011. These final sample weights are representative of the population at large and are employed in all of my regression models to ensure the consistency of

estimates (DuMouchel & Duncan, 1983). In 2011, the BRFSS switched to iterative proportional fitting to generate its sample weights to include more demographic variables as well the proportions of telephone and cellular phone ownership. Additional weighting variables included education level, marital status, and home ownership. The BRFSS uses random-digit dialing to call potential respondents, but did not include cellular phones until 2011. Because of this change in sampling and weighting methods, data for 2011 and 2012 are not used in the analysis.

The advantage these data offer is the number of observations. Across the 26 years sampled, usable data was gathered for more than 4.7 million people. Since the goal in much of the analysis is to estimate a relatively small effect, a dataset of this size is needed to obtain precise estimates. Further, the scope of the data, spanning all 50 states and the District of Columbia for more than two decades, provides for ample variation in the policies that will be analyzed.

In addition to these data, I also use data from the American Chamber of Commerce Researchers Association (ACCRA) on prices across more than 300 U.S. cities for a variety of goods and services. These data were collected from 1990 through 2010, spanning most of the 26 years of BRFSS data. However, the BRFSS data contains consistent information only on the state of residence. To match the city-level ACCRA data with the individual-level data, a composite price was formed by taking quarterly statewide averages weighted by city populations.

These statewide average prices where then used to compose six different price indices. The indices were constructed using weights reported with the ACCRA data which are constructed based on the expenditure share of each item. These weights are calculated based on the Bureau of Labor Statistics Consumer Expenditure Survey. The six price indices constructed are for grocery food items, fruits and vegetables, fast food, alcohol, medical services, and exercise equipment. The components of these indices are described below.

Price Indices Constructed from ACCRA Data

Grocery Food Items: USDA Choice T-bone steak (price per pound), 80% lean ground beef (price per pound), whole fryer chicken (price per pound), Starkist or Chicken of Sea brand chunk light tuna (6.0-6.125 oz. can), Grade A large eggs (dozen), Blue Bonnet or Parkay brand margarine (one pound), the lowest price white bread (24 oz. loaf), cane or beet sugar (4 or 5 pounds), Kellog's Corn Flakes or Post Toasties (18 oz.), and Coca-Cola (2 liters).

Fruits and Vegetables: White or red potatoes (10 lb. sack), bananas (price per pound), lettuce (approx. 1.25 lb. head), Del Monte or Green Giant brand sweet peas (15-17 oz. can), Hunt's. Del Monte, Libby's, or Lady Alberta brand peaches (29 oz. can), and the lowest price whole kernel corn (10-16 oz.).

Fast Food: McDonald's Quarter-Pounder with Cheese, 11"-13" thin crust cheese pizza from Pizza Hut or Pizza Inn, and a thigh and drumstick from KFC or Church's.

Medical Services: Price of a routine examination of an established patient from a general practitioner, and the price of an adult teeth cleaning from a dentist.

Alcohol: Constructed only for the years 1990 to 2004 consisting of a 750-ml bottle of J&B Scotch, a 6-pack of 12 oz. Budweiser or Miller Lite beer (1990-1999), a 6-pack of 12 oz. Heineken's beer (2000-2004), and various 1.5 liter bottles of wine.

Exercise Equipment: Saturday evening non-league price per game of bowling, and a can of three Wilson or Penn brand tennis balls.

Along with these price indices, I will also use the price of a carton of Winston king-size cigarettes, data for which was collected from 1990 until 2003. Because these data are collected quarterly and at the city level, however, and the BRFSS data it is combined with is monthly and at the state level, there will be considerable

measurement error. Assuming this error is random based on the construction of these indices, it will only serve to bias estimates towards zero. However, the ACCRA data tends to be collected from large metropolitan areas, which may not be representative of a state at large. If this introduced a systematic bias that is correlated with BMI, it could skew the results of an estimation of the effect of these prices on BMI. All results using this data should be interpreted in this light, but importantly this potential problem in using this data merely adds to a list discussed in greater detail later.

Additional data on the state-level excise tax rates on cigarettes and beer were collected respectively from *The Tax Burden on Tobacco* and *The Brewer's Almanac*. These and other data will be described in more detail in the chapters to come. Data on the state-level monthly unemployment rate and the Consumer Price Index (CPI) were collected from the Bureau of Labor Statistics. All of the data mentioned here was merged into a single dataset and analyzed using the STATA/SE 12.0 statistical package.

2 The Impact of Cigarette Taxes on Body Weight and Obesity

2.1 Introduction

Conventional wisdom has long held that smoking can be used as a method of weight control. In the 1930s, Lucky Strike Cigarettes advised both men and women to "Reach for a Lucky instead of a sweet" to "keep a slender figure no one can deny." In economics, we would call smoking and sweets substitutes, and changes in the consumption of one will lead to changes in consumption of the other. It is for this reason that some have pointed at the efforts to reduce smoking over the past few decades to explain some of the rise in obesity over the same time period.

Research suggests that cigarette smoking suppresses the appetite (Jo, Talmage, & Role, 2002), and that concern over weight gain can often be a deterrent to quitting (Pirie et. al., 1992). Indeed, clinical studies have shown that about 79% of people who quit smoking gain weight, on average about 5 pounds (Dept. of Health and Human Services, 1990). Recent population studies have found mixed effects, however, with some studies pointing to large increases in weight with reduced smoking, and others suggesting weight loss with reduced smoking.

In this paper, I will estimate the impact of tax policy for cigarettes on body weight, and piece out the location and mechanisms of these effects. I use state-level excise taxes on cigarettes as an exogenous source of variation in smoking behavior and multiple regression analysis to estimate the effect for a large sample of cross-sectional data over multiple decades. I find that a one dollar increase in the tax on cigarettes leads to 0.08 point decline in Body Mass Index (BMI), a measure of body fat used to diagnose obesity. For a person of average height, this translates to a loss of 0.5 pounds. These findings are robust to changes in specification and consistent with the results found when using smoking bans in bars, restaurants, and private workplaces in place of the tax.

To advance the literature further, I use both multiple regression and quantile regression to estimate differences in the size of the effect across the BMI spectrum. The results indicate that the weight loss is substantially larger for those categorized as obese (BMI ≥ 30). This result may be partially explained by the fact that heavy smokers tend to have higher weights than light smokers.

Using data on the consumption of fruits and vegetables, exercise behavior, and doctor's visits, I attempt to piece out the mechanisms of this weight change. I find that increases in the cigarette tax are associated with an increase in fruit and vegetable consumption, but not associated with a change in exercise or doctor's visits. These results suggest that the mechanism responsible for the observed weight loss is dietary changes. Fruit and vegetable consumption is associated with a lower risk of obesity.

Lastly, I use both the tax on cigarettes and smoking bans for restaurants, bars, and private workplaces as instruments for smoking behavior to estimate the effect of smoking on BMI. The results are mixed, showing both large and small reductions in weight with changes in smoking, depending on the sample and measure of smoking used. Some of these discrepancies might be resolved by a larger dataset which measures the quantity of cigarettes smoked per day, but other explanations are explored as well.

Overall, the results provides strong evidence that increases in the cigarette tax lead to weight loss primarily for obese smokers through the mechanism of dietary change. A one dollar increase in the tax is estimated to lead to a 0.5 percentage point reduction in obesity, implying 1.2 million people would move out of the obese classification. These effects should be considered when proposed tax increase are considered.

2.2 BACKGROUND

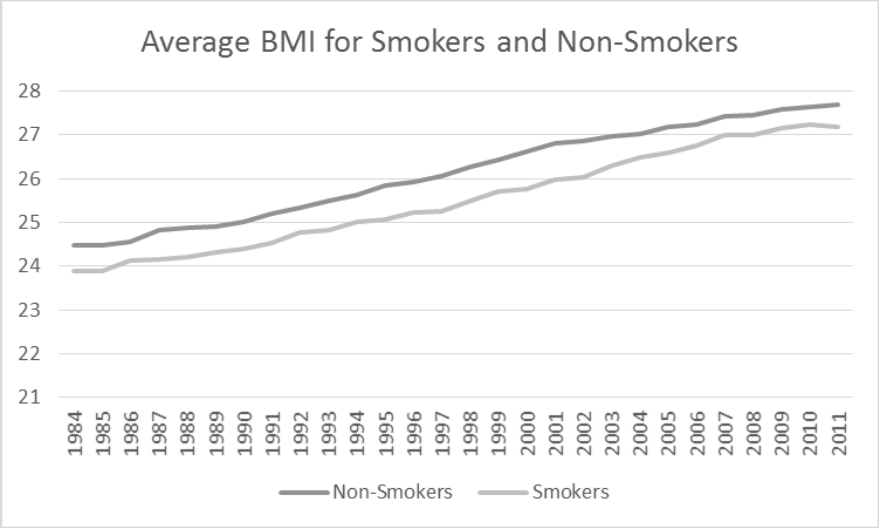
Several papers have examined the impact of smoking reduction on obesity. A 1990 U.S. Surgeon General's report reviewed 15 medical studies showing an average weight gain of about 5 pounds for those who quit smoking compared to those who did not (Dept. of Health and Human Services, 1990). These studies also

show that about 79% of quitters gained weight, compared to 56% of continuing smokers who gained weight over the same time period, which ranged from 1.5 months to 6 years with a median of 2 years. However, even taking just from the studies which followed up within three months, average weight gain for quitters was still around 4 pounds.

The evidence that smoking cessation leads to weight gain has led some to wonder how much of the rise in obesity over the past few decades is attributable to the concurrent fall in smoking rates. Data from the Behavioral Risk Factor Surveillance System (BRFSS) suggests that in 1984, the average American weighed about 157 pounds, and only 9% could be classified as obese. By 2011, the average American weighed 176 pounds, and roughly 27% could be classified as obese. Over the same period, smoking prevalence fell from 28% in 1984 to 14.2% in 2011 (CDC, 2011).

Observing that the fall in smoking over this period can be attributed in large part to an increase in the real price of cigarettes over this time period, a 2004 paper by Chou, Grossman and Saffer concludes that a significant portion of the weight gain may be "an unintended consequence of the anti-smoking campaign." Regressing BMI on the price of cigarettes, they find that these price increases explained 22% of rise in obesity between 1984 and 1999. The impact of cigarette prices on BMI was second only to the increases in the per capita number of restaurants in terms of explaining the trend in obesity.

Despite the correlation between smoking prevalence and obesity, smokers have seen an increase in body weight similar to non-smokers. Between 1984 and 2011, the average BMI of non-smokers rose 13.1% while the average BMI of smokers rose 13.8%. Further, though it is true that smokers tend to have lower weights than non-smokers, obese smokers are not less obese than obese non-smokers. The average BMI of obese smokers is 34.5, while the average BMI of obese non-smokers is 34.6. Smoking is not quite as strong a defense against obesity as some have suggested.



Responding to the results of Chou, Grossman and Saffer, Gruber and Frakes (2006) employed a similar methodology, but used the excise taxes on cigarettes rather than the price of cigarettes in order to estimate the impact of smoking reduction on body weight. The authors argue that excise taxes are "a more exogenous measure than price." Using the same BRFSS data, they find that a one dollar increase in the excise tax on cigarettes led to 0.15 point reduction in BMI. They attribute the change in the direction of this effect to the inclusion of year fixed effects instead of a quadratic time trend, and the use of taxes instead of prices.

Gruber and Frakes (2006) acknowledge the considerable magnitude of their results. Their instrumental variables regression suggests that quitting smoking due to an increase in taxes leads to an average 5.7 point reduction in BMI, which translates to 33 pounds for a person of average height. While they cite possible reasons for the result, such as increased exercise or a conscious effort to diet, they call for further research to better estimate the effects of smoking reduction on body weight.

One possible explanation for the Gruber and Frakes result is that the excise tax violates the exclusion restriction when instrumenting for whether or not a person smokes. The tax may still be operating through

the quantity of cigarettes smoked, producing an upwardly biased estimate. Additionally, Gruber and Frakes do not present any falsification tests to see if the tax if correlated with other state-level trends.

A 2006 paper by Rashad, Grossman, and Chou include cigarette taxes in a regression on BMI using data from the National Health and Nutrition Examination Survey (NHANES) and finds positive effects of the tax on BMI for woman only. They also look at the effects of smoke-free air laws which restrict the ability to smoke in certain places, such as private workplaces, restaurants, and bars. However, the authors assemble these bans into a single variable which is the sum of an indicator variable for each individual ban, making their results uninterpretable.

Baum (2009) sought to better control for state-specific time trends using panel data from the National Longitudinal Survey of Youth (NLSY). Baum finds that increasing in cigarette taxes by $0.77 would increase BMI by about 0.6 points, or about 3.5 pounds for a person of average height. Like the previous research, however, these results are significantly higher than those observed in the medical literature since the average of 3.5 pounds applies to the entire population, and not just to those who quit or reduced their smoking. Baum also finds similar effects for the cigarette price, reconciling some of the disagreement in the previous literature.

Adding to the debate is a 2009 paper by Courtemanche, which used the NLSY and the BRFSS, but allows for longer lags to estimate the long-term impacts of the cigarette tax on body weight. Courtemanche concludes that a $1 increase in the tax reduces average BMI between 0.13 and 0.59 points. These results appear robust to changes in the specification. Additional estimations in the paper suggest that changes in eating and exercise behaviors may explain the results, but Courtemanche calls for future research to better determine the path of these effects.

Nonnemaker et. al. (2009) further questions the methods of the original work done by Chou, Grossman, and Saffer (2004). Using the same methodology, the authors find highly significant effects of the price of

tennis balls and dentist visits on BMI. Dentist visits in particular seemed to explain 31% of the increase in BMI between 1990 and 1999. Nonnemaker et. al. instrument for cigarette prices using the cigarette tax, and also include state-specific time trends in order to estimate the impact of smoking reduction on body weight. They find small but positive coefficients more consistent with the medical literature, but not statistically significant. Nonnemaker et. al. demonstrate the potential for unobserved heterogeneity bias when using prices by showing that the price of tennis balls and dental visits had large and significant effects on obesity, but their final specification leaves little variation to estimate on.

Overall, the preponderance of evidence suggests that increases in the cost of cigarettes leads to decreases in average weights. However, there is little research which attempts to determine the pathways of this effect, or their differences for different segments of the population. Smokers may still be substituting smoking for more calories, but compensating for this shift with an increase in physical activity, shifting to a healthier diet, or by making other investments in their health. The consequences of these changes in weight due to changes in cigarettes taxes have also been largely unexplored.

2.3 METHODS

Following the analytical framework laid out in section 1.2, I use state level taxes on cigarettes as an exogenous source of variation on smoking behaviors operating through the price of cigarettes. To find the effect of these taxes on BMI and obesity, I estimate the following reduced-form equation:

$$BMI_{its} = \beta_0 + \beta_1 CigTax_{ts} + \beta_2 X_{its} + \beta_3 UR_{ts} + Year_t + State_s + \varepsilon_{its}$$

where *i* denotes the individual, *t* denotes the time, and *s* denotes the state. *BMI* is the individuals body mass index, *CigTax* is the excise tax placed on cigarettes, *X* is a set of individual-specific covariates, *UR* is the state-level unemployment rate in time *t*, and *Year* and *State* are year and state fixed effects respectively. The individual covariates contained in *X* include age group, gender, race, education level, marital status, and income.

Controlling for state and year trends, broader state-level economic conditions, and individual-specific covariates, cigarette taxes serve as an exogenous influence on cigarette consumption. Therefore, in this model it is differences in these tax rates within states, compared to other states without a change in their rates that will estimate the causal impact of these taxes on body weight.

To see if these results are consistent with other policy changes, I also look at the effect of smoke-free air laws which prohibit smoking in bars, restaurants, and private workplaces on BMI. Further, I can look for evidence that the tax is endogenous by bringing in covariates separately. If the tax on cigarettes is related to age, say, then the estimate should change as age is added in. However, independence from covariates related to both smoking and BMI is supportive of the notion that the tax is independent from all factors which impact smoking and BMI. I can also look for an effect on a variable which is not impacted by the tax, such as height, in search of potential endogeneity.

The next step of the analysis is to estimate differential impacts and the mechanisms of the weight change. First I will estimate the same equation as above separate for those classified and being of normal weight, being overweight, and being obese. Then, to get a better idea of the differences in the size of the effect at different levels of BMI, I use quantile regression to estimate the same equation. I will also estimate the impact of the tax on smokers and non-smokers by again separating the analysis for each group.

Smoking may be related to weight through several different channels. The most forefront of these channels is through changes in diet. These changes may not just be in total calorie consumption, but also in the composition of those calories. To get a sense of how much this mechanism matters, I will estimate the impact of the cigarette tax on fruit and vegetable consumption. Increased consumption of fruits and vegetables is associated with a lower risk of obesity (He, et al., 2004). If the tax is shown to have an effect on their consumption, this would indicate that cigarette consumption is a substitute for them, which could have weight-related consequences.

Another channel in which smoking might impact weight is through changes in health which make physical activity easier. Smokers have lower lung capacities and a lowered physical stamina which may reduce their calorie expenditure and effect weight (Dept. of Health and Human Services, 2004). If this is a primary mechanism by which the tax affects weight, then the tax should also affect exercise behaviors.

Smoking may also be impacting weight through the complementarities in health. Following a model similar to the one laid out in Becker (2007), reduced smoking may spur other investments in health, such as lowering the risk of obesity-related illnesses by losing weight. These investments are made more beneficial by the reduced smoking, and should result in an observable change in other health investments. To determine whether or not this is a significant mechanism of the observed effect of the tax on BMI, I will estimate the effect of the tax on doctor's visits, which serve as a proxy for health investments.

2.4 DATA

The primary source of data is the Behavioral Risk Factor Surveillance System (BRFSS), which contains self-reported information on weight, smoking, and other demographic variables. This data is described in detail in section 1.3 along with additional data used from *The Tax Burden on Tobacco* and the *ACCRA Price Index*. Data on smoke-free air laws comes from *ImpacTeen*, and contains indicators for whether or not there was a state level ban on smoking in bars, restaurants, or private workplaces in a given year between 1990 and 2010. Because this data is annual, there is significant measurement error when it is merged with monthly observations. This error should only bias the estimates toward zero, but will limit the conclusions that can be drawn from the data.

The BRFSS data also contains self-reported information on various health-related behaviors, which may shed light on the mechanisms by which weight change occurs. Three in particular have been selected for this analysis and are described below.

BRFSS Health Behaviors

Fruit and Vegetable Consumption: A calculated variable estimating the average number of servings of fruits and vegetables the respondent consumes per day based on their responses regarding the frequency and quantity of consumption. This variable cannot be calculated for the years 2004, 2006, 2008, and 2010.

Exercise in the Past 30 Days: An indicator variable equal to 1 if the respondent answered "Yes" to the question, "During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?" and equal to 0 if they responded "No."

Doctor's Visit: An indicator variable equal to 1 if the respondent reported visiting the doctor for a routine checkup in the past year, and a 0 if they last visited a doctor for a routine checkup more than one year ago. A routine checkup is clarified to mean a general physical exam, and not an exam for a specific injury, illness, or condition.

Summary statistics for key variables used in the analysis are presented in table 2.1. Across all years and states, the average person faced a cigarette tax of $0.70 per pack, ranging from a low of $0.02 to a high of $4.35. Approximately 22% of people reported being current cigarette smokers, while 25% reported being former smokers.

Between the years 1984 and 2010, state-level excise taxes place on cigarettes changed 238 times. Four states changed their rate only once (Georgia, Missouri, South Carolina, and West Virginia), while Rhode Island led the pack with 12 changes. The vast majority of these changes were increases, however 6 times the state reduced their excise tax on cigarettes. The average standard deviation within states over the 26 year period is 0.4, ranging from 0.02 in Missouri to 1.02 in Rhode Island.

Table 2.1

Summary Statistics		
Variable	Mean	S.D.
BMI	26.73	(5.61)
Obese*	0.22	(0.41)
Cigarette Tax	0.77	(0.69)
Smoking Bans		
Private Workplace	0.23	(0.42)
Restaurant	0.22	(0.41)
Bar	0.14	(0.35)
Current Smoker	0.20	(0.40)
Former Smoker	0.27	(0.45)
Avg. Fruit/Veg. per Day	4.34	(3.12)
Exercise in past month	0.67	(0.47)
Checkup in last year	0.72	(0.45)

*Obese = BMI ≥ 30

2.5 Results

Controlling for covariates such as age, race, education, income, marital status, and gender, smokers have significantly lower BMIs than their non-smoking counterparts. Table 2.2 shows that on average, a smoker's BMI is one point lower than a non-smoker, which translates to about 6.4 pounds for a person of average height (67 inches). This estimation, however, cannot be treated as causal. Smoking and body weight are both endogenous choices on the part of the consumer. Both are correlated with unmeasurable aspects of consumer decision-making, and therefore the estimator will produce biased results of an indeterminate magnitude and direction.

Table 2.2

Relationship Between Smoking and BMI	
Variable	BMI
Current Smoker	-1.000***
	(0.016)
Covariates	Y
State FE	Y
Year FE	Y
R^2	0.1176
N	4,203,934

***p-value < 0.01

Some of the previous literature has attempted to estimate the impact of smoking on body weight using cigarette prices. These too could suffer from bias, however. The changes in the price of cigarettes may represent other market trends. Even controlling for state and year fixed effects, table 2.3 shows that the price index constructed for food, medical services and activity using 1990-2010 ACCRA data are significantly related to cigarette prices. Consumer behavior cannot be reliably described with this method.

Table 2.3

Results of a Regression of Cigarette Prices on Price Indices, 1990 - 2003					
	Food Index	Fast Food Index	Fruit/Veg. Index	Medical Index	Activity Index
Effect on Cigarette Prices	9.516***	1.029	-5.535	0.119**	44.956***
	(3.572)	(1.825)	(9.862)	(0.057)	(12.858)
Covariates	N	N	N	N	N
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R^2			0.9376		
N			1,792,629		

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Alternatively, taxes on cigarettes offer an exogenous source of variation in the price of cigarettes. None of the price indices are related to the tax at a statistically significant level. These results suggest that the tax component of cigarette prices is not related to trends which impact both cigarette prices and the prices of other goods which may impact body weight.

Table 2.4

Results of a Regression of Cigarette Taxes on Price Indices					
	Food Index	Fast Food Index	Fruit/Veg. Index	Medical Index	Activity Index
Effect on Cigarette Taxes	0.932*	-0.009	0.563	0.006	-1.133
	(0.502)	(0.214)	(0.966)	(0.005)	(0.854)
Covariates	N	N	N	N	N
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R^2	0.6635				
N	1,792,629				

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Only includes years 1990-2003

Further, taxes on cigarettes are a rich source of variation in smoking behavior. Table 2.5 presents the results of the impact of cigarette taxes on both smoking status and on the quantity of cigarettes smoked. In the latter case, the estimation could only be done for 1984-2000 data. After this period, BRFSS stopped collecting information on the number of cigarettes smoked. This limitation of the data prevents any kind of full structural estimation, but does not prohibit a reduced-form approach.

Table 2.5

Relationship Between Cigarette Taxes and Smoking					
Variable	OLS Current Smoker	Logit Current Smoker	OLS Current Smoker	Logit Current Smoker	OLS Cigarettes Smoked
Cigarette Tax	-0.006***	-0.008***	-0.037***	-0.038***	-2.403***
	(0.002)	(0.002)	(0.007)	(0.007)	(0.478)
Covariates	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
Dep. Mean	0.20	0.20	0.24	0.24	19.94
R^2	0.0768	0.0778	0.0654	0.0643	0.0639
N	4,185,732	4,185,732	1,387,458	1,387,458	302,040
Years	1984-2010		1984-2000		

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Marginal Effects presented for Logit Estimations

The results in table 2.5 also suggest significant differences in the results based on the years included in the estimation. A one dollar increase in the cigarette tax between 1984 and 2000 reduced cigarette smoking by 3.7 percentage points. Adding in the years between 2000 and 2010 drops this effect to 0.6 percentage points. This could be explained by falling smoking prevalence. There were more smokers in the 1984 to 2000 time period, and therefore more people on the margin who are sensitive to price changes resulting from a tax increase. The smokers that remain in the 2000 to 2010 period should be less price sensitive, seeing how they are smoking in spite of the higher prices.

The relationship between cigarette taxes and body mass index is shown in table 2.6 below. A one dollar increase in the present-month's cigarette tax results in a 0.08 point drop in BMI. For a person of average height, this corresponds to a loss of roughly half-a-pound. While this estimate is small, it does appear to be precisely estimated. The table presents robust standard errors clustered on state and year. The estimate for the impact of the cigarette tax is significant beyond the 1% level. This significance level is maintained when using so-called jackknife standard errors, but not when clustering only on the state.

To test the robustness of these estimates, I also present the results of regression run without the covariates. Including only state and year fixed-effects, the impact of the tax is statistically indistinguishable from the results when the covariates are included. To further test whether or not this analysis is merely picking up on noise, I present the results of the effect of the cigarette tax on height. There is no theoretical or practical relationship between the tax and height, and therefore we would expect to find no effect at all here. Indeed, that is what I do find. Cigarette taxes do not impact height, which is consistent with the notion that these taxes are an exogenous influence on smoking behavior.

Table 2.6

Reduced-Form Results of Cigarette Taxes on BMI and Height						
Variable			BMI			Height
Cigarette Tax	-0.069***	-0.072***	-0.075***	-0.074***	-0.078***	0.005
	(0.020)	(0.020)	(0.023)	(0.023)	(0.020)	(0.009)
Unemployment Rate		-0.015**	-0.019***	-0.021***	-0.021***	0.005
		(0.007)	(0.007)	(0.008)	(0.007)	(0.003)
Income			-0.010***	-0.005***	-0.010***	0.006***
			(0.000)	(0.000)	(0.000)	(0.000)
Some HS				-0.698***	-0.305***	0.455***
				(0.032)	(0.029)	(0.022)
HS Grad				-0.713***	-0.223***	0.656***
				(0.036)	(0.029)	(0.031)
Some College				-0.911***	-0.271***	0.926***
				(0.037)	(0.032)	(0.033)
College Grad.				-1.606***	-1.054***	1.061***
				(0.038)	(0.030)	(0.031)
25-30					1.364***	-0.139***
					(0.029)	(0.016)
30-35					1.976***	-0.267***
					(0.032)	(0.015)
35-40					2.383***	-0.343***
					(0.029)	(0.018)
40-45					2.708***	-0.423***
					(0.028)	(0.016)
45-50					3.012***	-0.489***
					(0.028)	(0.017)
50-55					3.202***	-0.591***
					(0.029)	(0.017)
55-60					3.276***	-0.656***
					(0.031)	(0.019)
60-65					3.004***	-0.759***
					(0.030)	(0.018)
65+					1.848***	-1.172***
					(0.026)	(0.017)
Male					1.176***	5.687***
					(0.016)	(0.011)

Divorced					-0.372***	0.099***
					(0.016)	(0.010)
Widowed					-0.104***	-0.064***
					(0.023)	(0.013)
Never Married					-0.178***	0.053***
					(0.017)	(0.010)
Black					1.643***	-0.060***
					(0.022)	(0.012)
Asian					-1.895***	-2.571***
					(0.049)	(0.026)
Hispanic					0.722***	-1.798***
					(0.027)	(0.032)
American Indian					0.909***	-0.281***
					(0.065)	(0.038)
Other					0.279***	-0.434***
					(0.041)	(0.025)
Beer Tax					-0.065***	-
					(0.015)	-
State FE	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y
R^2	0.0383	0.0383	0.0462	0.0530	0.1119	0.5388
N	4,716,854	4,716,854	4,198,256	4,198,227	4,198,211	4,198,211

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The effect of the tax on the prevalence of overweight and obesity is shown in table 2.7. A one dollar tax on cigarettes leads to a 0.5 percentage point decline in obesity, and a 0.4 percentage point increase in those considered to be of normal weight (BMI between 18.5 and 25). Perhaps the most encouraging element of this analysis is that it does not leave anything unaccounted for. The changes in these categories add up to zero, suggesting that few if any are being pushed below a normal weight. The results are also consistent with weight loss across the BMI spectrum, with the numbers of people classified as overweight remaining stagnant as the formerly obese move into the category, and the newly normal move out.

Table 2.7

Reduced-Form Results of the Cigarette Tax on Normal Weight, Overweight, Obese						
	OLS			*Logit*		
	Normal	Overweight	Obese	Normal	Overweight	Obese
Cigarette Tax	0.004**	0.001	-0.005***	-0.004**	-0.001	-0.004**
	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.002)
Covariates	Y	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y
R^2	0.0562	0.0497	0.1115	0.0623	0.0394	0.0851
N	4,198,211	4,198,211	4,198,211	4,198,211	4,198,211	4,198,211

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Marginal Effects presented for Logit Estimations

One important question is whether or not the weight change persists over time. Likewise, changes in weight may take time to completely manifest themselves after a tax change. To address these concerns, I included the tax rates lagged by 1, 6, and 12 months. Table 2.8 presents the results of these regressions. In each case the contemporary tax drives the results, implying that higher taxes in the past do not predict changes in body weight. Only current taxes appear to matter, signifying that the change is both quick to manifest itself and persistent so long as the tax remains in effect. In the same vein, future tax changes are not predictive of current trends.

Table 2.8

Reduced-Form Results for Lagged Cigarette Taxes on BMI				
Variable	Current Tax	1 Month Lag	6 Month Lag	12 Month Lag
Effect on BMI	-0.076***	0.036	-0.019	-0.017
	(0.020)	(0.041)	(0.020)	(0.017)
Current Tax	-	-0.113***	-0.063***	-0.067***
	-	(0.039)	(0.022)	(0.021)
Covariates	Y	Y	Y	Y
State FE	Y	Y	Y	Y
Year FE	Y	Y	Y	Y
R^2	0.1119	0.1119	0.1119	0.1119
N	4,198,211	4,198,211	4,198,211	4,198,211

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The last of my efforts to establish the credibility of the research methods used is to look at the effect of other policies which impact smoking. Table 2.9 presents the effect of statutory smoking bans in restaurants, bars, and private workplaces on BMI. Additionally, the table shows the effect of have any one, two, or three of these bans. While none of these bans show a statistically significant effect on BMI, they are consistent in their direction. While there exist various incarnations of a variable that captures smoking bans that would yield statistically significant results, the methods used here provide stronger evidence of one fact. Policies which reduce smoking also appear to be reducing weight. The next step of the analysis is to determine why.

Table 2.9

Reduced-Form Results for Smoking Bans on BMI						
	Private Workplace	Restaurants	Bars	Any 1	Any 2	Any 3
Effect on BMI	-0.040 (0.032)	-0.053 (0.032)	-0.051 (0.035)	-0.055 (0.033)	-0.037 (0.045)	-0.052 (0.052)
Covariates	Y	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y
R^2	0.0931	0.0931	0.0931	0.0931	0.0931	0.0931
N	3,882,081	3,882,081	3,882,081	3,882,081	3,882,081	3,882,081

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

To get at the causal mechanisms of this weight loss, I first break down where the weight loss is occurring. Table 2.10 presents the results of estimations done separately for normal weight, overweight, and obese individuals. Those classified as being of a normal weight (BMI between 18 and 25) and those classified as being overweight (BMI between 25 and 30) show no signs of weight loss. However, those classified as being obese (BMI over 30) show a statistically significant amount of weight loss. This suggests that reduced smoking has a negligible effect on weight for most people, but leads to weight loss only among the obese.

Table 2.10

Results for Cigarette Taxes on BMI, Separated by Weight Class			
Variable	Normal Weight	Overweight	Obese
Effect of Cigarette Tax on BMI	-0.011 (0.007)	0.001 (0.006)	-0.049** (0.024)
Covariates	Y	Y	Y
State FE	Y	Y	Y
Year FE	Y	Y	Y
R^2	0.0926	0.016	0.0474
N	1,766,298	1,495,407	936,506

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Separating the estimations in this way does not produced consistent estimates, however, as it does not allow for people to change which weight classification they fall under, and previous results show that a significant number move from obese to overweight due to the tax. To obtain consistent estimates, I also estimated the same reduced-form model using quantile regression. Quantile regression estimates the effect at the median effect and other quantiles, rather than mean effect estimated by ordinary least squares (Buchinsky, 1998). To explore the differences in weight loss across the BMI, spectrum, I estimated the 10^{th}, 25^{th}, 50^{th}, 75^{th}, and 90^{th} quantiles. These results are presented in table 2.11.

Table 2.11

Results of a Quantile Regression for BMI on Cigarette Taxes					
	10th	25th	50th	75th	90th
Effect on BMI	-0.052** (0.022)	-0.054** (0.023)	-0.074*** (0.024)	-0.108*** (0.033)	-0.110*** (0.036)
Covariates	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
N	4,198,210	4,198,210	4,198,210	4,198,210	4,198,210

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The results from table 2.11 confirm the finding that the weight loss is much larger among those higher in the distribution of BMI. For a person in the 90th percentile of the conditional distribution (what is left over after the covariates are accounted for), the tax on cigarettes is associated with a 0.11 point decline in BMI. This is about double the impact estimated at the 10th percentile of the conditional distribution. While there isn't much reason to think the effect is nonlinear in the tax, it does appear to be nonlinear in BMI. Economically speaking, this is to be expected. Those at the higher end of the conditional distribution presumably have a greater benefit to reducing their smoking, and would be the most responsive to changes in the costs.

The differences between obese and non-obese smokers is made evident in table 2.12, which shows the results of a regression of BMI on cigarettes smoked. This estimation only contains data from 1984 to 2000, when the BRFSS contained a variable estimating the number of cigarettes smoked per day based off of self-report use. The results show that among only smokers, smoking more is associated with a higher BMI. The results indicate an addition 0.013 points of BMI per cigarette smoked.

Table 2.12

Relationship between Cigarettes Smoked and BMI among Smokers

Variable	BMI
Cigarettes per Day	0.013***
	(0.002)
Covariates	Y
State FE	Y
Year FE	Y
R^2	0.0828
N	302,040

***p-value < 0.01

A smoker in the 25th percentile smokes about 10 cigarettes per day. In the 75th percentile, they smoke about 20 cigarettes per day. In the 90th percentile, they smoke 40 cigarettes a day. The results from table

2.12 suggest that moving from 10 cigarettes to 20 cigarettes per day will add nearly one pound to a person of average height. Moving to 40 cigarettes per day will add another 1.67 pounds. While smoker in general have lower weights than non-smokers, it appears that heavy smokers weigh more than light smokers. This conclusion pans out in the raw data as well. The average BMI for someone who smokes between 1 and 20 cigarettes a day is 24.6. For someone who smokes more than 20 cigarettes a day, the average BMI is 26.8. That's the difference between being classified as "normal weight" and "overweight".

To determine which behavioral changes are being made that could potentially result in weight loss, I use self-reported measures of fruit and vegetable consumption, an indicator for whether or not a person has been to the doctor in the last year, and an indicator for whether or not they have engaged in exercise in the past month and run a regression to see if these measures are impacted by the tax rate on cigarettes or by a strong smoking ban. This strong ban is equal to 1 when a state currently bans smoking in private workplaces, restaurants, and bars, and is equal to 0 if it has only two or fewer of those bans. The results are presented in table 2.13.

Table 2.13

Relationship Between Cigarette Taxes and Smoking Bans w/ Other Health Behaviors

Estimation	OLS	OLS	Logit	OLS	Logit
Variable	Fruit/Veg. per Day	Doctor's Visit	Doctor's Visit	Exercise	Exercise
Cigarette Tax	0.455**	0.001	0.001	-0.012	-0.013
	(0.218)	(0.003)	(0.003)	(0.009)	(0.009)
Strong Smoking Ban	0.114	-0.009	-0.009	0.023	0.021
	(0.406)	(0.007)	(0.007)	(0.016)	(0.016)
Covariates	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R^2	0.3857	0.0802	0.0684	0.2871	0.2330
N	2,278,672	3,081,790	3,081,790	3,878,527	3,878,527

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01
Marginal Effects presented for Logit estimations.

Cigarette taxes only appear to have a statistically significant impact on fruit and vegetable (FV) consumption. A one dollar increase in the cigarette tax is associated with an increase in consumption by nearly half of a fruit or vegetable per day. With average consumption at about 4.3 FV per day, this sizable effect could explain a great deal of the observed effect of cigarette taxes on BMI. A simple regression of BMI on FV, including covariates and state and year fixed effects, shows that increased FV consumption is associated with lower BMIs. Each FV corresponds to a 0.03 point reduction in BMI. It is also clear, by similar methods, that smokers consume many fewer FV per day. About 0.22 less per day conditional on covariates.

A strong smoking ban, where the state outlaws smoking in private workplaces, restaurants, and bars, does not have a strong effect on any of the measure of behavior. The effect on doctor's visits is significant at the 10% level, but the reasons for such an effect could be operating through both smokers and non-smokers whose health improves due to reduced indoor smoking. While this result is interesting in its own right, it may not shed much light on the mechanisms for weight change.

Interesting is the lack of an effect observed on the measure of exercise. Theory suggests that taxes and smoking bans will have an unambiguously positive effect on exercise, but here both positive and negative effects are observed, though none are distinguishable from zero. Interpreted in light of the theoretical framework, the results of table 2.13 suggest that the primary effect of taxes on BMI operate through changes in diet. High taxes alter the composition of a person's diet in a way that leads to a reduction in food energy intake that dominates any change in the metabolism.

The weak effects of the smoking bans are not surprising considering the results presented in table 2.9. The choice to include only a strong ban was based on the variation in this indicators variation. The effects of other indicators, including all of those presented in table 2.9, where also estimated, but none yielded significant results except for a variable which was equal to the number of bans present, which also had a

negative effect on doctor's visits significant at the 10% level. However, the coefficient on a categorical variable like this is uninterpretable.

Lastly, it is important to take note of potential limitations of this approach. Table 2.14 presents the estimates for the effect of the tax on BMI separated by smoking status. The effect of the tax on cigarettes is significant for current smokers, former smokers, and never smokers. If the cigarette taxes are impacting the weight of non-smokers, this would seem to violate the exclusion restriction, and undermine the assumption that the cigarette tax is an exogenous influence on smoking. This could be remedied with a change in the theory, allowing for some kind of "second hand" effect of the reduced smoking. However, this could also be explained by the nature of the data. Some people may not take up smoking when they otherwise would have due to the tax. These people would fall into the "never smoker" category, but would be legitimately impacted by the tax in a way that could affect their weight. They are smokers who never took up the habit.

Table 2.14

Impact of Cigarette Tax on BMI by Smoking Status

	Dependent Variable: BMI			
Variable	Current Smokers	Former Smokers	Never Smokers	Ever Smokers
Effect of Cigarette Tax on BMI	-0.087***	-0.052**	-0.051**	-0.059***
	(0.028)	(0.024)	(0.026)	(0.020)
Covariates	Y	Y	Y	Y
State FE	Y	Y	Y	Y
Year FE	Y	Y	Y	Y
R^2	0.0908	0.1084	0.1298	0.098
N	879,440	1,154,172	2,152,120	2,033,612

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

One potential way to test this violation of the exclusion restriction is to overidentify the model using both the cigarette tax and the smoking bans to predict smoking behavior. In the reduced form models estimated above, the identifying assumption is that the tax on cigarettes is uncorrelated with all omitted factors which

impact BMI, conditional on the covariates included. An alternative approach is to estimate a structural version of the model in which the tax on cigarettes affects BMI only by operating through changes in smoking behavior. This approach makes an identical assumption about the independence of the cigarette tax, but gives the effect of a one unit change in smoking behavior rather than the effect of a one unit change in the tax. In neither case can the identifying assumption be tested. However, including a second source of variation in smoking behavior leaves some information that can be used to test this assumption.

Table 2.15 presents the first and second stages estimates of three structural models, all of which are overidentified by the inclusion of both the cigarette tax and smoking bans as instruments for smoking behavior. For these models, the variable capturing smoking bans ranges from 0 to 3, and indicates the strength of the ban in place. A '3' indicates that the state bans smoking in private workplaces, restaurants, and bars. A '2' indicates a ban in only two of these areas, and a '1' indicates a ban in only one of these areas. A '0' indicates that none of these three bans was in place at the time of the interview. What distinguishes these estimations and models are the measures of smoking behavior, and the time periods in the data used.

The first column measures smoking behavior by an indicator for whether or not a person currently smokes, and makes use of the full dataset. Current smoking is defined by having smoked more than 100 cigarettes in their lifetime and reporting smoking in the last month. The results show that both the cigarette tax and the smoking ban variable significantly impact smoking prevalence. A one dollar increase in the tax leads to a 0.4 percentage point drop in smoking prevalence, while an increase in the strength of the ban reduces prevalence by 0.2 percentage points. The relevance of these instruments in the first stage can be tested by an F-test against the null hypothesis that the instruments are irrelevant (Shea, 1997). With an F-statistic of 11.32, the null hypothesis can be rejected.

In the second stage, smoking is estimated to lead to a 10 point increase in BMI. Despite this rather dubious result, Wooldridge's overidentification test for use with robust standard errors gives a χ^2 of 0.001, which is not significant. Therefore I cannot rule out the validity of these instruments based on the test (Wooldridge, 1995). A 10 point increase in BMI due to smoking would suggest that the average smoker weighs about 64 pounds more than they would if they were to quit smoking. If this is true, then noting that the average smoker has a BMI of 25.6 means picking up cigarettes has saved millions from the dangers of being *under*weight. There is a simple explanation for this result, however. Cigarette taxes and smoking bans do more than just lead people to quit smoking. They also reduce smoking among smokers. An indicator variable for whether or not a person smokes at all is unquestionably correlated with a measure of how many cigarettes a person smokes, and taxes and bans on smoking have an impact on how many cigarettes a person smokes. The result is a downwardly biased estimate for the effect of the tax and bans on smoking status, which will inflate the second stage results.

The results in the second column of table 2.15 limit the data to the 1990-2000 samples, but estimate the same model used for the first column. These results are provided for comparison to the third column. This smaller sample lacks the variation needed to estimate the model, however. While cigarette taxes appear to be a more important predictor of smoking status, bans on smoking do not. This is almost certainly because the time period included saw only two bans go into effect. The F-statistic for the first stage instruments is 3.77. In the second stage, smoking leads to a 7.3 point increase in BMI. Wooldridge's overidentification test cannot conclude that either instrument violates the exclusion restriction, and gives a χ^2 of 0.018.

The third column presents the results for a model which uses an estimate of the average number of cigarettes consumed per day to capture smoking behavior. This variable is constructed based on responses to question on the average number of days the respondent smokes per month and the average number of cigarettes consumed on those days. Unfortunately, this data is limited to the 1990-2000 samples, and the

results of column 2 show a limited impact of smoking bans in this time period. To get the most consistent estimates possible, I limit the sample only to those who smoke.

The results shown in the third column suggest that a one dollar increase in the cigarette tax reduces the number of cigarettes smoked per day by about two. A stronger smoking ban reduces use by about 0.8 cigarettes per day. These appear to be highly relevant factors in the first stage, with an F-stat of 13.00. In the second stage, an extra cigarette smoked leads to an increase in BMI of 0.06 points, or about one-third of a pound for a person of average height. This estimate is not statistically significant, however. Nevertheless, I ran Wooldridge's overidentification test, which gives a χ^2 of 0.917, and cannot reject the hypothesis that the instruments are valid.

Table 2.15

Instrumental Variables Estimation of Effect of Smoking on BMI			
	First Stage		
Instrument	Current Smoker	Current Smoker	Cigarettes Smoked
Cigarette Tax	-0.004***	-0.017***	-1.961***
	(0.001)	(0.006)	(0.430)
Smoking Ban	-0.002***	-0.001	-0.784*
	(0.001)	(0.005)	(0.410)
F-stat	11.318	3.767	13.000
	Second Stage		
Instrumented Variable	BMI	BMI	BMI
Current Smoker	10.110***	7.263	-
	(3.627)	(5.321)	-
Cigarettes Smoked	-	-	0.058
	-	-	(0.082)
Covariates	Y	Y	Y
State FE	Y	Y	Y
Year FE	Y	Y	Y
Overid χ2	0.001	0.018	0.917
Years Included	1990-2010	1990-2000	1990-2000
N	3,870,559	1,072,285	220,807

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Data limitations make it impossible to extract a complete picture of the effect of smoking on weight. The limited number of smoking bans implemented in the 1990s, and the smaller sample sizes for the BRFSS in those years, prevents a robust estimation of a structural model. Nevertheless, the results from the third column of table 2.15 are not inconsistent with the notion that the tax on cigarettes and smoking bans work to change BMI exclusively by changing smoking behavior.

2.6 Discussion

Cigarette smoking and weight are strongly correlated with one another. By simple observation, one would be led to conclude that smoking helps to reduce weight, or to keep excess weight off. Smokers tend to have a BMI one point lower than their non-smoking counterparts, and conventional wisdom has long tied smoking to thinness. The results presented here, however, fall contrary to that claim. When smoking is varied exogenously, it leads to weight loss. Across the entire population, a one dollar rise in the cigarette tax leads to a 0.08 point decline in BMI. If these effects are attributed only to smokers, this would imply an average weight loss of about 4.5 pounds for a person of average height.

The results presented in table 2.14 suggest that these effects are not attributable to only smokers, however, which presents a significant challenge to the assertion that the cigarette tax is exogenous. In effect, these results seem to violate the "exclusion restriction", as they must be influencing something other than smoking. Another explanation is in the data being used. Because I have used repeated cross-sections, instead of following the same people over time, the tax on tobacco could be sorting people into different groups. In other words, the category of "never smokers" includes those who would have otherwise smoked if the tax on cigarettes hadn't been so high. This explanation seems more likely in light of the robustness of the estimates presented in table 2.6, and the lack of an effect on irrelevant variables like height. Nevertheless, the taxes on cigarettes may also be affecting non-smokers through their

relationships with smokers, and the results presented here should be interpreted as the full effect of the tax policy.

These results also depart from those found in the medical literature, where numerous clinical studies show that those who quit smoking tend to gain weight. The methods used here do not allow for conclusions about the effect of quitting smoking, however. The results of table 2.15 show severe problems with the methods used here when they are interpreted only though those who quit smoking. Instead, the results in this paper are only applicable to those who reduce their smoking as a result of the tax. Cigarettes may still be suppressing the appetite of this group, while the reduction leads to changes in diet that lead to additional weight loss. Resolving this disparity should be a primary focus of future research.

Missing from much of the previous literature is an exploration of how smokers differ from each other. The results here indicate that heavy smokers are much more likely to be overweight or obese than light smokers, and are much more responsive in terms of their weight change when it comes to changes in the tax. Thus, it is those who may be most at risk of a serious health condition that are most impacted by the policy. Where previous researchers have declared obesity to be an unfortunate side-effect of the anti-smoking campaign, the results here offer a silver lining.

The mechanisms for this weight change appear to be primarily dietary. Increases in the tax on cigarettes leads to an increase in fruit and vegetable consumption. Fruit and vegetable consumption is associated with lower body weights and a lower risk of obesity, and these results may be indicative of a shift towards a healthier diet on the part of smokers. No effect is observed on exercise behavior or on doctor's visits.

Fruit and vegetable consumption is associated with lower body weight and a lower risk of obesity. Further, smokers consume fewer fruits and vegetables than their non-smoking counterparts. The results of table 2.13 suggest that this is the sort of consumption that is substituted with smoking. There are a couple of explanations for this behavior. One is that smoking reduces the appetite for such foods or suppresses their

taste. The other is that reduced smoking raises the marginal benefit of a healthier diet by reducing the risk of health complications due to smoking that would likely precede health complications due to a poor diet. The lack of an effect on exercise behavior is evidence in favor of the former explanation. Increased exercise would serve many of the same goals as improving diet.

One major limitation on the interpretation of the results discussed so far is the effect of the tax seen on non-smokers. While the effect of cigarette taxes is largest for current smokers, a significant effect is observed both for former smokers and those who have never smoked. This would seem to violate the assumption that the cigarette tax is independent from other factors which impact BMI, as non-smokers are unaffected by the tax.

There are some explanations for these results that are consistent with this assumption, however. First is that the cross-sectional data does not follow the same people over time, and the effect observed for never smokers may be driven by those who would have taken up smoking if not for the tax. This population of people may be significant. Table 2.5 shows that smoking prevalence is reduced by 0.8 – 3.8 percentage points due to a one dollar increase in the tax. Some of this change is likely due to non-smokers choosing to never take up the habit.

The second explanation is that smokers have an effect on non-smokers. Non-smoking spouses of smokers, for example, may find it less costly to improve their own diet when their spouse is doing the same. If smokers are improving their health due to the tax, this may be inspiring their non-smoking relations to do the same. If there are spill-over effects such as these, they would be an important factor to consider when evaluating these taxes. Smokers may be imposing an externality on non-smokers beyond the effects of second-hand and environmental smoke.

Another limitation is in the magnitude of the effect. Table 2.15 gave the results of an instrumental variables estimation, where the tax on cigarettes and smoking bans instrumented for smoking behavior. Using an

indicator variable for whether or not a person currently smokes to measure smoking behavior suggests that smoking cigarettes leads to a 10 point increase in BMI. The "compliers" in this estimation are those who quit smoking due to an increase in the tax or the introduction of a smoking ban, and the results of the first stage suggest that this group is quite small. However, those most sensitive to changes in the tax rate may also be those most likely to invest in improving their health after quitting. Quitting smoking altogether may free up a large portion of income and increase demand for health overall.

Another explanation for the results in table 2.15 is that the tax is not a viable instrument for whether or not a person smokes in this case. Because the tax impacts the quantity of cigarettes a person smokes, and this quantity may impact BMI, the tax would violate the exclusion restriction of this model, as whether or not a person smokes systematically mis-measures the quantity of their smoking. The appropriate model would use cigarette taxes and bans to instrument for the quantity of cigarettes smoked for smokers alone (using both smokers and non-smokers would downwardly bias the first stage estimates). This is done in the third column of table 2.15, but the results found in the second stage are not statistically significant. Nevertheless, they suggest the average smoker in this local group gains about 7.5 pounds by smoking a pack of cigarettes a day.

Nonlinearities may also be a potential problem for estimation, but the inclusion of quadratic terms for cigarette taxes does not increase the variance in BMI explained, and the quadratic term does not have a statistically significant coefficient. Further, an analysis of the residuals (e.g. a partial residual plot) gives no reason to suspect nonlinearities. This notably contrasts with the conclusions of Chou, Grossman, and Saffer (2004).

Taken together, the results of this paper suggest that the convention wisdom about cigarette smoking and body weight is wrong. Reduced smoking due to increased taxes likely leads to weight loss for most of the

smoking population, but especially for smokers who are obese. This conclusion provides an important insight for policymakers to consider when deciding on changes to the tax on cigarettes.

3 THE IMPACT OF ALCOHOL TAXES ON BODY WEIGHT AND OBESITY

3.1 INTRODUCTION

Alcoholic beverages are a major source of caloric energy for Americans, providing 4.4% of total caloric intake (Block, 2004). Though they constitute a significant portion of the American diet, alcoholic beverages do not have significant nutritional value, and are generally consumed for recreation. Despite this fact, alcoholic beverages have not been a major focus of study or scrutiny when it comes to public policies aimed at reducing obesity.

Top 10 Calorie Sources for the US Population

Food Group	% of Total Energy
Sweets, desserts	12.3
Beef, pork	10.1
Bread, rolls, crackers	8.7
Mixed dishes	8.2
Dairy	7.3
Soft drinks	7.1
Vegetables	6.5
Chicken, fish	5.7
Alcoholic beverages	*4.4*
Fruit, juice	3.9

Source: NHANES 1999-2000 (Block, 2004)

Evidence is mixed as to whether or not alcohol consumption is a contributing factor to obesity. Epidemiological studies suggest that increased alcohol consumption leads to increased body weight, especially if it occurs before meal consumption, except in heavy drinkers (Jéquier, 1999). However, several recent studies show an inverse relationship between alcohol consumption and obesity (Wang et. al., 2010; Yeomans, 2010). These latter studies have made use of population data and self-reported weights and alcohol consumption and find the correlation between alcohol intake and weight gain.

To my knowledge, only one study in the economics literature has looked at this relationship. Chou, Grossman, and Saffer (2004) find that a 10% increase in the price of alcohol leads to a 0.27 percentage point decline in obesity. This relationship is interpreted as causal, but may suffer from problems due to the correlation between alcohol prices and the prices of goods which also impact body weight and obesity. Understanding this relationship is important to future policies which may aim to reduce the prevalence of obesity.

The primary goal of this paper is to estimate the impact of alcohol taxes on body weight and obesity. To answer this question, I obtained state-level excise taxes placed on beer from the Brewers Almanac, and merges them with data from the Behavioral Risk Factor Surveillance System (BRFSS). Alcohol taxes in general have remained largely stagnant, however the variation across the 26 years and 50 states of BRFSS data is significant enough to allow for this estimation. Additionally, I will examine the question of whether or not alcohol consumption is a risk factor for obesity by using these taxes as an instrument for average daily consumption of alcohol.

The results of this analysis confirm many of the recent findings. Alcohol consumption has an inverse relationship with weight. This relationship is deceiving however, as the causal estimations show that higher taxes placed on beer lead to lower weights, especially for those in the 90^{th} percentile of the BMI distribution. Further, an instrumental variables approach reveals that consuming one alcoholic drink per day leads to a 1.7 point increase in BMI, or about 10 pounds for a person of average height.

These results suggest that the calories consumed in alcoholic beverages are largely additive, and there is little offsetting from other calorie sources when consumption is increased. This weight gain may not be permanent, however. Changes in beer taxes only appear to affect body weight for a short period of time, with the effect nearly disappearing after 12 months. Together, these findings suggest that alcohol is an important risk factor for obesity, but that tax policies may not be an effective tool for reducing that risk in the long-run.

3.2 BACKGROUND

Numerous studies have catalogued the many health consequences associated with alcohol consumption. Alcohol consumption leads to nearly 100,000 deaths each year in the United States by contributing to many chronic conditions such as liver disease, stroke, liver cirrhosis, and several cancers as well as acute causes such as motor-vehicle accidents, homicides, and suicides (CDC, 2012). Alcohol use has been shown to have

a J-shaped relationship with ischemic heart disease, with moderate alcohol consumption leading to approximately 30,000 fewer deaths each year in the United States. All told, alcohol consumption is one of the leading causes of death in the U.S. and the economic costs associated with heavy drinking have been estimated at $223.5 billion annually (Bouchery, Harwood, Sacks, Simon, & Brewer, 2011).

Consistent with economic theory and the law of demand, previous studies have shown that taxes placed on alcohol increases the price of alcohol, which decreases overall consumption. Across 38 studies which reported the price elasticity of alcohol, the median elasticities were -0.50 for beer, -0.64 for wine, and -0.79 for spirits (Elder, et al., 2010). Reductions in drinking also has been shown to translate into a reduction in the consequences of alcohol use, including decreases in alcohol-related motor-vehicle accidents, fewer deaths due to liver cirrhosis, and reduced violence. In this section, I will review those studies, and comment on gaps this left in the literature that this research intends to fill. Given the size of this literature, I will restrict myself only to those which examine the effects of taxes on alcohol.

Many studies have examined the effect of beer taxes on motor vehicle accidents and fatalities. Chaloupka, Saffer, & Grossman (1993) find that beer taxes significantly reduced motor-vehicle fatalities at all ages, and particularly among youths aged 18-20. According to their findings, a 10% increase in the tax on beer led to a 1% reduction in alcohol-related fatalities at all ages and a 1.6% reduction in youth fatalities. More recent studies, however, have concluded that beer taxes have an insignificant effect on alcohol-related traffic fatalities (Dee, 1999; Ruhm, 1995; Mast, Benson, & Rasmussen, 1999). These papers advocate for more elaborate means of controlling for unobserved factors which may influence drinking behavior and support for alcohol taxes.

Another of the most studied health outcomes of excessive alcohol consumption is how it relates to instances of liver cirrhosis. Most of these studies, however, predate this author's birth and most focus on the price of alcohol rather than a tax. One study, however, found that a one dollar increase in the tax on

spirits led to a 5.4% reduction in age-adjusted cirrhosis mortality rates for adults over 30 years old (Cook & Tauchen, 1982). While this is less true for alcohol-related motor vehicle accidents and fatalities, the literature on the influence of taxes on alcohol-related deaths due to liver cirrhosis and other diseases is thin. Given that taxes have been a primary policy tool for millennia, and are among the legislators most direct method of influencing prices, this is somewhat surprising.

Missing from the economic literature is research addressing the impact of alcohol consumption on obesity. Alcohol consumption directly contributes to a person's weight by increasing energy intake. Further, alcohol consumption has been shown to compound the health problems associated with obesity, such as coronary heart disease and even breast cancer (Colditz, et al., 1991). Given that between 51 and 67% of American report consuming alcohol, this relationship merits greater study.

Epidemiologic evidence is mixed with regards to the relationship between alcohol intake and body weight. Most studies suggest that increased alcohol consumption leads to increased body weight, however, except in heavy drinkers (Jéquier, 1999). Short-term clinical studies show that drinking before a meal does not lead to a person reducing their calorie intake during the meal, but longer-term studies show that people consuming 1-2 drinks per day did substitute alcohol for food, keeping their average energy intake constant (Breslow & Smothers, 2005).

Chou, Grossman, and Saffer (2004) do control for the price of alcohol and alcohol squared in their investigation of the causes of the rise in obesity over the past few decades, but this may suffer from the same endogeneity problems as the price of cigarettes demonstrated by Nonnemaker et. al. (2009). Their findings suggest that at the mean, a 10% increase in the price of alcohol leads to a 0.27 percentage point decline in obesity. To my knowledge, the Chou, Grossman, and Saffer paper is the only one attempting to estimate a causal impact of alcohol consumption on overall obesity rates.

3.3 METHODS

Section 1.2 laid out a theoretical framework for estimating the effect of alcohol policy on body weight. The excise tax on beer, conditional on state and year fixed effects, is an exogenous source of variation in alcohol consumption on which I can measure the impact. To find the effect of these taxes on BMI and obesity, I estimate the following reduced-form equation:

$$BMI_{its} = \beta_0 + \beta_1 BeerTax_{ts} + \beta_2 X_{its} + \beta_3 UR_{ts} + Year_t + State_s + \varepsilon_{its}$$

where *i* denotes the individual, *t* denotes the time, and *s* denotes the state. *BMI* is the individuals body mass index, *BeerTax* is the excise tax placed on a gallon of beer, *X* is a set of individual-specific covariates, *UR* is the state-level unemployment rate in time *t*, and *Year* and *State* are year and state fixed effects respectively. The individual covariates contained in *X* include age group, gender, race, education level, marital status, and income.

Controlling for state and year trends, broader state-level economic conditions, and individual-specific covariates, taxes on beer serve as an exogenous indicator of changes in alcohol consumption. Therefore, in this model it is differences in these tax rates within states, compared to other states without a change in their rates that will estimate the causal impact of these taxes on body weight.

A key question in the previous literature has been the extent to which increased alcohol consumption contributes to weight gain and obesity. Alcohol is a direct source of calories, but increased consumption could be offset by reductions in calorie consumption elsewhere. To estimate how much alcohol consumption contributes to weight gain and obesity, I use an instrumental variables approach with the beer tax acting as the instrument for average alcohol consumption per day.

To determine the validity of this instrument, I introduce another policy change with significant variation in the time period of the sample: Blood Alcohol Concentration (BAC) limits for operating a motor vehicle. These limits increase the full price of alcohol consumption by imposing legal penalties on those who

consume alcohol and then operate a motor vehicle. The inclusion of this second instrument provides information on which the identifying assumptions can be tested.

To further get a sense of how alcohol consumption impacts BMI and obesity, I also estimate the equation above using quantile regression. Quantile regression estimates the effect at the median effect and other quantiles, rather than mean effect estimated by ordinary least squares (Buchinsky, 1998). By estimating the effect at the 10^{th}, 25^{th}, 50^{th}, 75^{th}, and 90^{th} percentiles, I can test whether or not the effect is the same for people of different weight classifications.

3.4 DATA

The primary source of data for this analysis is the BRFSS, which is described in more detail in section 1.3. The BRFSS data provides individual-specific data on weight, height, age, gender, education, income, marital status, and race. Using these data, I am able to construct BMI and indicators for being normal weight, overweight, or obese. Additionally, using self-reported measures of drinking behavior, I construct an estimate of the average number of alcohol drinks consumed per day for each respondent. Respondents are told that a "single drink" refers to 12 oz. of beer, 5 oz. of wine, or 1.5 oz. of 80-proof distilled spirits or liquor.

To assess the validity of the research design, I also use data from the American Chamber of Commerce Researchers Association (ACCRA) on prices across more than 300 U.S. cities for a variety of goods and services. These data, and the methods used to construct the price indices used here, is described in detail in section 1.3.

Data on state-level excise taxes on beer comes from *The Brewers Almanac* (2012), which details all tax changes since the ratification of 21^{st} Amendment, which ended prohibition in America. While these changes are sparse, averaging just 4.67 per state, there are nevertheless still 59 changes between 1984 and 2010.

30 states and the District of Columbia changed their tax at least once in the time period, while 20 states did not. New York had the most changes, with six. On average, states changes their tax rate 1.16 times between 1984 and 2010. Because the research methods include robust time trends, these rates are left in their nominal form.

Data on legal limits for blood alcohol concentration (BAC) in the bloodstream was gathered from the *Digest of State Alcohol Highway Safety-Related Legislation* (1983-1998) and the *Alcohol Policy Information System*. In 1984, the vast majority of states had laws which made operating a motor vehicle with a BAC of 0.10 or higher illegal. A number of states had higher limits (Colorado, Georgia, Iowa, and Virginia), while some others did not make operating a motor vehicle under the influence of alcohol a crime per se (Kansas, Kentucky, Maryland, Massachusetts, South Carolina, Tennessee, and West Virginia). The last state to adopt a "per se" BAC limit was Massachusetts in 2003. However, in all of these cases, state's without "per se" laws set legal limits which served as *Prima Facie* evidence in court.

In cases where states did not have "per se" laws, the legal limit which served as the legal definition of driving under the influence of alcohol was used. In all other cases, the "per se" limit was used. Overall, there were 53 changes in the BAC limit across the 50 states and 26 years of the sample. Only two states, Oregon and Utah, did not change their laws at any time in the sample. In both cases, the BAC limit remained constant at 0.08. In most cases, states had a BAC limit of 0.10 until around 1994 or 2003, two years in which many states lowered their limits to 0.08. However, the exact timing of these changes varies significantly between states, creating a natural experiment on which a causal effect can be estimated.

Table 3.1 gives summary statistics for key variables used in this analysis.

Table 3.1

Summary Statistics		
Variable	Mean	S.D.
BMI	26.73	(5.61)
Obese	0.22	(0.41)
Beer Tax	0.51	(2.09)
BAC Limit	0.09	(0.01)
Avg. Drinks per Day	0.43	(1.20)

3.5 RESULTS

Controlling for covariates such as age, race, education, income, marital status, and gender, those who drink more have significantly lower BMIs than their non-drinking counterparts. Table 3.2 shows that on average, each drink per day is associated with decrease of 0.11 points of BMI. For a person of average height, this means each drink corresponds to weighing 0.7 pounds less. This estimation, however, cannot be treated as causal. Drinking and body weight are both endogenous choices on the part of the consumer. Both are correlated with unmeasurable aspects of consumer decision-making, and therefore the estimator will produce biased results of an indeterminate magnitude and direction.

Table 3.2

Relationship Between Drinking and BMI	
Variable	BMI
Drinks per Day	-0.112***
	(0.007)
Covariates	Y
State FE	Y
Year FE	Y
R^2	0.1169
N	3,772,876

***p-value < 0.01

Previous work has examined the impact of alcohol consumption on body weight using the price of alcohol. However, as I show in table 3.3, a price index for the price of beer, wine, and spirits may be related to other

prices which also impact weight. The price index is constructed from the 1990-2004 ACCRA data, which contains quarterly prices for 750 ml bottle of scotch, a six-pack of 12oz beer, and a 1.5 liter bottle of wine. The results here show that alcohol prices are correlated with the prices of other goods, and may not be suitable as an exogenous influence on drinking behaviors when estimating the effect of drinking on body weight.

Table 2.3

Results of a Regression of Alcohol Prices on Price Indices					
	Food Index	Fast Food Index	Fruit/Veg. Index	Medical Index	Activity Index
Effect on Alcohol Prices	0.088**	0.036	0.128*	-0.001	0.012
	(0.042)	(0.028)	(0.074)	(0.001)	(0.065)
Covariates	N	N	N	N	N
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R^2	0.9427				
N	1,841,204				

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Only includes years 1990-2004

The effect of alcohol consumption on body weight may be better estimated using taxes placed on beer. Table 3.4 gives the results of the same regression run for table 3.3, but replacing the price index of alcohol with state-level excise taxes on beer. Unlike alcohol prices, the beer tax is not correlated with other price indices, such as the price of grocery food.

Table 3.4

Results of a Regression of Beer Taxes on Price Indices					
	Food Index	Fast Food Index	Fruit/Veg. Index	Medical Index	Activity Index
Effect on Beer Taxes	0.088	0.007	-0.137*	0.001	-0.137
	(0.060)	(0.022)	(0.073)	(0.001)	(0.182)
Covariates	N	N	N	N	N
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R^2	0.9998				
N	2,064,243				

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Only includes years 1990-2004

Table 3.5 shows that these beer taxes also have a significant effect on alcohol consumption in general. A one dollar increase in the tax on a gallon of beer (or about $0.10 per 12oz beer) results in 0.05 fewer drinks consumed per day. This finding alleviates any concerns that the state-level excise taxes on beer don't have enough variation to estimate an impact. It is also important to note that these taxes are in nominal form, and have not been adjusted from their statutory levels.

Table 3.5

Relationship between Beer Taxes and Alcohol Consumption	
Variable	Drinks per Day
Beer Tax	-0.047***
	(0.007)
Covariates	Y
State FE	Y
Year FE	Y
R^2	0.0584
N	3,772,876

***p-value < 0.01

The effect of beer taxes on body mass index is shown in table 3.6 below. A one dollar increase in the present-month's tax on a gallon of beer results is a 0.065 point drop in BMI. For a person of average height,

this corresponds to the loss of roughly half-a-pound. If the one dollar tax were collected per 12 oz. drink, these results suggest the average person would lose about 5 pounds. These results are somewhat sensitive to changes in the specification, however, hovering around an estimate of -0.04 until education is controlled for. The tax on beer does not predict differences in height, a component of BMI which should not be effected by alcohol consumption.

Table 3.6

Reduced-Form Results for Beer Taxes on BMI and Height						
Variable	BMI					Height
Beer Tax	-0.042**	-0.032*	-0.038**	-0.070***	-0.065***	-0.013
	(0.017)	(0.019)	(0.015)	(0.026)	(0.015)	(0.013)
Unemployment Rate		-0.013**	-0.017**	-0.018**	-0.021***	0.005
		(0.007)	(0.007)	(0.008)	(0.007)	(0.003)
Income			-0.010***	-0.005***	-0.010***	0.006***
			(0.000)	(0.000)	(0.000)	(0.000)
Some HS				-0.697***	-0.305***	0.455***
				(0.032)	(0.029)	(0.022)
HS Grad				-0.712***	-0.223***	0.656***
				(0.036)	(0.029)	(0.031)
Some College				-0.910***	-0.271***	0.926***
				(0.038)	(0.032)	(0.033)
College Grad.				-1.606***	-1.054***	1.061***
				(0.038)	(0.030)	(0.031)
25-30					1.364***	0.934***
					(0.029)	(0.022)
30-35					1.976***	-0.139***
					(0.032)	(0.016)
35-40					2.383***	-0.267***
					(0.029)	(0.015)
40-45					2.708***	-0.343***
					(0.028)	(0.018)
45-50					3.012***	-0.423***
					(0.028)	(0.016)
50-55					3.202***	-0.489***
					(0.029)	(0.017)
55-60					3.276***	-0.591***
					(0.031)	(0.017)
60-65					3.004***	-0.656***
					(0.030)	(0.019)

65+					1.848***	-0.759***
					(0.026)	(0.018)
Male					1.176***	-1.172***
					(0.016)	(0.017)
Divorced					-0.372***	5.687***
					(0.016)	(0.011)
Widowed					-0.104***	0.099***
					(0.023)	(0.010)
Never Married					-0.178***	-0.064***
					(0.017)	(0.013)
Black					1.643***	0.053***
					(0.022)	(0.010)
Asian					-1.895***	0.124
					(0.049)	(0.084)
Hispanic					0.722***	-0.060***
					(0.027)	(0.012)
American Indian					0.909***	-2.571***
					(0.065)	(0.026)
Other					0.279***	-1.798***
					(0.041)	(0.032)
Cigarette Tax					-0.078***	-
					(0.020)	-
State FE	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y
R^2	0.0383	0.0383	0.0461	0.0530	0.1119	0.5388
N	4,716,854	4,716,854	4,198,256	4,198,227	4,198,211	4,198,211

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The changes in weight do not appear to be permanent, however. Table 3.7 shows that the effect of the tax on beer diminishes with more distant lags when the previous period's tax rate was higher than the contemporary one. Further, having a higher tax just one month prior, holding the current tax constant, is associated with a weight increase, suggesting a quick response to tax changes.

Table 3.7

Reduced-Form Results for Lagged Beer Taxes on BMI				
Variable	Current Tax	1 Month Lag	6 Month Lag	12 Month Lag
Effect on BMI	-0.065***	0.070***	0.014	0.005
	(0.015)	(0.018)	(0.020)	(0.010)
Current Tax	-	-0.145***	-0.090**	-0.073***
	-	(0.025)	(0.043)	(0.022)
Covariates	Y	Y	Y	Y
State FE	Y	Y	Y	Y
Year FE	Y	Y	Y	Y
R^2	0.1119	0.1119	0.1119	0.1119
N	4,198,211	4,198,211	4,198,211	4,198,211

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

While alcohol is a direct source of calories, there is debate among epidemiologists and other public health researchers about effects of alcohol consumption on body weight and the risk of obesity. Some have argued that increases in alcohol consumption are generally compensated by reductions in the consumption of other energy sources. Other studies have found that increased alcohol consumption is associated with lower weights and a lower risk of obesity. Alternatively, increased alcohol consumption is not offset by reductions elsewhere, and leads to an energy imbalance that causes increased weight and an increased risk of obesity.

To test these competing hypotheses, I use beer taxes as an exogenous instrument for self-reported alcohol consumption, measured by the average number of drinks consumed per day. In the second stage, I estimate the coefficient for the number of drinks per day on BMI. This estimate can then be compared to calorie consumption needed to sustain such a weight change and the average calories contained in alcoholic beverages. If the estimate is associated with an increase in calories consumption which is smaller than the calories found in an average drink, then this would suggest some offsetting.

In addition to the tax on beer, I also include the BAC limit for legally operating a motor vehicle. A lower limit increases the full price of alcohol consumption by increasing the penalties associated with drunk driving.

Thus, a decrease in the limit should reduce the quantity of alcohol consumed. Because these policies are formed in a similar fashion to the tax on beer, they will serve as another exogenous influence on drinking behavior on which the effect on BMI can be estimated. Including these limits will over-identify the model and allow for a test of the exclusion restriction using the Wooldridge (1995) test.

The results of the instrumental variables estimation is presented in table 3.8. In the first stage, the state-level excise taxes placed on gallons of beer are associated with a reduction in alcohol consumption of 0.046 drinks per day per one dollar increase in the tax. Likewise, the BAC limit is associated with reduced consumption as the limit falls. A change in the BAC limit of 0.01 is associated with drinking 0.007 fewer alcoholic beverages per day. These estimates are both statistically significant, and they appears to be highly relevant with an F-statistic of 27.27. In the second stage, a drink per day is associated with a 1.7 point increase in BMI. For a person of average height, this corresponds to an increase in body weight of about 10 pounds. The Wooldridge overidentification test produces a χ^2 of 0.663, meaning I cannot reject the hypothesis that both instruments are valid.

Table 3.8

Instrumental Variables Estimation of Drinking on BMI		
	First Stage Drinks per Day	*Second Stage* BMI
Beer Tax	-0.046***	
	(0.007)	
BAC Limit	0.686**	
	(0.269)	
Drinks per Day		1.722**
		(0.733)
F-Statistic	27.27	-
Overid χ2	-	0.663
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.0584	-
N	3,772,876	3,772,876

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

A 10-pound increase in body weight is large, but in line with expectations if there is only partial replacement of other calories by the increase in alcohol consumption. An additional drink is defined by the BRFSS as 12 oz. of beer, 5 oz. of wine, or 1.5 oz. of 80-proof distilled spirits or liquor. The USDA reports that the average calorie count is about 153 for a 12 oz. can of regular beer, 123 for 5 oz. of wine, and 97-110 for 1.5 oz. of distilled spirits such as vodka or gin (USDA, 2013). While the ideal method for estimating the contribution of each calorie to body weight is disputed, using the Mifflin equation for estimating the resting metabolic rate, an extra 10 pounds requires an additional 100 calories per day (Mifflin, et al., 1990).

The Mifflin equation is estimated on population data, and models the resting metabolic rate as a function of weight, height, and age. The function estimated was found to be the most predictive of several methods (Frankenfield et. al., 2005). While there is considerable debate over its efficacy for individuals, the Mifflin equation explains 71% of the variance in resting energy expenditure for the entire population. Since I am dealing in population averages, these rough estimates should be consistent enough to draw broad conclusions.

Because men and women have been shown to differ in their resting metabolic rates, I separated the analysis by gender. The results shown in table 3.9 suggest that there is little difference in the amount of weight loss associated with the beer tax for men and women. This may be due to differences in the response to the tax or similarities in male and female metabolism when it comes to alcohol.

Table 3.9

Differential Impacts of Beer Tax on BMI by Sex		
Variable	Men's BMI	Women's BMI
Beer Tax	-0.067***	-0.086***
	(0.018)	(0.022)
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.094	0.1282
N	1,744,083	2,454,128

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

To determine whether or not the effect of the beer tax is different across the BMI spectrum, I estimated the same regression shown in table 3.6 using quantile regression. The results are presented in table 3.10. They indicate that the effects are concentrated among the obese. The 10th percentile of the conditional distribution, the 75th percentile, and the 90th percentile show a negative relationship between beer taxes and BMI which is significant at the 5% level. The magnitude is significantly higher at the 90th percentile, however. In this part of the distribution, a one dollar increase in the beer tax led to a 0.2 point drop in BMI. For a person of average height, this translates into about 1.25 pounds.

Table 3.10

Results of a Quantile Regression for BMI on Beer Taxes					
	10th	25th	50th	75th	90th
Effect of Beer Tax on BMI	-0.065**	-0.029	-0.063*	-0.110**	-0.198**
	(0.029)	(0.029)	(0.035)	(0.045)	(0.081)
Covariates	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
N	4,198,210	4,198,210	4,198,210	4,198,210	4,198,210

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Despite the clear results of the quantile regression, how the beer tax translates into changes in weight classification is not so clear. Table 3.11 shows the effect of beer taxes on an indicator variable equal to 1 if the person is considered obese (BMI > 30) and 0 otherwise, another indicating whether a person is considered overweight (BMI between 25 and 30), and a third indicating whether a person is of normal weight (BMI between 18.5 and 25). The results suggest some movement between classifications. In the case of obesity, an OLS estimation suggests a one dollar tax increase on beer lowers obesity by 0.5 percentage points. This estimate is significant at the 1% level. Using the more appropriate logit estimation, however, this statistical significance vanishes.

Table 3.11

Impact of Beer Tax on Normal Weight, Overweight, and Obese						
	OLS			Logit		
	Normal	Overweight	Obese	Normal	Overweight	Obese
Beer Tax	0.005***	-0.000	-0.005***	0.005***	-0.000	-0.003
	(0.002)	(0.002)	(0.001)	(0.002)	(0.002)	(0.002)
Covariates	Y	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y
R^2	0.1117	0.0503	0.0564	0.0853	0.0399	0.0624
N	4,198,210	4,198,210	4,198,210	4,198,210	4,198,210	4,198,210

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01
Marginal Effects presented for Logit Estimations

Table 3.11 shows a clear effect for those classified as being of normal weight though. A one dollar increase in the beer tax increases those of normal weight by 0.5 percentage points. This estimate is highly significant in both the OLS and logit estimations. There seems to be no effect on the prevalence of overweight. This is consistent with a general effect of weight loss across the BMI spectrum, however. The ranks lost due to those moving from overweight to normal weight are filled by those moving from obese to overweight.

3.6 Discussion

Previous research has tied alcohol consumption to a lower risk of overweight any obesity. Indeed, each daily drink is associated with a BMI 0.11 points lower. Considering the high calorie nature of most alcoholic beverages, however, this finding is dubious. Using the excise tax placed on beer as an exogenous source of variation in beer consumption, I find that a one dollar increase in the tax per gallon results in 0.05 fewer drinks consumed per day. If this tax were placed per 12 oz. drink, the results suggest this would lead to 0.5 fewer drinks consumed per day.

The effect of the tax on alcohol consumption carries directly on to BMI and obesity. A direct source of calories, alcohol consumption can produce the energy imbalance described in section 1.2, and lead to

overweight or obesity. My results suggest that a one dollar increase in the tax on beer will result in a 0.07 point reduction in BMI, or about half of a pound for a person of average height. This effect appears to be much larger for people of higher weights. When estimated at the 90^{th} percentile (a BMI of about 32.7) using quantile regression, the same one dollar tax leads to a 0.2 point reduction in BMI, or a loss of about 1.25 pounds.

One limitation in the interpretation of these results is that they do not appear to be related to a significant reduction in obesity. There does appear to be a significant increase in the number of people classified as being of a normal weight, however. Because weight loss across the BMI spectrum has an ambiguous effect on those classified as overweight, with previously obese people moving into the category as newly normal weight people are moving out, it is unclear what the effect of these tax policies would be on diagnoses. Generally, these findings suggest that alcohol is an important risk factor for obesity, but that tax policies may not be an effective tool for reducing that risk in the long-run.

The long-term effects of the tax appear to be diminishing. The strongest impact is seen with the concurrent tax rate, with the effect declining as the tax is lagged. A one dollar increase in the tax on beer 12 months ago only leads to a 0.02 point decline in BMI. This translates to about 0.11 pounds, a very small but still statistically significant change. These findings seem to limit the use of such taxes as a policy tool aimed at reducing overweight and obesity, though consumers do appear rather responsive to them.

Nonlinearities also do not appear to be an issue in alternative specifications of the model. Quadratic terms for the tax on beer are not statistically significant, and residual analysis leaves no reason to think nonlinearity is a major issue here.

Perhaps the larger contribution of this paper, then, comes from the instrumental variables design. Taxes on beer provide exogenous variation in alcohol consumption. From this variation, I can estimate the impact of a drink per day on BMI. The findings presented show that an extra drink per day leads to 1.7 point

increase in BMI. For a person of average height, this is equal to about 10 pounds. Because this is an estimate of the population effect, the use of Mifflin's population estimates for resting metabolic rates should be consistent, and suggests daily caloric intake increases by about 100 calories.

This 100 calorie increase represents a large share of the calories that would be consumed in an average alcoholic drink. Keeping consistent with the BRFSS definitions of a drink, the average beverage would have 97-153 calories in it. This suggests there is little offsetting when alcohol intake is increase. Calories from alcohol are largely additive.

One other finding in the previous literature is a significant difference between the effects on men and women. I find no evidence of such a difference. Future work should seek to reconcile these findings with the literature. More robust measures of taxes on alcohol may also provide for better estimates. The beer tax used here is only a small fraction of that total tax, which includes a much larger sales tax.

Recent policy proposals which target high calorie snacks and sugar sweetened beverage in an effort to combat obesity have been silent on similar proposals for alcohol. Among the ten largest sources of calories, alcohol may contribute directly to the obesity problem by leading to an imbalance in energy intake and output. It is evident from the research presented here that alcohol is playing an important role in this area, but it remains to be seen what the right policies might be.

4 THE HEALTH CONSEQUENCES OF CIGARETTE AND ALCOHOL TAXES

4.1 INTRODUCTION

Each year, cigarette smoking and alcohol consumption claim the lives of 520,000 people in the United States alone, accounting for more than 1 out of every 5 deaths (Mokdad, Marks, Stroup, & Gerberding, 2004). It is no surprise, then, that these topics have amassed a massive literature that investigates the causes and consequences of smoking and drinking. There remains an opening in the literature, however, for the effects of tax policies aimed at smoking and drinking.

This paper seeks to find population estimates for the impact of taxes on cigarettes and alcohol on various health outcomes. Because the literature on the effects of smoking and drinking on cancer and liver disease respectively is well covered, these health outcomes will focus on issues related to obesity, including hypertension, diabetes, cholesterol, and arthritis. Previous chapters have explored the relationship between smoking, drinking, and obesity, but obesity per se has not been tied to a risk of death. Rather, it is the consequences of health behaviors which cause obesity that also cause death. This paper will explore those other consequences.

The unexplored effects of alcohol and tobacco taxes on health conditions related to obesity merit study due to the trends in obesity over the past few decades. An increasing number of Americans are now classified as obese, or having a Body Mass Index (BMI) greater than 30. At the same time, policies aimed at curbing smoking and drinking remain popular with lawmakers seeking to improve public health. Understanding how these issues interact will provide important guidance to these policymakers as they weigh each proposal.

It is also important to investigate how the effects of smoking and drinking on obesity play out in other obesity-related health conditions. Hypertension is not directly related to smoking, but may be impacted

through smoking's effect on diet and exercise (Dept. of Health and Human Services, 2004). Low-to-moderate alcohol consumption has been shown to have a j-shaped relationship with hypertension, with 1-2 drinks per day reducing a person's risk, but more than 2 drinks increasing it (Sesso, 2010). Policies which aim to reduce drinking may have the unintended consequence of increasing rates of hypertension. A similar story can be told about diabetes and cholesterol.

Arthritis, the nation's most prominent disability, affects 52.5 million people in the U.S., but is only tied to smoking and drinking through a relatively rare form of arthritis known as rheumatoid arthritis. The most common form, osteoarthritis, can be caused by obesity, which puts extra strain on knee joints. The evidence provided in chapters 2 and 3 suggest that the effects of alcohol and tobacco on obesity are unlikely to lead to significant changes in rates of arthritis, however, since these effects are not larger than a few pounds. Thus, arthritis acts as something of a falsification test, where significant and sizable effects would be interpreted as evidence of misspecification in the model.

The methods used to estimate the impacts of these policies are simple. State-level taxes, conditional on state and year fixed effects, broader state-level economic conditions, and individual-specific covariates, provide for exogenous variation in cigarette and alcohol consumption on which a causal estimate can be found with simple regression. To ensure the estimates are not the product of statistical noise, a logit model is also employed. The chief reason for this design is to provide policy-relevant estimations.

To estimate the impact of taxes on health outcomes, I use data from the Behavioral Risk Factor Surveillance System (BRFSS), which is described in more detail in section 1.3. These data contain indicator variables for whether or not a respondent reports being told by a doctor that they have hypertension, diabetes, high cholesterol, or arthritis. State-level excise taxes on packs of cigarettes are collected from *The Tax Burden on Tobacco* and state-level excise taxes placed on gallons of beer are collected from *The Brewers Almanac*.

These taxes on beer will serve as a proxy for alcohol taxes in general to the degree that they are correlated with each other.

The results show reductions in the prevalence of hypertension and diabetes with increases in the cigarette tax. A one dollar increase in cigarette taxes leads to a 0.78 percentage point reduction in hypertension rates, and a 0.24 percentage point reduction in diabetes prevalence. In terms of the population, a nationwide one dollar increase would prevent 523,000 fewer cases of hypertension and 63,000 fewer cases of diabetes.

For taxes on beer, a one dollar increase increases rates of hypertension, while reducing the prevalence of diabetes. A one dollar increase in the beer tax leads to a 0.25 percentage point increase in hypertension rates. This translates to about 170,000 more people with chronic high blood pressure. For diabetes, a one dollar increase in the beer tax results in a 0.15 percentage point decline in diabetes prevalence, or about 39,000 fewer cases. These results suggest that the average person is on the left side of the j-shaped relationship between alcohol and hypertension, but that few benefit from any protective effect of alcohol with relation to diabetes.

Neither the taxes on cigarettes or beer have a significant effect on instances of high cholesterol or arthritis. The latter case confirms priors, but the former does not. Both smoking and drinking are tied to blood cholesterol levels, which should be compounded with any effects cigarettes and alcohol taxes have on diet and exercise. The insignificant effects found here should be a focus of future research.

4.2 Background

Over the last 50 years, a massive literature on the health consequences of smoking and drinking has been produced by researchers working in dozens of fields of study. As such, it would be impossible to review the entire body of work here. Therefore, this literature review will focus mainly on the comprehensive reviews

of the literature done in recent years. I have separating this review into four sub-sections, which will review the links that smoking and drinking have with hypertension, high cholesterol, diabetes, and arthritis.

4.2.1 Smoking, Drinking, and Hypertension

Hypertension, or high blood pressure, increases the risk of ischemic heart disease, or reduced blood supply of the heart muscle (Lewington, Clarke, Qizilbash, Peto, & Collins, 2002). Hypertensive heart disease is responsible for more than 30,000 deaths annually in the United States, and ischemic heart disease is responsible for another 350,000 (Hoyert & Xu, 2012). Hypertension also leads to a faster progression of kidney failure in those with chronic kidney disease (Krzesinski & Cohen, 2007). Chronic Kidney disease due to hypertension is responsible for nearly 30,000 deaths annually in the United States (Hoyert & Xu, 2012). All told, hypertension is one of the most prominent preventable risk factors for heart disease in the world.

The causes of hypertension are complex and numerous, though age, genes, environmental factors, dietary salt intake, low consumption of fruits, dietary fat intake, lack of exercise, high weight, and alcohol intake have all been implicated (Whelton, He, Appel, & et. al., 2002). In the United States, 31% of adults have hypertension, which is characterized by a systolic blood pressure level of 140 mmHg or higher and a diastolic blood pressure level of 90 mmHg or higher. Men and women develop hypertension at similar rates overall, but men are more susceptible to the disease before the age of 45. African Americans are much more likely to have high blood pressure than whites and Hispanics (Go, et al., 2013).

Research on the effects of smoking on blood pressure date back more than 100 years. Erich Hesse documented an increase in subject's heart rate and blood pressure immediately after smoking in 1907 (Hesse, 1907). The nicotine in cigarettes operate through adrenergic mechanisms, or in other words increasing levels of adrenaline (epinephrine) and to a smaller degree noradrenaline (norepinephrine). Increases in these neurotransmitters leads to increases in the smoker's blood pressure and heart rate (Dept. of Health and Human Services, 2010). However, these increases are short-term.

There is no evidence that smoking is associated with hypertension, and population studies suggest that smokers have lower average blood pressures than nonsmokers (Dept. of Health and Human Services, 2004). Similar to smoking and body weight, however, smokers who quit may still see their blood pressure drop on average, particularly if smoking leads to changes in other risk factors for high blood pressure, such as obesity.

More than 40% of adults who are obese also have hypertension. This is partly due to their shared risk factors in a poor diet and lack of exercise, but there is some evidence that obesity can cause hypertension which is more resistant to pharmaceutical treatments (Dorresteijn, Visseren, van den Meiracker, & Spiering, 2011). By influencing the risk factors for obesity, changes in cigarette smoking could lead to changes in hypertension rates, which may convey further reductions in the risk of heart disease beyond those conveyed directly.

Alcohol consumption does have an effect on hypertension, though there is uncertainty about the potential reductions in blood pressure at light-to-moderate alcohol consumption (Sesso, 2010). There is strong evidence that heavier drinking of more than two drinks per day in men and one per day in women increases the risk of hypertension. Drinking below this threshold, however, has been linked to a reduced risk of ischemic heart disease (CDC, 2012), but whether or not the mechanism for this is through reduced blood pressure is still a matter of debate. Clinical studies show that reducing alcohol consumption to no more than one drink per day for men and half a drink per day for woman lowers blood pressure in hypertensive individuals (Whelton, He, Appel, & et. al., 2002). What has not been established, however, is the effects of alcohol consumption on the blood pressure of those without hypertension, where low-to-moderate alcohol consumption could be beneficial.

4.2.2 Smoking, Drinking, and High Cholesterol

Hypercholesterolemia, or high levels of cholesterol in the blood, increases the risk of atherosclerosis and coronary heart disease, or the clogging of the arteries that supply blood and oxygen to the heart. While there are many risk factors for atherosclerosis and coronary heart disease, hypercholesterolemia is the critical factor by which the others operate (Bhatnagar, Soran, & Durrington, 2008), and people with high cholesterol (more than 200mg/dL) double their risk of heart disease (CDC, 2011). Coronary heart disease is the leading cause of death in the United States, claiming nearly 600,000 lives each year (Hoyert & Xu, 2012).

Different types of cholesterol are thought to have different effects. Specifically, low-density lipoprotein (LDL) cholesterol is more strongly associated with a higher risk of atherosclerosis and heart disease, while high-density lipoprotein (HDL) is thought to be protective by carrying LDL cholesterol to the liver to be removed from the body (Carmena, Duriez, & Fruchart, 2004). These two types of cholesterol are commonly referred to as the “good” (HDL) and “bad” (LDL) cholesterol.

High cholesterol has some genetic risk factors, but is largely the result of dietary choices and obesity (Bhatnagar, Soran, & Durrington, 2008). Consumption of foods high in fat, particularly saturated fat, lead to high cholesterol levels in the blood and increase the risk of hypercholesterolemia (Sacks & Katan, 2002). Clinical studies also suggest that overweight and obese individuals can lower their blood cholesterol by losing weight (Ito, McGowan, & Moriarty, 2011). Overall, 33.5% of American adults have high cholesterol (CDC, 2011).

Smokers tend to have high levels of LDL cholesterol and lower levels of HDL cholesterol in their blood, which can lead to hypercholesterolemia (Dept. of Health and Human Services, 2010). Further, several studies have shown that people who take up smoking see a decrease in their HDL cholesterol, and those who quit smoking see an increase (Dept. of Health and Human Services, 1990). Further, hypercholesterolemia’s strong association with obesity may open a different channel by which smoking could affect blood

cholesterol levels. Changes in smoking that lead to weight loss could influence cholesterol levels beyond the direct effect of smoking.

Alcohol consumption has been linked to higher HDL cholesterol levels, with more drinking leading to higher HDL cholesterol (Hulley & Gordon, 1981). Low-to-moderate drinking is also associated with lower LDL cholesterol levels, which may be a pathway by which such levels of drinking reduce the risk of heart disease (Langer, Criqui, & Reed, 1992). There is little recent research on the relationship between alcohol consumption and high cholesterol in the overall population, however. It is unclear whether or not policies which affect drinking will affect the prevalence of high cholesterol.

4.2.3 Smoking, Drinking, and Diabetes

Type 2 Diabetes mellitus (referred to hereafter as just diabetes) is a metabolic disease characterized by high blood sugar caused by insulin resistance on the cellular level. Diabetes is the seventh leading cause of death in the United States, with nearly 75,000 deaths each year (Hoyert & Xu, 2012). When it is left untreated, diabetes can also lead to a greater risk of heart disease and chronic renal failure (CDC, 2011). Further, diabetic persons are more likely to suffer from hypertension, damage to the retina of the eye, nervous system diseases, amputations, and dental diseases. Considering these other conditions, diabetes contributes to another 160,000 deaths each year.

While diabetes does have genetic factors, it is primarily caused by dietary choices (Risérus, Willett, & Hu, 2009). Particularly, diets high in saturated fats and trans fatty acids greatly increase the risk of developing diabetes. These diets reduce the sensitivity of insulin receptors, which allows cells to absorb glucose. The insensitivity of insulin receptors leaves a higher level of glucose in the blood stream, and if this level surpasses the rate at which the kidneys can reabsorb it, it causes the complications associated with diabetes (CDC, 2011). Overall, 11.3% of American adults have diabetes.

Many studies have shown that smoking increases a person's risk for diabetes by impairing insulin sensitivity (Dept. of Health and Human Services, 2010). Further, smoking causes greater complications in those with diabetes through negative effects on metabolic control, which can also interfere with treatments for the disease. Smokers who quit smoking also experience a reduced risk of diabetes. These results consistent across race and gender.

Research has shown that alcohol consumption has a j-shaped relationship with the risk of diabetes for both men and women. A comprehensive review of 20 studies showed protective effects for an average of one drink per day, and deleterious effects when drinking rose beyond three drinks per day (Baliunas, et al., 2009). Most of these studies, however, controlled for a person's weight, which may change due to differences in alcohol consumption. If greater alcohol consumption leads to higher weight, this may change the overall effects of alcohol on the risk of diabetes.

The largest preventable cause of diabetes, however, is obesity. One study found that being obese is associated with 42.1% increase in the relative risk of diabetes (Chan, Rimm, Colditz, Stampfer, & Willett, 1994). Another found that every kilogram of weight lost reduced the risk of diabetes by 16% (Hamman, et. al., 2006). Bariatric surgery has also been shown to resolve diabetes in 78.1% of patients (Buchwald & et. al., 2009). Central abdominal fat also puts people at a greater risk of developing diabetes (Anjana, et al., 2004).

Previous research has directly links cigarette smoking to diabetes, and shown a U-shaped relationship for alcohol consumption. However, if these behaviors also affect body weight, that too may play an important role in the development of diabetes. Alcohol in particular, which tends to lead to central abdominal weight gain, may be of particular importance. To my knowledge, no research has attempted to estimate the causal impact of factors which influence cigarette and alcohol consumption on diabetes.

4.2.4 Smoking, Drinking, and Arthritis

Arthritis is a disorder involving the inflammation of one or more joints. There are more than 100 different types of arthritis, though the most common is osteoarthritis (CDC, 2013). While arthritis is not a primary cause of death, it can result in substantial pain and loss of functioning. Autoimmune forms of arthritis, such as rheumatoid arthritis, also increase the risk of respiratory infections and pneumonia, which are the cause of nearly 200,000 deaths annually (Hoyert & Xu, 2012). About 25% of American's have arthritis, nearly two-thirds of which are under the age of 65 (CDC, 2013).

Smoking is not directly linked to most kinds of arthritis, though there is some evidence that it increases the risk of rheumatoid arthritis. A 1993 study examined more than 50,000 adult Finns over 16 years and found current smoking nearly quadrupled the relative risk of rheumatoid arthritis in men, but not woman (Heliövaara, Aho, Aromaa, Knekt, & Reunanen, 1993). Other studies, however, found an increased risk of rheumatoid arthritis in woman who smoke (Vessey, et. al., 1987; Karlson, et al., 1999). A 1996 study confirmed the increased risk among identical twins with different smoking behaviors (Silman, Newman, & Macgregor, 1996).

Alcohol has been linked to a decreased risk of rheumatoid arthritis (Voight, et. al., 1994; Källberg, et. al., 2009; Maxwell, et. al., 2010) and an increased risk of gout (Choi, et. al., 2004; Choi, 2005), a type of arthritis caused by elevated levels of uric acid in the blood leading to recurrent attacks of acute arthritis. These types of arthritis only affect about 1-3% of the population, however.

Overweight and obesity are major contributors to osteoarthritis, the most common form of arthritis in America. Each five kilograms of weight is associated with a 36% increase in the risk for knee osteoarthritis (March & Bagga, 2004), and obesity is responsible for 25% of cases of hip osteoarthritis (Gelber, 2003). Further, 89% of obese patients who undergo bariatric surgery report relief of pain in one or more joints (Lementowski & Zelicof, 2008).

While the direct effects of smoking and drinking on arthritis are most likely minor, they may have larger effects to the degree in which they impact overweight and obesity. To my knowledge, no research has attempted to estimate the causal impact of factors which influence cigarette and alcohol consumption on arthritis.

4.3 METHODS

Chapters 2 and 3 have already demonstrated the role cigarette and beer taxes play on body weight and obesity. Obesity is an intermediate risk factor for other health outcomes, however. If the taxes on cigarettes and beer influence body weight, they should also influence these other health outcomes. Four health outcomes in particular are of interest to this analysis: hypertension, diabetes, high cholesterol, and arthritis.

Based on the review of the medical literature, we would expect there to be no relationship between cigarette taxes an hypertension, where there is no direct medical link. However, building from the results in chapter 2, where more cigarette smoking increases body weight, and a higher body weight increases the risk of hypertension, the expected effect of cigarette taxes on hypertension is negative. The same is true for diabetes, high cholesterol, and to some extent arthritis. Smoking is directly linked to a greater risk of diabetes and high cholesterol, and has a link to relatively rare forms of arthritis. These effects would compound with the effect of higher weights. In the case of arthritis, only a very small effect, if any, should be expected. Osteoarthritis can be caused by obesity, but changes in body weight due to changes in the cigarette tax are unlikely to cause the magnitude of weight change needed to have a measurable effect on arthritis rates. Therefore, in a Bayesian sense, the estimate for arthritis acts more as a falsification test for potential endogeneity.

The effect of beer taxes on these health outcomes is less clear cut. In the case of hypertension, the previous literature has identified a j-shaped relationship, where 1 to 2 drinks per day may lower blood pressure, while more than 2 drinks per day will raise it. The results shown in chapter 3 show that alcohol consumption

can lead to weight gain as well, which increases the risk of hypertension. Therefore, the direction of the effect of beer taxes is ambiguous, and its estimation should shed light on which of these effects dominates for the average person. This is also the case for diabetes and cholesterol levels, where a similar j-shaped relationship has been observed. Alcohol consumption has been linked to a decreased risk of rheumatoid arthritis, and an increased risk of gout. Because this link has only been loosely established, and these forms of arthritis are relatively rare, no significant effect is expected. As with cigarette taxes, this should act mostly as a falsification test for potential endogeneity.

Following the analytical framework presented in section 1.2, health outcomes such as hypertension, diabetes, high cholesterol, and arthritis, would depend on smoking, drinking, diet, and exercise. This simplistic approach overlooks many other factors which impact these diseases, and which make a direct estimation impossible. However, the model does offer a complete picture of how policy changes, which are assumed to be exogenous from these other impacts, affect the disease outcomes.

To determine the impact tax policy on the health outcomes listed, I estimate the following reduced-form equation:

$$HO_{its} = \beta_0 + \beta_1 CigTax_{ts} + \beta_2 BeerTax_{ts} + \beta_3 X_{its} + \beta_4 UR_{ts} + Year_t + State_s + \varepsilon_{its}$$

where i denotes the individual, t denotes the time, and s denotes the state. HO is a health outcome (a binary variable equal to one if the person reports being diagnosed with condition and a 0 if they report not having been diagnosed with it), CigTax is the excise tax placed on a pack of cigarettes, BeerTax is the excise tax placed on a gallon of beer, X is a set of individual-specific covariates, UR is the state-level unemployment rate in time t, and Year and State are year and state fixed effects respectively. The individual covariates contained in X include age group, gender, race, education level, marital status, and income.

Controlling for state and year trends, broader state-level economic conditions, and individual-specific covariates, taxes on cigarettes and beer serve as an exogenous indicator of changes in cigarettes and

alcohol consumption. Therefore, in this model it is differences in these tax rates within states, compared to other states without a change in their rates that will estimate the causal impact of these taxes on health outcomes.

Because these health outcomes can take time to develop and be diagnosed, I also estimate the model using lagged versions of the tax variables. However, the interpretation of this model should not be construed to mean that a contemporary price change leads to immediate effects. The inclusion of state and year fixed-effects make the observations comparable across states and years. The effect is identified on difference in the relative level of taxes within states compared to other states, and not on changes in the tax rate.

4.4 DATA

The primary source of data for this analysis is the BRFSS, which is described in more detail in section 1.3. The BRFSS data provides individual-specific data on weight, height, age, gender, education, income, marital status, and race. Additionally, the BRFSS reports responses to questions about various health conditions. Among these are questions about blood pressure, diabetes, cholesterol, and arthritis. Respondents are asked whether or not they have ever been told by a doctor that they have the condition. Efforts are also taken to differentiate between the reasons for the diagnosis. In the case of diabetes, those diagnosed only while pregnant are classified differently than other diagnoses.

In all cases, questions about these health conditions were not asked to all respondents, but were part of a rotating core of questions. In the case of hypertension, there respondents from all of the included states in all of the years except 2006, 2008, and 2010. Responses about diabetes diagnoses have been part of the fixed core and collected consistently since 1988. The question regarding a diagnosis of high blood cholesterol levels was asked from 1987 to 2005, and again in 2007 and 2009. Responses regarding arthritis diagnoses were collect from 1996 to 2005, and also in 2007, 2009, and 2010.

State-level taxes on beer and cigarettes were collected from *The Brewers Almanac* and *The Tax Burden on Tobacco* respectively. The tax on beer represents a per-gallon excise tax placed on distributers, while the tax on cigarettes represents an excise tax paid per pack. Additional data on the state-level unemployment rate comes from the Bureau of Labor Statistics. Table 4.1 gives summary statistics for key variables.

Table 4.1

Summary Statistics		
Variable	Mean	S.D.
Hypertension	0.450	(0.497)
Diabetes	0.065	(0.247)
High Cholesterol	0.306	(0.461)
Arthritis	0.259	(0.438)
Cigarette Tax	0.557	(0.558)
Beer Tax	0.510	(2.091)

4.5 RESULTS

With binary variables on the right-hand side, I estimated both a linear regression model and a logit model. In all cases, the results were consistent between the models. For the ease of interpretation, I will focus on the results of the linear regressions. Table 4.2 presents the results of these estimations of the impact of cigarettes and beer taxes on hypertension. In these estimations, the taxes on cigarettes and beer were divided by 100. As such, the point estimates should be interpreted as percentage point changes. A one-dollar increase in the cigarette tax results in a 0.78 percentage point decline in diagnosed hypertension rates. This finding contrasts with the effect of the beer tax, where a one dollar increase per gallon results in a 0.25 percentage point increase in hypertension rates. The latter case suggests that the protective effects of low- or moderate-alcohol consumption with regard to high blood pressure dominate.

Table 4.3 shows the impact of cigarettes and beer taxes on diabetes. Here, the effect of cigarettes and alcohol are in concert with one another. A one dollar increase in the cigarette taxes reduces diabetes prevalence by 0.24 percentage points, while a one dollar increase in the beer tax reduces prevalence by

0.15 percentage points. These results clarify the ambiguity in the medical literature on the effect of alcohol consumption on diabetes. Here the protective effects appear to be dominated by the effects on weight.

Table 4.2

Impact of Cigarette and Beer Taxes on Hypertension		
Variable	Hypertension	
	OLS	Logit
Cigarette Tax /100	-0.781***	-0.666***
	(0.279)	(0.251)
Beer Tax /100	0.252***	0.250***
	(0.080)	(0.085)
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.2975	0.2298
N	2,443,294	2,443,294

Table 4.3

Impact of Cigarette and Beer Taxes on Diabetes		
Variable	Diabetes	
	OLS	Logit
Cigarette Tax /100	-0.243***	-0.116**
	(0.065)	(0.924)
Beer Tax /100	-0.149***	-0.195***
	(0.041)	(0.880)
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.0622	0.1328
N	4,079,932	4,079,932

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Table 4.4 and 4.5 show the effects of the cigarette and beer taxes on high cholesterol and arthritis rates respectively. In neither are cigarette or beer taxes a significant cause of the health outcome. In the case of high cholesterol, the effect of cigarettes was predicted to be negative and the effect of alcohol was predicted to be ambiguous. The results suggest negative effects for both, but the estimates are not significant different from zero. As for the effects on arthritis, the results show no significant effect from either cigarette or beer taxes.

Table 4.4

Impact of Cigarette and Beer Taxes on High Cholesterol		
Variable	High Cholesterol	
	OLS	Logit
Cigarette Tax /100	-0.120	-0.158
	(0.255)	(0.244)
Beer Tax /100	-0.196	-0.219
	(0.149)	(0.154)
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.0726	0.063
N	1,821,374	1,821,374

Table 4.5

Impact of Cigarette and Beer Taxes on Arthritis		
Variable	Arthritis	
	OLS	Logit
Cigarette Tax /100	0.096	0.060
	(0.206)	(0.196)
Beer Tax /100	-0.625	-0.616
	(0.482)	(0.515)
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
R^2	0.1876	0.1739
N	1,669,535	1,669,535

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The results presented above are all estimated for the concurrent tax rate. These health outcomes may take some time to develop and result in a diagnosis, however. Table 4.6 shows the effect for lagged tax rates. The taxes are lagged by 1, 6, and 12 months. All of the estimates appear to be consistent across time, except for the effect of the beer tax on hypertension. The effect of the beer tax on hypertension appears to fad as the tax grows more distant in time. At six month, the tax is no longer a significant factor for hypertension, and by 12 months the point estimate falls close to zero. This contrasts with the remarkable consistency of the other estimates over time.

Another method of estimating the impact of these policies is to use them as instruments for the health behavior they impact. Cigarette taxes impact health by impacting smoking, and alcohol taxes impact health by impacting alcohol consumption. Following from the previous chapters, I can also use other policies, such as smoking bans and drunk driving laws, to overidentify the models and provide a test of the exclusion restriction, or the assumption that these policies do not impact other determinants of health through pathways other than smoking or drinking. Importantly, the following results all measure taxes in dollars, rather than cents.

Table 4.6

Impact of Lagged Cigarette and Beer Taxes on Health Outcomes				
	Hypertension	Diabetes	High Cholesterol	Arthritis
Cigarette Tax (1 Month Lag)	-0.729***	-0.235***	-0.082	0.044
	(0.267)	(0.067)	(0.251)	(0.204)
Cigarette Tax (6 Month Lag)	-0.954***	-0.258***	-0.193	-0.060
	(0.242)	(0.074)	(0.261)	(0.200)
Cigarette Tax (12 Month Lag)	-0.794***	-0.246***	-0.126	0.328
	(0.248)	(0.083)	(0.312)	(0.204)
Beer Tax (1 Month Lag)	0.209**	-0.148***	-0.194	-0.632
	(0.083)	(0.041)	(0.148)	(0.482)
Beer Tax (6 Month Lag)	0.089	-0.148***	-0.203	-0.648
	(0.124)	(0.040)	(0.149)	(0.481)
Beer Tax (12 Month Lag)	0.012	-0.147***	-0.193	-0.609
	(0.133)	(0.040)	(0.148)	(0.488)

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Tables 4.7 – 4.10 show the estimated coefficients for both the first and second stages of the instrumental variables approach. In all of these tables, I estimate two basic models. The first (left-hand column) estimates the impact of the cigarette tax and smoking bans on whether or not a person smokes, and uses the predictions of that binary variable to predict the health outcome. The second (right-hand column) estimates the impact of the cigarette tax and smoking bans on the number of cigarettes smoked for only those who current smokers, then uses the predictions for cigarettes smoked to predict the health outcome.

The reasoning behind each of these models is described in more detail in chapter 2, and as it is the case there, the second model using cigarettes smoked is considered ideal but limited by data. Taxes and smoking bans impact smoking outside of just the decision about whether or not to smoke at all. Additionally, because cigarette taxes and smoking bans have a small effect on smoking status, the results presented apply only to a highly localized group, prohibiting any broad conclusions. Notable, however, is the consistency of the first stage estimates across the different samples.

Table 4.7 examines the impact of smoking on hypertension. Smoking is associated with a 136 percentage point increase in the risk of hypertension, a result which is mathematically impossible. This is likely due to a weak first stage, where cigarette taxes and smoking bans do not significantly impact smoking status. An F-test of the relevance of these variables yields an F-statistic of 1.926, which is too low to make any conclusions based on these results. The second model, using cigarettes smoked, yields a strong first stage, with an F-statistic of 6.726. In the second stage, one cigarette per day is associated with a 1.7 percentage point increase in the risk of hypertension. With the average smoker smoking about 20 cigarettes per day, these results suggest a 34 percentage point increase in hypertension risk for the average smoker. While the magnitude of these estimates is worth examining, neither of the second stage estimates are statistically significant.

The results shown in table 4.7 are in disagreement with the full-sample reduced-form results shown in table 4.2. By the instrumental variable approach, a one dollar increase in taxes would provoke the average smoker to smoke about two fewer cigarettes per day, which translates to a 3.4 percentage point decline in hypertension risk. The reduce-form results estimate this effect to be about 0.8 percentage points. Some of this difference may be due to the differences in the samples used, however. The IV estimation used data only from 1990-2000, whereas the reduced-form results make use of the full 1984-2010 dataset. Previous findings have shown that the responsiveness to cigarette taxes has decreased in recent years, possibly because remaining smokers are less price sensitive.

Table 4.8 estimates the two-stage models for diabetes. Cigarette taxes and smoking bans appear to strong effect both smoking status and the number of cigarettes smoked per day. In the second stage, smoking is associated with a 42.6 percentage point increase in the risk of diabetes, and a 0.2 percentage point increase per cigarette smoked each day. The latter of these estimates was not statistically significant, but was in line with the reduced-form estimates in table 4.3. The coefficient on smoking status is about 10 times larger than what is estimated in the second model. Again, the small impact of taxes and bans in the first stage

may be the culprit here. If these impacts are underestimated, then the results of the second stage will be biased upwards.

Table 4.9 and 4.10 turn the analysis to cholesterol and arthritis, where no significant impact was found in the reduced form estimates. This is true for the IV approach as well, where no second stage results are found to be statistically significant. However, the estimates for the first model in table 4.9 produce a troubling result when the exclusion restriction is tested by the Wooldridge overidentification test. Here a $\chi 2$ of 8.567 rejects the hypothesis that these instruments are valid with a p-value of 0.003. Cigarette taxes or smoking bans appear to impact high cholesterol outside the pathway of whether or not someone smokes. Notably, however, these variables pass the overidentification test when used to estimate the number of cigarettes smoked per day.

Table 4.7

IV Estimation Results for Smoking and Hypertension		
Variable	Smoker	Cigs/Day
Cigarette Tax	-0.003*	-2.246***
	(0.002)	(0.520)
Smoking Bans	-0.001	-0.314
	(0.001)	(0.453)
F-statistic	1.926	6.726
Variable	Hypertension	Hypertension
Smoker	1.356*	-
	(0.700)	-
Cigarettes per Day	-	0.017*
	-	(0.010)
χ^2 *Overid*	0.047	2.939
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
First Stage R^2	0.0803	0.1653
N	2,123,780	144,743

Table 4.8

IV Estimation Results for Smoking and Diabetes		
Variable	Smoker	Cigs/Day
Cigarette Tax	-0.003***	-1.988***
	(0.001)	(0.457)
Smoking Bans	-0.002***	-0.773*
	(0.001)	(0.411)
F-statistic	10.482	8.267
Variable	Diabetes	Diabetes
Smoker	0.426***	-
	(0.157)	-
Cigarettes per Day	-	0.002
	-	(0.005)
χ^2 *Overid*	3.051	1.653
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
First Stage R^2	0.0844	0.0694
N	3,867,201	220,533

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Because the arthritis data is collected only after 1996, I am unable to estimate the effect of smoking bans on the average number of cigarettes smoked in the first stage for table 4.10. The number of cigarettes smoked is only producible for data between 1984 and 2000, leaving only 5 years of data on which to estimate the coefficient. Unfortunately, there is no variation in smoking bans throughout the United States in this time period.

Table 4.9

IV Estimation Results for Smoking and High Cholesterol		
Variable	Smoker	Cigs/Day
Cigarette Tax	-0.003**	-1.692**
	(0.002)	(0.700)
Smoking Bans	0.000	-0.544
	(0.001)	(0.658)
F-statistic	1.870	3.339
Variable	High Chol.	High Chol.
Smoker	-0.451	-
	(0.644)	-
Cigarettes per Day	-	0.008
	-	(0.015)
χ^2 *Overid*	8.567	0.530
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
First Stage R^2	0.0708	0.0555
N	1,678,098	88,072

Table 4.10

IV Estimation Results for Smoking and Arthritis		
Variable	Smoker	Cigs/Day
Cigarette Tax	-0.000	-2.967*
	(0.002)	(1.738)
Smoking Bans	0.001	-
	(0.001)	-
F-statistic	1.169	2.918
Variable	Arthritis	Arthritis
Smoker	-1.395	-
	(1.097)	-
Cigarettes per Day	-	0.019
	-	(0.029)
χ^2 *Overid*	0.153	-
Covariates	Y	Y
State FE	Y	Y
Year FE	Y	Y
First Stage R^2	0.0941	0.1158
N	1,664,592	26,486

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Turning to the impact of alcohol consumption on these health outcomes, tables 4.11 – 4.14 give the estimates for a two-stage model in which beer taxes and BAC limits while operating a motor vehicle are used to predict drinks consumed per day, the predicted values of which are used to predict the health outcome. The identifying assumption here is that beer taxes and BAC limits do not impact these disease outcomes except through changes in the average number of drinks consumed per day. Because the model

is overidentified, this assumption can be partially tested for whether or not both instruments satisfy or do not satisfy this assumption.

Table 4.11 estimates the impact of drinks per day on the risk of hypertension. The results of the first stage are consistent with the results found in chapter 3, but are not statistically significant. This may be due to the reduced sample size which includes reports of hypertension. Nevertheless, the second stage shows that an extra drink per day reduces the risk of hypertension by 8.6 percentage points. This is in line with the reduced-form results in table 4.2, which uses a slightly larger sample due to the inclusion of the BAC limit.

Table 4.12 gives the results for diabetes. Notably, the first stage results for the impact of the BAC level are about double the estimates everywhere else, and is highly significant. Nevertheless, the F-statistic for the importance of these instruments is weak at 4.839. In the second stage, a drink per day is associated with a 6 percentage point increase in diabetes risk. This estimate is not statistically significant, however.

Table 4.11

IV Estimation Results for Drinking and Hypertension	
Variable	Drinks/Day
Beer Tax	-0.045*
	(0.0025)
BAC Limit	0.292
	(0.314)
F-statistic	2.073
Variable	Hypertension
Drinks per Day	-0.086*
	0.050
χ^2 *Overid*	0.948
Covariates	Y
State FE	Y
Year FE	Y
First Stage R^2	0.0650
N	2,359,113

Table 4.12

IV Estimation Results for Drinking and Diabetes	
Variable	Drinks/Day
Beer Tax	-0.045
	(0.035)
BAC Limit	0.682***
	(0.247)
F-statistic	4.839
Variable	Diabetes
Drinks per Day	0.060
	0.061
χ^2 *Overid*	2.542
Covariates	Y
State FE	Y
Year FE	Y
First Stage R^2	0.0487
N	3,650,795

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

Tables 4.13 and 4.14 examine the impact of alcohol consumption on high cholesterol and arthritis, where no effect was observed in the reduced-form results. Likewise, no significant effect was observed in the two-stage estimates. Notably, however, the Wooldridge overidentification test for the arthritis estimates finds that one of these instruments may not be valid. Since alcohol consumption is not predicted to impact arthritis, it is unclear what these results mean.

Table 4.13

IV Estimation Results for Drinking and High Cholesterol	
Variable	Drinks/Day
Beer Tax	-0.125**
	(0.056)
BAC Limit	0.362
	(0.285)
F-statistic	3.300
Variable	High Chol.
Drinks per Day	0.377*
	0.212
χ^2 *Overid*	3.668
Covariates	Y
State FE	Y
Year FE	Y
First Stage R^2	0.0539
N	1,765,569

Table 4.14

IV Estimation Results for Drinking and Arthritis	
Variable	Drinks/Day
Beer Tax	-0.074
	(0.058)
BAC Limit	0.371
	(0.362)
F-statistic	1.277
Variable	Arthritis
Drinks per Day	0.169
	0.255
χ^2 *Overid*	5.310
Covariates	Y
State FE	Y
Year FE	Y
First Stage R^2	0.0411
N	1,559,259

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

One important limitation highlighted by the IV analysis is the weakness of the instruments in influencing smoking and drinking behavior. While the taxes are seen to have a significant effect in the full sample, these smaller samples may not offer the same level of precision. The inclusion of the BAC limits also offers insight into the effects of those policies, and offers a chance to test the identifying assumption of the analysis. Interestingly, this test questions the validity of the instruments when used to predict the effect of smoking

and drinking on arthritis. While the effect is theorized to be zero, the policies are nevertheless assumed to be exogenous. Thus, it is unclear whether or not the effects estimated are consistent.

4.6 Discussion

Smoking and drinking have numerous, well studied effects on health. From cancer to heart and liver disease, smoking and drinking account for nearly 520,000 deaths annually, and are the 1st and 3rd leading causes of death in the United States (Mokdad, Marks, Stroup, & Gerberding, 2004). Chapters 2 and 3 of this dissertation have shown that smoking and drinking also impact obesity, the risk factors for which are the second leading causes of death in the U.S. It is pertinent, then, to assess how policies which aim to or have the effect of reducing smoking and alcohol consumption impact other health outcomes related to obesity.

Hypertension is a major risk factor for heart disease and afflicts more than 67 million people in the U.S. Diabetes afflicts 26 million, and is the 7th leading cause of death in the U.S. High Cholesterol is a problem for 71 million American, and like hypertension is a major risk factor for heart disease. Arthritis is the country's most common cause of disability, affecting 52.5 million U.S. adults. Together, these represent some of the largest health concerns in the country. In light of the results found in the previous chapters, estimating population effects for a common policy tool like the excise tax is long overdue.

The results presented in this chapter suggest that cigarette taxes have significant effects on health outcomes like hypertension and diabetes. Hypertension is not directly tied to smoking, but is tied to it through the mechanism of changing diet and exercise behaviors. Diabetes is directly tied to smoking, but may be worsened by the mechanism of changing diet and exercise behaviors. Overall, a one dollar increase in the cigarette tax would result in a 0.781 percentage point reduction in hypertension rates, and a 0.243 percentage point reduction in diabetes prevalence. In terms of the population, this would translate to 523,000 fewer people with hypertension, and 63,000 fewer people with diabetes.

Taxes on beer also have an impact on hypertension and diabetes. In the case of hypertension, increases in the beer tax are associated with an increase in hypertension rates. This may be due to alcohol's more complex j-shaped relationship with hypertension. The average person consumes one alcoholic drink about every two days, below the point at which alcohol consumption could become dangerous for blood pressure. So for the average person in the sample, the reduction in consumption associated with a change in the beer tax pushes them up the left side of the j-shaped curve, putting them at greater risk of hypertension. A one dollar increase in the beer tax leads to a 0.252 percentage point increase in hypertension rates. This translates to about 170,000 more people with chronic high blood pressure.

Diabetes prevalence has a negative relationship with the beer tax, despite a similar j-shaped relationship between alcohol consumption and diabetes in the literature. A one dollar increase in the beer tax results in a 0.149 percentage point decline in diabetes prevalence, or about 39,000 fewer cases. This suggests that the j-shaped relationship bends much sooner for diabetes than it does for hypertension. Most people put themselves at a greater risk of diabetes with increased alcohol consumption.

There appears to be no significant effect of cigarette and beer taxes on instances of high cholesterol or arthritis. In the former case, this is contrary to expectations. Smoking and drinking are not strongly tied to cholesterol, but cholesterol is strongly tied to diet, which changes in smoking and drinking can impact. The results are consistently negative over time, with greater and greater lags for the tax rates, but never statistically significant. Further research should be conducted to see if this effect is real. In the latter case of arthritis, the insignificant results are consistent with expectations. While smoking and drinking have an effect on cases of rheumatoid arthritis, this is a rare form of the disease. The most common form, osteoarthritis, can be caused by obesity by putting extra strain on the knee joints. However, changes in the tax lead to relatively small changes in weight, and themselves are unlikely to be the cause of osteoarthritis. One point of concern, however, is the magnitude of the estimate on the beer tax. While it is never significant, it is consistent throughout the lagged tax rates, and it is sizable enough to merit further study.

Another limitation in the interpretation of these results is the inconsistency in the effect of the beer tax on hypertension rates, which appears to fade over time. There does not seem to be a clear theoretical or practical explanation for this result that would remain consistent with the effect on diabetes over time. One possibility is that diagnoses of diabetes may stick more than diagnoses of hypertension. That is, doctors will check to see if your blood pressure has been brought under control and tell you that you no longer have chronic high blood pressure, whereas they won't tell you that you no longer have diabetes. If this is the case, then an adjustment to the increased beer tax could explain the results.

It is also important to note another possible explanation for the results seen here. The data used contain only self-reported instances where the respondent has been told by a medical professional that they have one of these health conditions. It is possible that the taxes on cigarettes and beer merely affect the likelihood that a person would go to the doctor and receive a diagnosis. For example, if higher cigarette taxes lead to fewer doctor's visits, this may lead to fewer reported cases of hypertension simply because hypotensive or not, people aren't going to the doctor as often. This alternative explanation is addressed in part in chapter 2, where cigarette taxes are shown to have no impact on the frequency of doctor's visits.

The results presented here speak generally to the importance of studying the unintended consequence of public policy. Policies which may be aimed at improving public health could be causing harm in other related areas of health. Likewise, there could be unintended benefits to such policies. Understanding these relationships and estimating their magnitudes is and will remain an important area of study.

5 CONCLUSION

Smoking and drinking are two of the most important risk factors for non-communicable diseases. The other two are diet and exercise. Together, these health behaviors are a factor in the deaths of 36 million people globally every year. Around the world, governments are evaluating the policy tools at their disposal to reduce these numbers. This dissertation has focused on the effects of tax policy for cigarettes and alcohol and examined their effects on obesity and obesity-related diseases which lead to or are themselves non-communicable diseases.

The results have told an intricate and important story. Cigarette smoking and alcohol consumption are tied to body weight and obesity in a significant way. Higher taxes on these goods lead to lower weights and in the case of a cigarettes, a reduced risk of obesity. These effects also impact other health outcomes. Higher taxes on cigarettes leads to fewer cases of hypertension and diabetes, the latter being a condition only tied to smoking through its effect on diet and exercise. Higher taxes on alcohol leads to an increase in hypertension rates, confirming the j-shaped relationship found in the medical literature, but fewer cases of diabetes.

All of these results represent unintended consequences of those policies, which were conceived largely to address the externalities of second-hand smoke and alcohol abuse, prevent cancer and liver disease, or to simply raise revenue. In some of these cases, the results overturn the predictions of the conventional wisdom. Smoking, which is associated with lower weight and weight control, may lead to weight gain for heavier smokers. In other cases, the results confirm the theoretical predictions of the medical literature. Low-to-moderate alcohol consumption reduces a person's risk of hypertension.

In the context of the population, these effects are not small. They suggest that a one dollar increase in the tax on a pack of cigarettes and a gallon of beer would result in the loss of about 1 pound for the average

person. This would move about 3 million people out of the obesity category. The tax increase would also result in a net reduction of 353,000 cases of hypertension, and a combined 102,000 fewer cases of diabetes. These are figures that simply cannot be ignored when evaluating the merits of changing these policies.

Of course, the research done here has its limitations. Cigarette taxes appear to effect the weights of those who have never smoked. This could be explained by the nature of data, where individuals are not tracked over time, and the effect could be due to compositional changes in response to the tax. In other words, the tax pushes some people never take up smoking. This would not explain why reduced smoking leads to weight loss, however. Some evidence was found for dietary changes, but estimates were not significantly different from zero. Understanding these mechanisms remains a gap in the literature that should be explored further.

Another limitation of this work is the lack of persistence in the effect of beer taxes on weight and on hypertension. In both cases, the effect weakens with further lags of the tax. When the tax is lagged by 12 months, both of the effects seem to disappear. This may indicate that the effects of the tax are temporary, and that people adjust their behavior over time to return to a steady-state weight and risk of hypertension. Curious is the fact that the same does not hold for cases of diabetes, but this could be explained by hesitation on the part of physicians to overturn a diabetes diagnosis.

Future work in this area should focus on these limitations, and explaining the mysteries they leave unanswered. It should also carry these methods over to other nations, particularly to low- and middle-income countries where increasing rates of non-communicable diseases are a growing focus of policy makers. Many countries seek to avoid the experience of the United States when it comes to the detrimental health consequences of alcohol and tobacco consumption. Understanding how policies aimed at reducing their consumption effect other risk factors such as obesity and obesity-related conditions will be an important part of that effort.

These findings also fit into a larger literature on cigarettes, alcohol, and obesity. My findings in chapter 2 suggest that a $1 increase in the cigarette tax leads to a decline in BMI of 0.08 points. This is roughly half of what Gruber and Frakes (2006) find, and in the opposite direction from Chou, Grossman, and Saffer (2004). The findings are also significantly smaller than Courtmanche (2009), who finds a reduction in BMI between 0.13 and 0.59 points. Likewise, my findings for the effect of the tax on the risk of obesity is also smaller than what is found in the previous literature.

The reason for these differences may be due to the timeframe examined. My data runs from 1984 to 2010, while the others use data ending as early as 2000. Some recent literature has suggested that the effect of cigarette taxes has fallen over time (Callison & Kaestner, 2014). From a theoretical perspective, this may be due to the reduction in smoking over the same time period. Those who have not yet quit smoking are likely the ones who draw the most benefit from it, and are the least responsive to changes in the price. As much is seen in table 2.15, where I present first-stage results for the effect of taxes on smoking prevalence.

Between 1990 and 2010, a one dollar increase in cigarette taxes was associated with a 0.4 percentage point decline in smoking prevalence, from a base of 21.6 percent. Using price data from *The Tax Burden on Tobacco*, I calculate the price of a pack of cigarettes for the average person in this time period to be $3.27. If the tax is passed on entirely to the consumer, this would imply that a 10% increase in the price due to an increase in the tax would reduce prevalence by 0.61%, or an elasticity of -0.061. However, for 1990-2000, the average price for cigarettes was $2.11, and a $1 increase in the tax was estimated to lead to a 1.7 percentage point drop in prevalence over a base of 23%, implying a price elasticity of prevalence of -0.156. These price elasticities fall above and below, but not out of line with, the -0.126 price elasticity of prevalence found by Tauras (2006).

Without trying to extrapolate how the tax increase influence the price, where a significant amount of research suggests the price may increase by more than the tax (Chaloupka and Warner, 2000), I can also

look only as the tax elasticity of prevalence. Callison & Kaestner (2014) find that a 100% increase in the tax reduces smoking by about 5%. From 1990-2010, the average cigarette tax faced by consumers was $0.62. Increasing this tax by 100% would imply a 0.248 percentage point reduction in smoking, or a 1.2% change. Here my results would seem to suggest that people are less responsive to cigarette taxes than previously estimated.

Table 2.15 also shows the effect of the cigarette tax on the number of cigarettes smoked for 1990-2000. Here I find that a $1 increase in the tax leads to an average reduction of 1.96 cigarettes per day, where the average among smokers is about 20 cigarettes per day. Use the same average price per pack of $2.11, the data suggests smokers will reduce their smoking by about 10% for a 47% increase in price. This implies a price elasticity of demand of about -0.213. For context, Gallet and List (2003) survey 368 studies which estimate the price elasticity of demand for cigarettes and find a median estimate of -0.40. However, Chaloupka and Warner (2000) report a range of estimates from -0.14 to -1.23, with most falling between -0.2 and -0.5. My results fit in well with these previous findings.

The price elasticity of beer has also been widely studied. Leung and Phelps (1993) find an average estimate for the price elasticity of beer of -0.3, with most falling between -0.2 and -1.0. Likewise, in a meta-analysis of the literature, Gallet (2007) finds a median price elasticity of -0.36 across 315 studies. For alcohol in general, he finds a median price elasticity of -0.497. Several studies have also looked at the price elasticity for heavy drinkers, and found a significantly smaller elasticity. A review of ten such studies found an average elasticity for heavy drinkers of -0.01 (Wagenaar, Salois, & Komro, 2009).

There is limited evidence as to how much of the beer tax is passed on to consumers. Kenkel (2005) finds that a $1 increase in beer taxes would lead to a $2 increase in beer price. He finds similarly large price increases for wine and spirits as well. Young and Beilinska-Kwapisz (2002) also find that retail prices of

alcohol rise more than the amount of the tax, with a one dollar increase in the beer tax leading to a $1.86 increase in the retail price of beer.

Data limitations also make translating the results presented here into price elasticities of demand for alcohol difficult. The results are estimated with state-level excise taxes on gallons of beer, which produce roughly 10.67 drinks. So a $1 increase in the tax translates to about a $0.094 increase in the tax per drink. The ACCRA data described in section 1.3 includes retail prices on six-packs of beer, which average $6.16 over the time period, or about $1.03 per drink. The $0.10 tax increase was associated with a decline in alcohol consumption of 0.046 drinks per day, where the average drinks consumed per day was 0.43.

If the tax on beer is fully passed on to the consumer, the results imply a price elasticity of demand for beer of about -1.17. If the price is passed on to the consumer at a 2:1 ratio, this would imply a price elasticity of -0.59. Considering the literature on alcohol prices and taxes, the latter estimate would appear to be the most consistent, and fits in reasonably well with other estimates of the price elasticity. However, with an average beer tax of $0.51, the tax elasticity of beer is estimated to be -0.055. In other words, it takes large tax increases to move consumption.

Overall, my estimates fit in well with the previous literature. Where they expand on it is in examining how these responses to the change in the price of cigarettes and alcohol leads to other health outcomes. I find a significant impact on body weight and obesity, along with an impact on hypertension and diabetes. While the price elasticities for smoking prevalence and alcohol consumption are low, they are nevertheless significant. Large increase in these taxes would have a mild effect on use, but a significant effect on obesity, hypertension, and diabetes. In his seminal work, *An Inquiry into the Nature and Causes of The Wealth of Nations*, Adam Smith writes:

> “Sugar, rum, and tobacco, are commodities which are no where necessaries of life, which are become objects of almost universal consumption, and which are therefore extremely proper subjects of taxation.” (Book V, Chapter III)

There it is.

APPENDIX

From 1984 to 1994, the BRFSS is conducted in a select number of states. After 1994, data is consistently collected from all 50 states and the District of Columbia. To examine whether or not this mix of states in the early years of the sample plays an important role in the effects observed, I repeat the analysis seen in tables 2.6, 3.6, and 4.2-4.4 for the years 1990-2010 including only the 45 states which are available through that entire time period. Those states include Alabama, Arizona, California, Colorado, Connecticut, Delaware, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Vermont, Virginia, Washington, West Virginia, Wisconsin, and the District of Columbia. The results presented in tables below show the coefficients for the tax on cigarettes or alcohol respectively.

Appendix Table 1

Regression Results for Impact of Cigarette and Beer Taxes on Health Outcomes					
	BMI	Hypertension	Diabetes	Cholesterol	Arthritis
Cigarette Tax	0.079***	-0.794***	-0.279***	-0.300	-0.027
	(0.022)	(0.306)	(0.072)	(0.283)	(0.220)
Beer Tax	0.066***	0.253***	0.151***	-0.204	-0.781
	(0.015)	(0.080)	(0.041)	(0.150)	(0.538)
Covariates	Y	Y	Y	Y	Y
State FE	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y
R2	0.1129	0.2986	0.0623	0.0723	0.1889
N	3,814,070	2,247,263	3,690,119	1,663,892	1,499,682

*p-value < 0.10; **p-value < 0.05; ***p-value < 0.01

The results here show no significant differences from the results found using the entire dataset. This alleviates any concern that the results presented throughout this dissertation are being driven by changes in the sample of states included over the years of the analysis.

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Education

University of Illinois at Chicago, Spring 2014 (Expected)
Ph.D., Economics

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Certificate in the Teaching of Economics

Arizona State University, Spring 2009
B.S. in Economics, Political Science, and Psychology

Dissertation

The Health Consequences of Tobacco and Alcohol Policy

- *The Impact of Smoking Policy on Obesity*
  Measures the effect of cigarette taxes and bans on body weight through the channels of diet, physical activity, and other investments in health for different populations.
- *The Impact of Alcohol Policy on Obesity*
  Measures the effect of alcohol taxes and drunk driving policies on body weight through the channels of diet, physical activity, and other investments in health for different populations.
- *The Impact of Smoking and Alcohol Policy on Other Health Outcomes*
  Measures the effect of cigarette and alcohol taxes on health outcomes both directly related to consumption of cigarettes and alcohol, and indirectly related through diet and exercise, such as hypertension, diabetes, high cholesterol, and arthritis.

Research Interests

Primary Fields
Health Economics, Public Economics, Applied Microeconomics

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Behavioral Economics, Environmental Economics, Public Choice, Labor Economics, Sports Economics

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Microeconomics, Macroeconomics, Environmental, Health, Public Finance, Sports, Statistics, Econometrics

Teaching Experience

Department of Economics, University of Illinois at Chicago

Fall 2011 – Present, *Visiting Lecturer*

Economics 121: Principals of Macroeconomics
Economics 218: Intermediate Microeconomics for Business
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Supervised 2 Honors College Capstone Theses and 1 Honors College Research Project

Fall 2010 – Spring 2011, *Teaching Assistant*

Intermediate Microeconomics, Economics of Sports and Entertainment

Winner of the 2013 Oscar Miller Award for Teaching Excellence

Publications and Works in Progress

- *Quality of Alternatives, Institutional Preference, and Institutional Commitment Among First-Year College Students* (with Morris A. Okun and Natasha Mitric), Educational Psychology, 2009. Measures the effect of quality alternatives on institutional commitment and finds that students who attend a university ranked low in their preferences are more likely to drop out within their first year.
- *Effect of Marijuana Legalization on Consumption and Price*
  Using a novel dataset, this paper estimates the effects of the legalization of marijuana in Colorado and Washington on use and price.

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