The Spillover Effects of Smoking

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THESIS

Submitted as partial fulfillment of the requirements for the degree of
Doctor of Philosophy in Economics in the
Graduate College of the University of Illinois at Chicago, 2016

Chicago, Illinois

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DEDICATION

I dedicate this thesis to my parents for their love and support which has made this dissertation project a reality.

ACKNOWLEDGEMENTS

I have been fortunate to have a distinguished dissertation committee of accomplished scholars that has had a tremendous influence on my approach to this project and scientific work in general. I am indebted to Frank Chaloupka for chairing my committee and his generous advising and feedback. I thank Frank for his support through my various academic adventures, and sharing his data which made chapter 2 possible. In addition, I am also grateful to the other members of my dissertation committee: Ben Feigenberg, Darren Lubotsky, Deirdre McCloskey, and Tim Classen. I have benefited from being under their tutelage and receiving their thoughtful comments, constructive feedback, and great advice. The committee's enthusiasm for economics and scientific inquiry has truly been inspiring and has helped contribute to my own personal and intellectual development. I hope to continue their scientific tradition in my own academic work.

During the preparation of this thesis, I have additionally benefitted from comments and conversations from Art Carden, Harry David, Pavel Dramski, Irina Horoi, Michael Kelley, and Pauline Khoo, and I thank them. For both of the dissertation chapters I have attended numerous conferences and workshops where I received invaluable feedback and I thank the participants for improving the quality of my thoughts. I am also thankful for assistance from the economics department staff members Erin Toussaint, Carol Martell, and Jacqueline Sifuentes for helping me navigate the graduate program.

I am grateful to the Institute for Humane Studies for financially supporting my graduate studies through the Daniel Searle Fellowship, and the Mercatus Center for supporting my studies through the Adam Smith and Visiting Dissertation Fellowships that have intellectual engaged me and provided diverse perspectives on economic thought. I also thank the Charles Koch Foundation for providing a dissertation grant during my studies which provided time to work on

ACKNOWLEDGEMENTS (CONTINUED)

this project. I thank the Interuniversity Consortium for Political and Social Research (ICPSR) at the University of Michigan for providing access to the National Survey of Midlife Development and the National Survey of Children's Health. Lastly, I thank my parents for their encouragement and support during my long and sometimes tumultuous journey through graduate school.

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LIST OF ABBREVIATIONS

ACA	Affordable Care Act
BLS	Bureau of Labor Statistics
CPI	Consumer Price Index
DD	Differences in Differences
ESHI	Employer Supplied Health Insurance
FPL	Federal Poverty Level
HIPAA	Health Insurance Portability Accountability Act
MIDUS	National Survey of Midlife Development
NCHS	National Survey of Children's Health
NLSY79	National Longitudinal Survey of Youth 1979 Class
OLS	Ordinary Least Squares

SUMMARY

The large reduction in the number of smokers from nearly half of the population to about a sixth is one of the largest public health successes of the past fifty years. Evaluating the effects of such a large reduction in smoking on outcomes beyond the health of the smoker has been a salient and recurring topic in health economics. Discourse on these spillovers from smoking has often reached mixed conclusions. In this thesis, I evaluate two specific outcomes related to smoking such as the effect on earnings and the health of children. In the first chapter, I estimate the effect of smoking on the earnings of the smokers. In the second chapter, I examine the role of smoking behavior on the production of health capital in children and explore variations by race and socioeconomic status.

Chapter 1 explores the relationship between smoking and earnings using longitudinal data that contains a representative sample of Americans and a subsample of twins and siblings. I find smokers tend to earn less than non-smokers by approximately sixteen to eighteen percent. I attempt to investigate the origins of this earnings gap by investigating whether the proposed explanations, namely, that addiction related productivity declines or disproportionate health care usage actually contribute to the earnings gap. My analysis reveals that employer-supplied health insurance appears to be the primary mechanism that results in the earnings gap of smokers. Estimates from the sibling and twin fixed effects models reach similar conclusions, but with smaller magnitudes. Further analyses reveal that the negative effect on earnings increases with age and varies with gender, but, no differences in earnings are found between former smokers and non-smokers. The results suggest that smokers bear at least some of cost of smoking and firms adjust compensation based on the full range of worker quality incorporating health attributes.

Chapter 2 evaluates the relationship between prenatal smoke exposure and health in childhood. Decades of research have established a link between prenatal smoke exposure and health at birth. Yet, only a paucity of research has credibly examined outcomes later in childhood. I exploit exogenous variation in state cigarette taxes to estimate the causal impact of prenatal smoke exposure on multiple measures of children's well-being such as asthma, severity of asthma, and health status. I find an economically and statistically significant reduction in asthma rates. A one-dollar increase in state excise taxes on cigarettes reduces the prevalence of asthma by 1.7 percentage points with larger reductions for non-white children and children from poorer households. Furthermore, tax changes reduce inequality in children's health by SES since lower SES households appear to be more responsive.

Chapter 1

WHO PAYS FOR SMOKERS?

1.1 Introduction

Since the publication of the Surgeon General's report (U.S. Department of Health, Education, and Welfare, 1964) on the harmful effects of cigarette consumption, a vast literature has emerged exploring the economic consequences of smoking. Early attempts to quantify the economic costs of smoking predicted large labor market impacts arising from poor health that would translate into earnings reductions reflecting the reduced productivity and increased healthcare costs of smokers (Luce and Schweitzer, 1978; Oster, Colidtz, and Kelly, 1984). Yet, empirical investigations of smokers and their earnings have proven more challenging because smokers and non-smokers tend to differ on observable characteristics and likely differ on unobservable characteristics. As a result, causal evidence on the effects of smoking on earnings has been elusive[1].

To address the concerns from differential selection into smoking, studies investigating the effect of smoking of earnings have often invoked strong restrictions and exclusion restrictions. These studies have employed distinct approaches such as covariance restrictions (Auld, 2005), instrumental variables (Anger and Krasinksa, 2010; Van Ours, 2004), longitudinal PSID and NLSY data (Grafova and Stafford, 2009; Levine, Gustafson, and Velenchik, 1997; Cowan and Schwab, 2011). The estimates for the effect of smoking on earnings from these range from eight

[1] Chaloupka and Warner, (2000) contains a thorough selection of early studies evaluating the effect of smoking on wages.

to twenty-four percent, respectively. Despite the clever attempts at addressing the concern of unobservable variables, the studies generally estimate the overall wage penalty (except for Cowan and Schwab [2011] who examine earnings penalties emerging from employer supplied health insurance). Although many of these studies show that smokers earn less than non-smokers, it remains unclear whether the earnings reductions are driven by the diminished productivity of smokers, their use of health care services, or differences in skills between smokers and non-smokers

To identify the effect of smoking on earnings, I exploit variation within families using twin and sibling fixed effect models to address concerns of differential selection on observed and unobserved skills. Next, I attempt to disentangle hypothesized mechanisms that contribute to the earnings penalty such as health care costs versus addiction related productivity declines, by exploiting the provision of firm level employer supplied health insurance (ESHI). I compare the earnings of smokers versus non-smokers with ESHI to the earnings of smokers without ESHI versus non-smokers to examine whether health care costs or addiction related productivity are the primary factors that cause the reduction in earnings of smokers. To conduct the analysis, I use data from the first three waves of the National Study of Midlife Development on a representative sample of Americans and a special of twins and siblings.

I find that smokers tend to earn about seventeen percent less than non-smokers in the full sample with controls. Causal estimates for the effect of smoking on earnings from twin and sibling fixed effects models produce smaller effects of fifteen to sixteen percent reduction in earnings for smokers, but overall the effect of smoking on earnings is statistically similar vis-à-vis national representative of America. Two possible explanations for the reduction in the earnings of smokers are that smoking reduces productivity from addiction, and smoking raises

health care costs. Back of the envelope estimates for the effects of addiction related productivity on earnings are negative and large, but are statistically indistinguishable from zero. On the other hand, the estimates for smokers with ESHI are both economically and statistically significant and larger than estimates from the whole sample. As a result, the empirical facts are consistent with the hypothesis that smoker's higher health care costs are a primary driver of their reduction in earnings. Lastly, former smokers appear do not appear to bear an earnings penalty.

Analyzing the effects of smoking behavior is important because smoking is still a prominent negative health investment, as of 2015 around 17% of Americans continue to smoke (CDC, 2015), and discussions on tobacco control policy remain prominent in the public sphere. Scholars have often disagreed about the social costs of smoking and whether smokers pay their own way (Manning et. al, 1989; Chaloupka and Warner, 2000) by bearing the costs of their actions or whether smokers impose negative spillovers on society. Therefore, the findings of this paper have implications for both economics and public policy because they suggest smokers at the minimum pay for at least some of their health care costs through lower earnings. Understanding how smoking habits influence earnings contributes to the persistent discourse on sin taxes and also helps raise our understanding of wage dynamics more specifically how firms adjust compensation on the full dimensions of worker quality incorporating worker's health investments.

The wage dynamics between health insurance and health investments like smoking are especially salient since legislation at both the national level such as the Health Insurance Portability and Accountability Act (HIPAA) and state level such as through smoker protection laws impose restrictions that prohibit differential insurances prices beyond a certain threshold for smokers. Moreover, the findings of this paper are timely and relevant since recent legislation

such as the Affordable Care Act (ACA) also imposes price differentials on how much individuals can be charged for health insurance based on smoking status in addition to age and gender. The expansion of public insurance and the decoupling of insurance with employment raise important implications for the incidence of health behaviors like smoking.

1.2 Conceptual Framework

1.2.1 Wage-Benefit Trade-offs

The analysis presented in this paper examining the earnings losses of smoker invokes one of the fundamental assumptions of economics: the wage is the marginal product of labor. The wage includes a monetary component and non-monetary components. Money wages, although a substantial form of compensation to labor, exclude fringe benefits such as health insurance, vacation, and other perks contributing to the remaining wage. Following the health insurance framework from Currie and Madrian (1999) and Gruber (2000), workers are compensated based on their level of productivity and trade-off fringe benefits for money wages. As shown in equation 1, total earnings reflect the marginal product for individual employee i. Earnings in cash are denoted by E are the difference between the wage denoted by W and non-monetary compensation denoted by C. Health coverage is one form of non-monetary compensation that varies across individuals and included in C. Health insurance is offered at the firm level, and because of transactions costs and legislative restrictions on differential pricing, firms do not individually price health insurance. As a result, firms are unable to adjust compensation for fringe benefits, but they can adjust total compensation by reducing monetary earnings.

$$(1) \quad E_i = MPL = W_i - C_i$$

For smoking, the possible mechanisms that influence earnings include both fringe and non-fringe benefits. Table 1 contains estimates from Berman, Crane, Seiber, and Munur (2014) synthesizing the various projections from the public health literature on why smokers might earn less than non-smokers. The projections are disaggregated by various addiction and health insurance costs, and provide bounds for the economic costs of smoking. As table 1 illustrates, smoking could also influence wages through addiction related productivity declines such as smoking breaks, missed days of work, nicotine withdrawal in addition to the disproportionate use of fringe benefits such as insurance. The bounds for the estimates overlap in terms of whether health care costs versus productivity have larger impacts, but it is evident that health insurance costs are a substantial portion of the economic costs of smoking. Nevertheless, both pathways ultimately translate into an overall reduction of earnings for smokers. Smokers do have an advantage over non-smokers in terms of pension costs because non-smokers live longer and require pension payments for a longer period of time. Yet, the pension savings from smoking are fairly small with respect to health costs and work-related productivity.

Even though firms may not directly observe individual smoking habits, they can directly observe smoking breaks and symptoms of addiction. Nicotine withdrawal begins once a smoker has completed and also influences the smoker's physiological state which would have a direct effect on the marginal product of the smoker. Besides direct work effect, other measures of productivity such as sick days, reveal that smokers tend to have higher absence (Lundborg, 2010; Halpern et al, 2007). Health care costs also reveal a differential between smokers and non-smokers and serve as an indirect indicator because they work through fringe benefits also might reflect a measure of unproductivity.

Equation 2 provides a detailed framework for the empirical relationship between smoking and earnings. It shows that observed characteristics of individual *i* denoted by *X* such as age and gender along with unobserved characteristics such as ability, tastes, and preferences denoted by *Z* also influence earnings. Yet, estimating the effect of smoking on earnings poses empirical challenges because if smoking is correlated with unobserved attributes such as productivity that positively affect wages, then the failure to account for these regressors biases the smoking coefficient. The direction of bias depends on which unobserved mechanisms have bigger impacts. For example, the omission of ability from the regression estimation would produce a downward bias on the coefficient of smoking denoted by *S* in OLS estimates since it is positively correlated with earnings and negatively correlated with smoking. The omission of other attributes such as tastes and preferences that could be positively correlated with smoking and wages would produce an upward bias on the effect of smoking on earnings. Attempting to control for these attributes, especially for tastes and preferences is difficult.

$$(2) \quad E_i = \alpha_i + \beta S_i + \gamma X_i + \pi Z_i + \varepsilon_i$$

1.2.2 Empirical Evidence on the Trade-off Between Wages and Fringe Benefits

The theory of equalizing differences relates the relationship between wages and non-monetary compensation (Rosen, 1981). Empirical assessments of the theory of compensating differentials have shown that often workers tend to incur costs of their benefits. Seminal work by Gruber (1994) examines the effect of mandated maternity benefits and finds that it reduces the earnings for women of childbearing age. More recently, Thomas (2016) finds that employers adjust on other margins in addition to earnings. Notably, she finds maternity leave changed the composition of women's labor supply with more family oriented women remaining in the labor force. Since firms are unable to distinguish between family oriented and career oriented women,

ultimately it results in fewer promotions for all women. Overall, the trade-off between earnings and fringe benefits has been a consistent finding (Lubotsky, 2004) and has been demonstrated for workers' compensation (Fishback, 1994), obesity (Bhattacharya and Bundorf, 2011) and for smoking (Cowan and Schwab, 2015), among others.

The two notable studies investigating worker health investments and their earnings demonstrate how firms adjust wages in response to health behaviors such as smoking and obesity. First, Bhattacharya and Bundorf (2011) extend the theory of compensating differentials from health policies to health attributes to examine how obesity influences wages. They pool data across person-years of National Longitudinal Survey of Youth 1979 to employ a differences in differences (DD) research design that exploits the differences in earnings between the obese and non-obese and the differences in earnings between individuals in ESHI jobs versus non-ESHI jobs. The strength of the DD design is that it addresses the concern of unobserved ability since they exploit variation in insurance which tends to be offered at the firm level. The limitations of their DD approach is still the threat of selection bias that hinders the interpretation of their DD analysis as a causal parameter. Second, Cowan and Schwab (2015) employ the same DD identification strategy on the NLSY79 data to examine the effect of smoking on earnings by now comparing differences between smokers and ESHI. Their findings reveal smokers with ESHI earn considerably less than smokers without ESHI.

1.3 National Survey of Midlife Development

The data are from the National Survey of Midlife Development in the United States (MIDUS) with data collection for MIDUS 1 (Brim et al, 2011) occurring in 1995 and 1996. MIDUS 2 (Ryff et. al, 2012) and MIDUS 3 (Ryff et al, 2015) occur in 2006 and 2014, respectively. The main sample of MIDUS contains a nationally representative sample of

Americans, with a subsample of twins and siblings, in addition to a large oversample of urban underrepresented groups. The retention rate from the first survey to the second survey was about 70%, and the retention during the third wave is 55%, with both non-response and mortality contributing to attrition. MIDUS includes data on health, employment, human capital, and labor market participation with rich retrospective data on family background.

Furthermore, I transform the wage data which are arranged on a categorical scale, to a continuous measure of income by using the midpoint values of the respective category. A strength of MIDUS is that income categories are spaced unevenly with more categories for lower levels of income, thereby improving precision of the income estimates. For the lowest income category, I use $1/3^{rd}$ the lowest value of income, and for the highest category of income I use 3/2 the maximum value of income. I use the CPI from BLS to deflate earnings from all three of the waves of MIDUS to constant 2006 dollars. Alternative transformations for top coding and bottom coding wage do not influence the results in a meaningful way. I then transform the earnings into log earnings. For education, I use an indicator variable for college attendance. Beyond earnings, measures of job difficulty on physical and mental health are available on a five-point Likert scale. Labor supply outcomes are based on the labor market participation during the survey year.

The descriptive statistics are shown in table 2 for both the nationally representative sample and by family subgroups (twins and siblings). Notable differences are visible for outcomes such earnings as the sibling sample tends to have higher earnings than the full sample. Nevertheless, the sibling and twin sample appear relatively similar on most characteristics to the full sample. Given the longitudinal component of MIDUS, the average age of the analysis is

about 48 years. About a fifth of the sample currently smokes with nearly half of the sample having smoked at one point in their lives.

Figure 2 shows the distribution of ages at smoking initiation. Most individuals began smoking in their teenage years with a trickle of individuals initiating as preteens a trickle who initiate during adulthood. In figure 3, I show the distribution of log earnings between smokers and non-smokers, and the figure reveals that smokers tend to earn less than non-smokers. To evaluate whether skills could possibly influence this gap, in table 2 I compare whether smokers and non-smokers are different on demographic and ability variables and noticeable differences are visible. Large gaps are visible in age, as smokers tend to be younger than non-smokers and more male. In addition to the demographic differences, smokers tend to have a fairly large difference in completed schooling with approximately one fewer year of schooling. A notable exception occurs on weight, as smokers tend to be the thinner than non-smokers.

The MIDUS sampling for twins and siblings involved a "snowballing" component. During selection for the representative sample, individuals were asked if they had a twin or a sibling, which led to the creation of the twin and sibling subsamples. As a result, the family subsample should be similar to the nationally representative sample. I formally assess whether the twin subsample is similar to the MIDUS representative sample, by comparing outcomes of family background variables by twins and singletons in table 3. I find the twin sample similar to the representative sample on all variables except for father's high school completion. Although MIDUS does not contain any information on maternal age, the stylized facts on twinning have established that older women are more likely to have twins in addition to certain ethnicities such as the Yoruba of Nigeria. The interpretability of twin estimates as average treatment effects (ATE) or local average treatment effects (LATE) has been discussed in the literature (Kohler,

Behrman, Schnittker, 2014) and I interpret estimates from the family fixed effects models as LATEs.

1.4 Estimation Strategy

1.4.1 Full Sample

To estimate the effect of smoking on earnings, I start with equation 3 as the baseline specification. In equations 3-5, *E* represents the dependent variable for individual *i* in family *s* at time *t* and includes earnings (in adjusted 2006 dollars), employment, and job characteristics. The key parameter of interest is β the coefficient for whether an individual is a smoker. I also use an alternative measure of smoking such as whether the respondent was ever a smoker or a former smoker. The use of former smokers allows me to compare in the pooled cross section whether individuals that have smoked before the survey experience any earnings penalties compared to never smokers even though they no longer smoker. In all specifications I include survey year fixed effects and a vector of *X* that includes covariates for age, age squared whether the respondent is non-white. The use of log earnings parametrizes the coefficient on smoking or the smoking penalty as a percent of earnings. I contrast the baseline specification with the preferred cross section specification that controls for schooling in addition to demographic characteristics. Lastly, I cluster all standard errors to reflect repeated person level observations and to address heteroscedasticity and autocorrelation over time.

$$(3) \quad E_{it}=\alpha+\tau_t+\beta S_{it}+\gamma X_{it}+\varepsilon_{it}$$

The baseline specification given by equation 3 compares smokers to non-smokers, and former smokers to never smokers. Since MIDUS has a longitudinal component, it enables me to estimate an individual fixed effects model over the pooled person-year data. The advantage of the fixed effect approach is that it addresses the concern of unobserved time invariant

characteristics that are omitted. Estimates from the fixed effects model compare within person changes on smoking status and its effect on earnings. The individual fixed effect model shown in equation 4 provides some evidence on the effect of smoking on the earnings of smokers by examining how earnings change for smokers who switch to non-smoking. In the study sample more individuals quit smoking than individuals report starting smoking as would be expected. Since a non-negligible number of individuals report transitioning from non-smoker status to smoker, I re-estimate the fixed effects model comparing former smokers to never smokers as an alternative measure of the effect of quitting smoking on earnings. For all regressions involving log earnings as a dependent variable with a limited dependent variable as a regressor, I use Kennedy's approach (1981) to approximate the effects of smoking on earnings.

$$(4) \quad E_{it}=\alpha_i + \tau_t + \beta S_{it} + \gamma X_{it} + \varepsilon_{it}$$

1.4.2 Family Design: Twins and Siblings

Earlier figure 1 revealed that smoking initiation tends to occur when individuals are young. Figure 3 presents detailed correlates on smoking behavior, namely why people initiate and continue to smoke. The list contains main factors ranging from family attitudes, peer pressure, sociodemographic factors, personality/social skills, stress, availability (Center for Substance Abuse, 1997). Because individuals raised in the same family share similar environments and genetics that influence the production of hard skills like ability, and soft skills such as personality, the use of within family models would mitigate the bias from traditional comparisons of smokers to non-smokers. Consequently, the use of within family variation addresses these concerns because unobservable attributes are smaller within families than outside of families and thereby producing improved estimates over the OLS estimates (Card, 2001). The correlates of why people smoke are consistent with prominent neoclassical and behavioral

theories of smoking behavior such as myopia, rational addiction, and regret (Becker and Murphy, 1988; Chaloupka, 1991; Thaler and Sunstein, 1998).

The sibling and twin analysis is motivated through the fact that they share genetics and common environments, therefore using within family variation might address some of the endogeneity concerns that are driven by shared environments. Twins are a "natural" experiment (Rosenzweig and Wolpin, 2000) and using twins and siblings has a long past in economic research. Monozygotic or identical twins share the same genetic makeup whereas dizygotic or fraternal twins share half the genetic makeup and essentially are siblings born at the same time. Analysis of the twin siblings imposes stricter restrictions compared to singleton siblings because twins are more similar than non-twin siblings as they are raised together and share a greater proportion of genetic material. More specifically, the twin design reduces threats of potential confounders from the sibling design such as differences in spacing, birth order, family size, in addition to differential parental endowments because twins have parents that are aged the same. Most often economists have used twins to estimate the monetary returns to education (Ashenfelter and Krueger, 1994; Miller, 1995; Ashenfelter and Rouse, 1998).

To causally estimate the effect of smoking on earnings, I employ a family design using a sample of siblings and twins. In the family design, identification of the smoking parameter comes from within families, by comparing the earnings of a sibling who smokes to the earnings of a sibling who does not smoke. Similarly, in the analysis for twins the coefficient on smoking is generated by comparing the earnings of a twin sibling that smokes to a twin sibling that does not smoke. In baseline specification for the family design given by equation 5, I replace the individual fixed effects with family fixed effects and now the identification arises from comparing the effect of smoking on earnings within families. Alternatively, I also estimate the

family design models controlling for education to examine how schooling differences influence the effect of smoking on earnings.

$$(5) \quad E_{ift} = \alpha_f + \beta S_{ift} + \gamma X_{ift} + \varepsilon_{ift}$$

Two important concerns arise with identifying the effect of smoking on earnings using within family variation. The first concern arises with the disparate smoking decisions within sibling sets. The same unobserved factors that induce people to smoke in the cross section of the representative sample might also influence the disparate smoking decisions within siblings. Under this scenario when the same unobservable factors influence smoking decision in both siblings and the full sample, then provided that twins and siblings are more similar to each other than the representative sample the bias from within family models will be smaller than the OLS cross sections from the full sample. Under these plausible conditions, then the within family estimates remain biased up but provide insight by bounding OLS estimates on the effect of smoking on earnings. Secondly, the within family estimates are likely to exacerbate measurement error in response on smoking status and this would introduce a downward bias and attenuate the coefficient on the earnings penalty.

1.5 Results

1.5.1 Main Results

Table 5 presents results from the full sample. The first column of panel A compares the earnings of smokers to non-smokers without any controls for schooling and it shows that the effect of smoking is an economically and statistically significant reduction of earnings by twenty-four percent. In the second column, by controlling for ability the earnings penalty declines to a shade under seventeen percent, approximately a decline of 25% percent. The large sensitivity of the smoking coefficient confirms differential levels of productivity and possibly

latent ability between smokers and non-smokers that is captured by controlling for schooling. Next, in columns 3 and 4 I introduce individual fixed effects to examine the effect of smoking status on earnings. Both columns reveal a statistically indistinguishable effect of changes in smoking status on earnings. Since a small portion of the sample switches smoking status between three waves and the reductions in sample size for later waves, it is likely that the study is underpowered to detect the within person effects of changing smoking status on earnings.

In Panel B, I compare individuals that are former smokers to never smokers. In column 1 without controls for schooling, former smokers earn approximately six percent less than individuals that are never smokers. Upon inclusion of controls for schooling, the earnings of former smokers are statistically indistinguishable from the earnings of never smokers which is consistent with the general pattern on the earnings of former smokers. About 2% of the sample returns to smoking regularly between surveys and as result ever smokers compares the change in quitting only on earnings. Similar to the earlier specification that looks at within individuals who change smoking status, the findings show no effect of being a former smoker on earnings. Likewise, in the analyses comparing smokers to non-smokers, I find statistically indistinguishable effects of smoking on earnings in the individual fixed effects specifications.

After comparing across the full sample, I present the main findings of this paper employing family fixed effects in table 6. Beginning with column 1 of Panel A where I examine the effects of smoking on earnings across the cross section, I find large reductions in earnings of around twenty-eight percent, and controlling for schooling in column 2 reduces the earnings penalty to around twenty percent. In all sibling models with and without family fixed effects, the effect of smoking in the sibling sample is large, statistically significant, and negative. In columns 3 and 4 I present estimates from the causal model employing sibling fixed effects. First in

column 3, I find the smokers earnings penalty to be around sixteen percent and controlling for schooling in column 4 does not affect the size or significance of the coefficient. The similar effect sizes even after controlling for schooling suggests that family fixed effects appear to handle differences in the coefficient arising from one measure of observed ability fairly well.

In panel B of table 6, I examine the effect of smoking on earnings both across and within twin pairs. Across twin comparisons beginning with column 1 show a large negative effect on earnings of around twenty-eight percent which is significant at the conventional levels of significance. Column 2 which includes a control for schooling which reduces the coefficients in a substantial and significant manner to about eighteen percent. The causal models with twin fixed effects in column 3 of shows a significant reduction in earnings of nineteen percent. Similar to the siblings, controlling for schooling does not influence the coefficient for the smokers' earnings penalty as it remains around eighteen percent. In both sibling and twin samples, smoking the past has a statistically indistinguishable effect on earnings.

The main takeaway from tables 5 and 6 is that the earnings penalty for smokers is large and consists of the aggregated earnings loss that incorporates the many factors from table 1. One possible explanation for the large magnitude of the impacts is that this analysis occurs mostly during middle age when workers have reached the flat portion of the earnings profile (Bhuller et. al, 2014) and this would influence the size of the earnings penalty. The secondary takeaway pertains to the econometric analysis. The family fixed effects models appear robust to the controls for schooling and might also be successful in controlling for other unobserved variable such as character skills along with tastes and preferences. As would be expected, the coefficient is smaller as comparisons move from the full sample to the more similar twin sample.

Furthermore, the earnings penalty appears to dissipate for former smokers both in the full sample and the family sample.

1.5.2 Understanding the Wage Differences between Smokers and Non-Smokers

A vast literature has shown that smokers earn less than non-smokers, yet these studies have been unable to disentangle the earnings penalty into components of selection, productivity, and health care costs. Even after addressing the ability differences which influence the decision to smoke, which I describe as selection, smokers and their habits might directly reduce productive as a result of nicotine addiction which produces physiological withdrawal either through taking smoking breaks along with the physical manifestations of addiction. Other evidence suggests that smokers tend to miss more days of work and may also suffer from more expensive illnesses (Lundborg, 2010). As a result, separating the effect of higher health expenses from productivity changes poses a challenge beyond addressing the selection problem. Since twins and siblings abate selection concerns then the estimates from Table 7, which compares across ESHI status, crudely functions as a test of whether worker productivity or health costs cause the reduction in earnings of smokers.

To distinguish between the addiction-productivity versus the health insurance hypothesis, I separate the sample into individuals with ESHI versus individuals without ESHI. Estimates from these models separate the effect of ESHI on earnings because both smokers with ESHI and without ESHI should be afflicted with addiction related productivity declines under an assumption that firms do not discriminate in hiring smokers. Support for plausibility of the anti-discrimination assumptions arises in the fact that insurance is offered at the firm level and not at the individual worker level. Furthermore, comparisons between former smokers and never smokers, conditional on ability, provides auxiliary evidence that indeed current health costs and

productivity influence earnings and not past health costs or addiction influence the differences in earnings.

For individuals without ESHI, I present findings in Panel A of table 7. In all specifications for the full sample and within siblings and twins, I find the effect of smoking on earnings to be largely negative but imprecisely estimated. The effect size varies from around seven percent to around twelve percent. It's also possible that smoking still incorporates a degree of productivity such as smoking breaks and time off in terms of illness and sick days, but that these negative effects of smoking such as taking smoke breaks as likely to be small and in this study not detected at the conventional levels of significance. Two notable difficulties arise in this analysis for heterogeneity. First, it suffers from the problem of limited sample size. Second, the conceptualization of earnings with income bins introduces measurement error for the dependent variable and thereby inflates standard errors for the effect of smoking.

On the other hand, estimates for smoking on individuals that do have ESHI are fairly large in magnitude and statistically distinguishable from zero. In column 1 without controls for schooling, ESHI smokers earn nearly twenty-five percent less than non-smokers, controlling for schooling reduces this gap to around eighteen percent of earnings. In columns 3 and 4 for siblings, the effects are large and negative at twenty-one and eighteen percent, respectively. For twins in columns 5 and 6, the effects are twenty and eighteen percent, respectively. Although the earnings reduction or smokers with ESHI is large and statistically different from zero, because of large confidence intervals these effects are statistically indistinguishable from the estimates for smokers without ESHI.

To provide a pattern of suggestive evidence in support that health care contributes to the earnings penalty for smokers, I examine for heterogeneity with correlates of disproportionate

health care usage. I separate the sample by age and gender to examine for heterogeneity on the dimension of health care costs. Although a priori it is unknown whether the effect of nicotine addiction varies with age and gender, it is however known that health insurance costs do vary considerably on the dimensions of age and gender. For age, I divide the sample into two groups based on the midpoint age of 44. Results in table 8 are shown for the old and young groups.

Evaluating the smokers' wage penalty by differences in age shows that in the full sample, older smokers are likely to have higher penalties than younger smokers. In fact, as column 2 of Panels A and B show, the earnings penalty for older smokers is approximately twice as a large than the smoking penalty for young smokers. For the sibling sample, the earnings penalty is about sixteen percent and is statistically similar in both the young and old sample. The most perplexing findings are for older twins. Unlike young twin estimates which are smaller than estimates from the full sample, the estimates for the earnings penalty is substantially larger in the twin sibling subsample. It is possible that twin specific idiosyncratic factors contribute to these estimates. For example, Bhai and Horoi (2015) show that twins tend to have higher rates of disabilities which might influence differences in health outcomes by smoking status. It should also be noted that with the reduced sample, confidence intervals for the twin estimates are large.

Since variation in healthcare costs could arise by gender, I also analyze the results separately by gender in table 9 to investigate potential heterogeneity. In panel A I examine the outcomes for men. The full sample with controls for schooling in column 2 produces effect sizes that are similar to the impacts of smoking estimated from sibling fixed effects models both with and without controls for schooling at around twenty percent. Twin fixed effects estimates are also similar in magnitude at seventeen percent, respectively, but they are more imprecisely estimated. Panel B presents the outcomes for women and the pattern of estimates follows that of

men. All columns of estimates for the effect of smoking on earnings are negative for women smokers in the full sample and within twins and siblings. Estimates for the wage penalty range from fourteen to twenty percent, and again the within family models produce larger estimates than the full sample. Although the wage penalty for women who smoke appears to be smaller than men who smoke, because of the large confidence intervals they are unlikely to be statistically different. A similar caveat applies to the causal comparisons within family and the cross sectional comparisons; as a result of the large standard errors the point estimates are statistically similar.

1.5.3 Comparing Smokers and Non-Smokers on Job Quality and Health Investments

In table 10 I check if labor force participation varies by smoking status[2]. The negative consequences of smoking for health are known and might cause a change in labor force participation because of non-random attrition from the labor market. Besides health reasons, differential labor force participation might occur because of unobserved differences that drive labor force participation. I examine the effect of smoking on full time employment in Panel A. Consistently across all samples including the full sample and the within family sample of twins and siblings, it is evidence that smoking does not affect labor force participation. In panel B I examine the effect of smoking on part time employment. I find a small negative effect on part time labor force participation for smokers, although the effect is statistically significant the magnitude of the coefficient on part time labor force participation is small at around one

[2] I also examine whether hours worked in a week vary by smoking status, and I find a pattern of similar results as for labor market participation.

percentage point. There are no differences in either full time or part time labor force participation in the family sample. The main takeaway from this table indicates that it is unlikely that labor force participation influences the margin on the incidence of smoking on ability.

In table 11, I use measures of job related physical and mental health questions to proxy for job quality and rigor of work. I assess whether smokers and non-smokers work in similar type of jobs. For the mental health toll of a job in panel A, I find modest negative effect of seven percentage point more mentally difficult job in column 1 for the full sample which is reduced by thirty percent upon inclusion of controls for schooling and is no longer significant at the conventional levels. The cross sectional comparisons show that work attributes for a job's toll on mental health are not different between smokers and non-smokers. In the next column I begin the family analysis by looking at within families. I find that twins and siblings are similar.

After evaluating job quality differences for mental health, I examine whether smokers and non-smokers have different types of jobs on measures of self-reported physical difficulty or the job toll on physical health. Interestingly, there is a consistent large distinction for physical toll of jobs for smokers versus non-smokers, however, there are no differences between former smokers versus never smokers. As a result, the large negative results on physical health pose a challenge and can be interpreted in three possible ways: (1) firms adjust on the quality of job thereby smokers have physically tougher positions; (2) smokers because of skill differences end up in physically tougher positions; (3) smokers perceive physical stress differently than non-smokers.

In table 12, I assess the hypothesis that one explanation for the differences in the earnings of smokers and non-smokers arises from differential rates of investments in health capital. I use measures of body weight as proxy for health investments. First, in Panel A: I examine the

relationship between smoking status and whether the respondent is obese. In all specifications that compare across the full sample and within siblings and twins, I find large statistically significant reductions in obesity rates among smokers. Former smokers are not statistically distinguishable from non-smokers except in column 1 where I exclude controls for schooling.

Next in panel B I examine the relationship between smoking status and whether the respondent is overweight. The pattern of results follows the findings in panel A. Smokers are consistently less likely to be overweight than non-smokers, and the effect sizes are similar across the full sample and within siblings and twins. All the effects are statistically different from zero at the conventional levels. No effects are seen for former smokers. The stylized facts of smoking acknowledge that smoking does have an effect on weight, and these findings tend to confirm that claim as smokers tend to invest positively in health on measures of weight compared to non-smokers. Such a result suggests that weight related health investments are unlikely to drive the earnings penalty for smokers.

1.6 Conclusion

Using data from multiple waves of the National Study of Midlife Development, this paper demonstrates a large earnings gap between smokers and non-smokers. My preferred estimates from causal twin and sibling fixed effects model show roughly the same earnings effects of smoking as estimates from the full sample at roughly a sixteen to eighteen percent reduction in earnings. Attempts to identify the pathways that influence the gap reveal that it is statistically non-existent in jobs that do not include employer supplied health insurance (ESHI), but much larger for smokers with ESHI. Analysis by subgroups reveals the earnings gap for smokers varies with age and gender, and disappears for former smokers.

The reduced earnings for smokers driven by ESHI present an interesting implication both for models of worker compensation and health insurance. If transactions costs are low, firms can adjust the price of health insurance to reflect differential costs of providing health care to smokers. Yet, transactions costs are high and the practical provision of health care benefits, however, precludes offering health insurance based on each person's health care costs. The amount of variability for the price of non-monetary benefits such as health insurance does not vary with health status like smoking. Even though firms cannot fully differentiate premiums on smoking, however, they can differentiate how they compensate employees. That implies that firms adjust compensation on overall worker thereby adjusting for frictions from the insurance market on the wage market. Thus, firms not only adjust compensation on offering fringe benefits but also on the disproportionate use of fringe benefits.

In contrast, standard economic models for insurance (Rothschild and Stiglitz, 1976) have taken the inability to charge higher premiums and assumed an inability for firms to adjust wages for more expensive individuals such as smokers. Under such a framework, insurance markets might function inefficiently with pooling equilibria because healthier workers or non-smokers end up paying too much and under insure whereas unhealthier workers such as smokers over insure and end up paying too little. Or the entire market might cease to exist with market failure. Differential wages based on healthy activities mitigate inefficiencies that arise from asymmetric information in health care markets, because workers that incur higher health costs are likely to receive lower wages to account for misallocation in the insurance markets.

Lastly, the findings of this paper indicate that the incidence of smoking does fall on smokers to an extent. Smokers appear to pay for smoking behavior with reduced earnings. Nevertheless, it remains a possibility that the shifting of health care costs onto smokers through

lower wages is not entirely complete, and the existence of negative spillovers on non-smokers might still occur. Overall, it is evident that smokers do bear some costs on the labor market for smoking.

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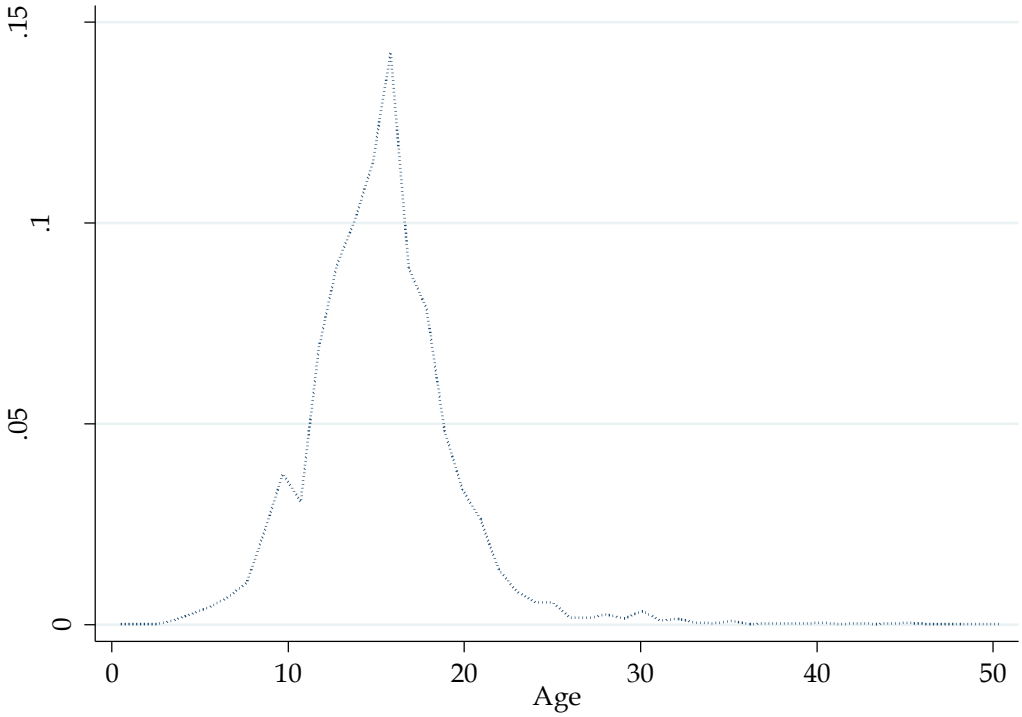
1.8 Cited Literature

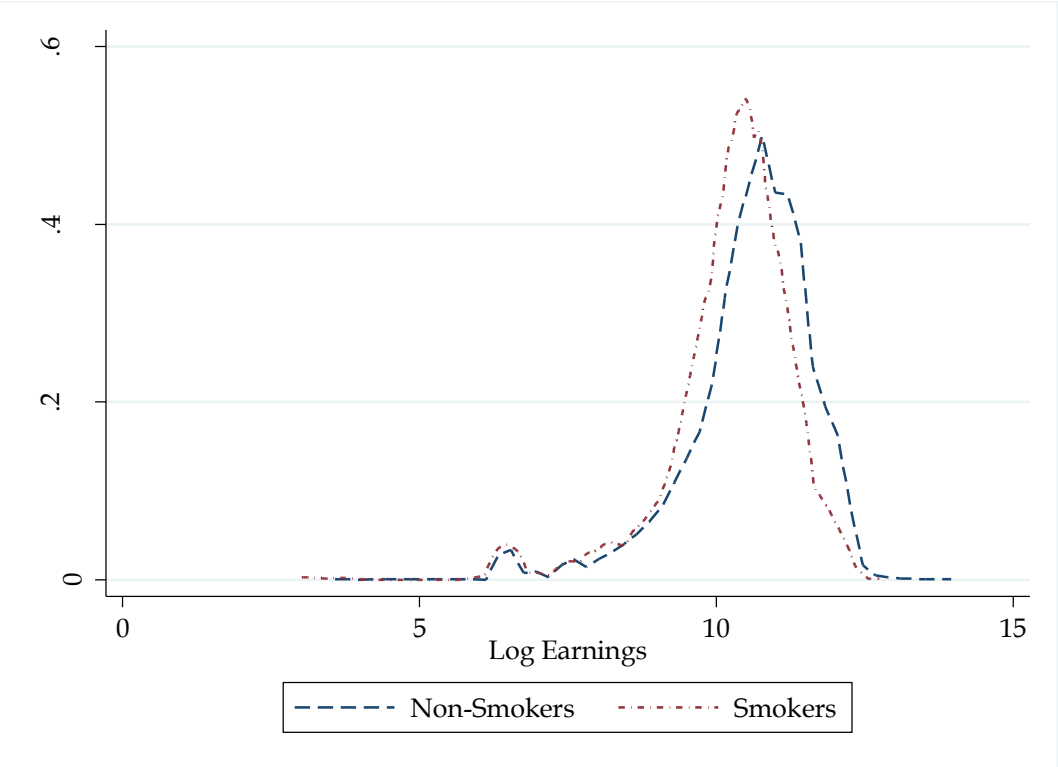
1.81. Figures

Figure 1
When do Smokers Initiate?



Source: National Study of Midlife Development (1996)

Figure 2
Log Earnings of Smokers versus Non-Smokers



Source: National Study of Midlife Development (1996, 2006, and 2014).

Figure 3
A Taxonomy of Why People Initiate Smoking

Sociodemographic	Environmental	Behavioral	Personal
Low SES	*Interpersonal Factors*	Low Achievement	Denial
Low Parental Education	Availability of Tobacco	Use of Alcohol or Drugs	Coping Mechanism
Single Parent Household	Parental Tobacco use	Risk Taking	Positive Utility
Development Challenges	Sibling tobacco use	Strong Peer Relations	Low Self-Esteem
Male	Peer Tobacco Use	Poor Health Investments	Negative Self-Image
Hispanic or Black	Anti-social activities	Weak Refusal Skills	Low Self-Confidence
	Percieved Factors	Stress	Lack of Self-Control
	Expectations of Tobacco use		Low well-being
	Social Support		
	Parental Tolerance		

Adapted from Center for Substance Abuse Prevention. Each column contains a list of factors that influence smoking initiation in youth.

1.8.2 **Tables**

Table 1
The Annual Costs of a Smoker (in 2010 dollars)

	Best Estimate	High Range	Low Range
Excess Absenteeism	$517	576	179
Presenteeism	462	1848	462
Smoking Breaks	3077	4103	1641
Excess Health Care Costs	2056	3598	899
Pension Benefits	-296	0	-296
Total Costs	5816	10125	2885

Source: Berman, Crane, Seiber, and Munur (2014). The table presents the differences in costs of employing a smoking employee versus a non-smoking employee. Presenteeism refers to the costs arising from nicotine withdrawal.

Table 2
Descriptive Statistics for MIDUS

	Random Sample		Sibling		Twin		All	
	Mean	STD	Mean	STD	Mean	STD	Mean	STD
Age	45.580	10.526	47.329	9.600	45.227	10.010	45.669	10.298
Female	0.499	0.500	0.524	0.500	0.534	0.499	0.505	0.500
Employer Insurance (ESHI)	0.580	0.494	0.570	0.495	0.582	0.493	0.583	0.493
Schooling	14.244	2.478	14.717	2.370	14.100	2.402	14.351	2.457
High School	0.942	0.233	0.973	0.162	0.938	0.240	0.949	0.221
Some College	0.669	0.470	0.758	0.428	0.651	0.477	0.687	0.464
College Grad	0.409	0.492	0.477	0.500	0.388	0.487	0.427	0.495
Non White	0.128	0.334	0.052	0.223	0.074	0.262	0.102	0.303
Earnings	51830	43073	59041	46269	52508	42374	54510	44322
Log Earnings	10.442	1.080	10.575	1.107	10.468	1.075	10.500	1.076
Smoke	0.219	0.413	0.186	0.389	0.208	0.406	0.207	0.405
Ever Smoke	0.517	0.500	0.448	0.498	0.457	0.498	0.490	0.500

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Standard deviations are next to the means of the variables in parenthesis.

Table 3
Testing for Sample Selection: Singletons vs. Twins

Question	Non-Twins	Twins	Difference	P-Value
Mother's Education				
Has less than High School	0.360	0.343	-0.017	0.29
Graduated High School	0.402	0.403	0.009	0.59
Attended Some College	0.129	0.130	0.009	0.45
College Graduate	0.108	0.108	0.0002	0.98
Schooling (Years)	11.20	11.44	0.242**	0.03
Father's Education				
Has less than High School	0.409	0.408	0.007	0.68
Graduated High School	0.325	0.293	-0.044**	0.04
Attended Some College	0.089	0.100	-0.010	0.28
College Graduate	0.177	0.191	0.014	0.33
Schooling (Years)	11.04	11.11	-0.076	0.60

Source: National Study of Midlife Development (1996). Notes: * $p<0.10$, ** $p<0.05$, *** $p<0.01$

Table 4
Are Smokers different than Non-Smokers?

	Non-Smoker		Smoker	
	Mean	STD	Mean	STD
Age	48.11	12.10	45.24	10.91
Female	0.50	0.50	0.51	0.50
Employer Insurance	0.57	0.50	0.53	0.50
Schooling	14.60	2.46	13.19	2.24
High School	0.96	0.20	0.89	0.32
Some College	0.72	0.45	0.52	0.50
College Grad	0.48	0.50	0.21	0.41
Non White	0.10	0.30	0.10	0.31
Earnings	55221	45972	41744	35082
Log Earnings	10.47	1.14	10.24	1.05

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Standard deviations are next to the means of the variables in parenthesis.

Table 5
The Earnings Impact of Smoking for the Full Sample

	Full Sample			
	1	2	3	4
Smoke	-0.235***	-0.168***	0.063	0.064
	(0.022)	(0.024)	(0.089)	(0.090)
Former Smoker	-0.061**	-0.019	-0.041	-0.041
	(0.025)	(0.025)	(0.072)	(0.072)
Covariates				
Education	Yes	No	Yes	Yes
Individual	No	Yes	No	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All individuals are under age 66, and all regressions include controls for race and age. The first row compares smokers to non-smokers, and the second row compares ever smokers, or former smokers to non-smokers. * p<0.10, ** p<0.05, *** p<0.01

Table 6
The Earnings Impact of Smoking for the Family Sample

	Family Level			
	1	2	3	4
Panel A: Siblings				
Smoke	-0.270***	-0.199***	-0.165**	-0.156**
	(0.033)	(0.037)	(0.072)	(0.073)
Former Smoker	-0.023	-0.024	-0.079	-0.062
	(0.038)	(0.038)	(0.101)	(0.103)
Panel B: Twins				
Smoke	-0.287***	-0.182***	-0.195**	-0.163**
	(0.038)	(0.045)	(0.078)	(0.080)
Former Smoker	0.014	0.045	-0.120	-0.112
	(0.046)	(0.047)	(0.107)	(0.112)
Covariates				
Education	No	Yes	No	Yes
Family	No	No	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: The first two columns compare across twins and the last two columns measure smoking between twin pairs. Clustered standard errors are in parenthesis. All individuals are under age 66, and all regressions include controls for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 7
The Earnings Impact of Smoking by Employer-Supplied Health Insurance

	ESHI					
	1	2	3	4	5	6
Panel A: No ESI	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0167***	-0.069	-0.112	-0.084	-0.117	-0.126
	(0.039)	(0.045)	(0.088)	(0.089)	(0.095)	(0.091)
Panel B: ESI						
Smoke	-0.255***	-0.154***	-0.211***	-0.187**	-0.208**	-0.176**
	(0.021)	(0.024)	(0.075)	(0.076)	(0.084)	(0.086)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 8
Examining for Variation by Age

	Age					
	1	2	3	4	5	6
Panel A: Old	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0.273***	-0.218***	-0.187***	-0.159***	-0.325***	-0.320***
	(0.031)	(0.033)	(0.075)	(0.078)	(0.095)	(0.095)
Panel B: Young	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0.192***	-0.119***	-0.154*	-0.150*	-0.096	-0.090
	(0.030)	(0.031)	(0.081)	(0.081)	(0.109)	(0.110)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 9
Examining for Variation by Gender

	Gender					
	1	2	3	4	5	6
Panel A: Men	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0.257***	-0.203***	-0.209***	-0.200***	-0.176	-0.162
	(0.026)	(0.031)	(0.074)	(0.075)	(0.124)	(0.123)
Panel B: Women	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0.219***	-0.140***	-0.198**	-0.188**	-0.201	-0.204
	(0.036)	(0.036)	(0.076)	(0.075)	(0.141)	(0.142)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 10
Does Smoking Affect Employment?

	Labor Supply					
	1	2	3	4	5	6
Panel A: Full Time	*Full Sample*		*Siblings*		*Twins*	
Smoker	-0.001	0.018	0.060	0.059	0.054	0.052
	(0.011)	(0.011)	(0.045)	(0.045)	(0.056)	(0.056)
Former Smoker	-0.011	-0.004	-0.033	-0.032	-0.035	-0.034
	(0.010)	(0.010)	(0.073)	(0.074)	(0.087)	(0.088)
Panel B: Part Time	*Full Sample*		*Siblings*		*Twins*	
Former Smoker	-0.021**	-0.017*	-0.008	-0.008	0.006	0.007
	(0.007)	(0.007)	(0.031)	(0.031)	(0.038)	(0.038)
Former Smoker	-0.004	-0.001	0.054	0.055	0.040	0.041
	(0.007)	(0.007)	(0.056)	(0.056)	(0.056)	(0.056)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 11
Does Smoking Affect Other Job Characteristics?

	Job Characteristics					
	1	2	3	4	5	6
Panel A: Mental Health	*Full Sample*		*Siblings*		*Twins*	
Smoke	0.073**	0.053	-0.179	-0.187	-0.281	-0.294
	(0.033)	(0.034)	(0.182)	(0.182)	(0.237)	(0.238)
Former Smoker	0.058	0.049	-0.425	-0.425	-0.408	-0.399
	(0.032)	(0.032)	(0.289)	(0.291)	(0.369)	(0.370)
Panel B: Physical Health						
Smoke	-0.192***	-0.148***	-.530**	-0.634*	-0.559**	-0.680*
	(0.031)	(0.032)	(0.211)	(0.334)	(0.268)	(0.393)
Former Smoker	0.004	0.049	0.015	0.021	-0.021	-0.011
	(0.028)	(0.028)	(0.344)	(0.342)	(0.423)	(0.420)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Table 12
Do Smokers Reduce their Health Investments?

	Weight					
	1	2	3	4	5	6
Panel A: Obesity	*Full Sample*		*Siblings*		*Twins*	
Smoke	-0.055***	-0.074***	-0.099***	-0.102***	-0.104**	-0.105**
	(0.013)	(0.013)	(0.035)	(0.036)	(0.044)	(0.044)
Former Smoker	0.030**	0.018	0.027	0.023	0.024	0.021
	(0.013)	(0.013)	(0.044)	(0.044)	(0.049)	(0.049)
Panel B: Overweight						
Smoke	-0.079***	-0.097***	-0.097**	-0.099***	-0.126***	-0.127***
	(0.015)	(0.015)	(0.040)	(0.040)	(0.044)	(0.044)
Former Smoker	0.008	0.012	0.031	0.031	0.007	0.008
	(0.014)	(0.014)	(0.045)	(0.045)	(0.048)	(0.048)
Covariates						
Education	No	Yes	No	Yes	No	Yes
Family FE	No	No	Yes	Yes	Yes	Yes

Source: National Study of Midlife Development (1996, 2006, and 2014). Notes: Clustered standard errors are in parenthesis. All regressions include covariates for race and age. * p<0.10, ** p<0.05, *** p<0.01

Chapter 2

THE EFFECT OF CIGARETTE TAXES ON ASTHMA AND INEQUALITY

2.1 Introduction

Asthma by far remains the most common chronic disease affecting children in the United States (CDC, 2012). Examining the prevalence of asthma by both socioeconomic status and race reveals significant disparities (Case, Lubotsky, and Paxson, 2002; Condliffe and Link, 2002). Children from wealthier households have lower rates of asthma than their counterparts from poorer households. Racial comparisons show a similar pattern of disparities in asthma prevalence as white children have lower rates of asthma than their Hispanic or black counterparts. Although the etiology of asthma includes both genetic and environmental influences, genetic explanations are uninteresting from a policy perspective. Maternal smoking during pregnancy, an environmental explanation, has often been hypothesized as a causal factor and has been extensively studied since the early 1950s. The general consensus from the early research indicates that in-utero smoke exposure is linked to poor child health outcomes (Law, 1996). Yet, the consensus on early life smoke exposure has to be interpreted with caution because the early research emphasizes simple comparisons that lack a rigorous research design.

In order to measure the causal impact of maternal smoking during pregnancy on later outcomes, I use quasi-experimental variation from changes in state cigarette taxes during the in-utero period to examine child health outcomes such as asthma, severity of asthma, and health status. Using data from the National Survey of Children's Health (NCHS) I estimate reduced form intention to treat models employing a differences-in-differences research design. I find that

a $1 increase in cigarette taxes during the in-utero period measured in constant 2007 dollars reduces the prevalence of asthma by 1.7 percentage points off of a mean of nearly fourteen percent. Further analyzing the results by SES reveals that children from the poorest households drive the result since cigarette taxes substantially reduce the prevalence of asthma in low SES children by roughly around 3.5 percentage points, whereas the impacts for high SES children are a much smaller reduction by 1.1 percentage points. Estimating the effect of cigarette taxes separately based on race, on the other hand, produce more consistent reductions in prevalence as Hispanic and black children show a higher responsiveness to cigarette taxes at negative 2.4 and 1.8 percentage points than white children at a negative 1.6 percentage points, respectively. In the entire analysis, the effects of in-utero cigarette taxes on self-reported health status and severity of asthma are statistically indistinguishable from zero. A natural implication from the differential responses to cigarette taxes is that in a counterfactual world with an absence of cigarette tax hikes would have higher health disparities based on SES.

The analysis in this paper has several important implications for health and human capital policy. First, the dynamic production of health capital makes childhood health a salient policy issue because poor health in childhood persists into adulthood (Case, Fertig, and Paxson 2005; Smith, 1998). As a result, unhealthy children tend to become unhealthy adults. Explorations of the socioeconomic gradient illustrate a similar trajectory as poor children tend to be sicker than their non-poor counterparts with the gap in health widening with age (Currie, Shields, Price, 2004; Chen et. al., 2004). Health gradients based on socioeconomic status similar to the one in the U.S. are ubiquitous in the international context (Currie and Stabile, 2004; Farrell et al., 2008). Moreover, the analysis in this study between child health and cigarette smoking is also timely as smoking prevalence has increased in the developing world with rising incomes.

In addition to health capital, the effects of poor health can spillover and influence the production of human capital. Respiratory illnesses such as asthma along with gastrointestinal disorders are the main causes of absences from school (Currie, Hanushek, Kahn, and Rivkin, 2009). Besides causing children to miss school, sickness can also affect cognition and as a result of poor health sicker children may have a harder time learning in school (Currie, et. al, 2013). Children coming from low SES backgrounds already have lower rates of academic achievement and educational attainment and poor health has been offered as an explanation for differential educational outcomes. Therefore, policies addressing health outcomes such as asthma could also influence and reduce cognitive gaps both based on race and SES.

Lastly, this paper contributes to the burgeoning literature on the fetal origins hypothesis and the intergenerational transmission of success. Most studies on fetal origins have focused primarily on how negative in-utero shocks have lasting impacts through adulthood. This study, on the other hand, explores the effects of a positive shock during childhood, an intermediate phase in life before adulthood. Understanding the effects of shocks during childhood can be useful in designing optimal remediation policies during younger ages when the returns on investment to interventions are likely to be higher than in adulthood. By examining the role of parental health behaviors on the impact of children's health, this study also contributes to the recently developed literature on the intergenerational transmission of success. Parental investments in health have important implications for children's health especially if adverse shocks show a pattern of persistence. Overall, the findings of this paper link the public health and economic inequality literatures by demonstrating the link between early life smoke exposure and childhood asthma, while credibly identifying one causal factor contributing to the SES gradient for childhood asthma.

2.2 Conceptual Framework

2.2.1 The Production of Health

Since the seminal work of Barker (1986) theorizing the link between adverse conditions in-utero and later life outcomes such as heart disease, a large body of literature[3] has empirically explored and extended the Barker hypothesis, also commonly referred to as the fetal origins hypothesis, to pollution, diseases, health insurance, and other shocks. Notable studies have credibly demonstrated that in-utero shocks that restrict nutrition such as from famine (van den Berg et. Al, 2006; Chen and Zhou, 2007) and exposure to the Islamic fasting (Mazumdar, 2011) can have long lasting negative impacts on both health and human capital. Other extensions beyond nutritional deficits have examined disease such as the 1918 Spanish flu (Almond, 2006; Neelsen and Strattman, 2012), and shorter run impacts of air pollution (Coneus and Spiess, 2012) among others.

The dynamic production of health and human capital theorized by Becker (2007) and Heckman (2007) forms the conceptual framework underlying the current analysis. The conceptual model consists of two periods, early childhood and late childhood, and focuses solely on health capital and for simplicity ignores potential human capital effects. A common assumption in studies dealing with shocks to children is the absence of parental behavioral responses, i.e. that parents do not reinforce or compensate for early life adverse shocks. Cigarette prices work through changing parental investments in the prenatal period given by I_0^H and affect subsequent periods by affecting both the stock of health capital and investments in health capital. As equation 6 shows, cigarette taxes influence parental investments because smoking is a

[3] Almond and Currie (2011) and Currie and Almond (2003) provide an overview of the rich literature in economics on the "fetal origins hypothesis."

negative health investment for child *i* at time *0* which is the prenatal phase of life. Quitting smoking therefore serves as a positive investment because it reduces exposure to smoke, but also because consumption changes from reduction in spending on cigarettes to alternative goods may also serve as an investment in child health that may promote health development. Equation 7 shows that at birth the child's stock of health given by θ_1^H depends on both fixed genetic endowments of health and adjustable environmental prenatal investments shown by I_0^H.

$$I_0^H = f(t_0^H) \tag{6}$$

$$\theta_1^H = f(\theta_0^H, I_0^H) \tag{7}$$

$$I_1^H = f(\theta_1^H) \tag{8}$$

$$\theta_2^H = f(\theta_1^H, I_1^H) \tag{9}$$

Health in childhood given by θ_2^H in equation 9 follows a similar trajectory. Equation 8 shows that investments during early childhood, given by I_1^H depend on the child's stock of health in the preceding period or early childhood. The stock of health during later childhood denoted by θ_2^H depends on both the stock of health in early childhood and flows such as parental investments in health during early childhood. Thereby, cigarette taxes serve as an exogenous positive shock since they mitigate exposure to cigarette smoke or through changes in maternal consumption patterns as they substitute away from smoking. Chapter 1 also shows that it might work through parental income. Even though negative health shocks may occur during the prenatal period, because of the iterative nature in the production of health capital, the impact of the shocks extend to health capital in later childhood. Further, if there was another period for adulthood they would adversely affect the production of health capital in adulthood.

From the conceptual framework, the impact of changes in cigarette taxes during the in-utero period can be derived and decomposed into two parts as shown in equation 10: a change

from endowments and a change from parental investments. The first term represents self-productivity because the value of health capital depends on the value of health capital in the preceding period. By raising the stock of health capital in early childhood, the productivity of the stock of health capital in the second period is greater. The second term represents cross productivity and captures the change in the stock of health during late childhood that happens from changes in the investment profile as a result of cigarette taxes. Therefore, changes in in-utero investments alter both endowments and investments through the life of the child.

$$\frac{d\theta_2^H}{\partial t_0^H} = \frac{\partial \theta_2^H}{\partial \theta_1^H}\frac{\partial \theta_1^H}{\partial I_0^H}\frac{\partial I_0^H}{\partial t_0^H} + \frac{\partial \theta_2^H}{\partial I_1^H}\frac{\partial I_1^H}{\partial \theta_1^H}\frac{\partial \theta_1^H}{\partial I_0^H}\frac{\partial I_0^H}{\partial t_0^H} \quad (10)$$

2.2.2 The Empirical Evidence on Smoke Exposure

Early work exploring maternal smoking and child health employs simple research designs that compare health outcomes of children from mothers that smoke versus children from mothers that did not smoke and finds a strong negative association between maternal smoking and child health; notably, children whose mothers smoke tend to have lower birth weight and shorter gestational ages (Weitzman et al, 2002). The negative effects of exposure still appear even if the exposure arises from paternal smoking (Martinez, 1996). The causal pathway from cigarette smoke and child health is hypothesized to occur because smoke exposes the fetus to notable teratogens such as nicotine, lead, and cyanide, and raises the possibilities of miscarriages. Such exposure alters the amount of nutrients the fetus receives and teratogens can also hamper organ development during the second trimester. Nevertheless, it is possible that the effect of smoking on child health can be ambiguous if it influences the composition of births, more specifically, if the selection effect dominates (smoking causes miscarriages among weaker

fetuses) versus a scarring effect (fetuses are sicker). Overall, the empirical evidence overwhelmingly suggests that the scarring effect dominates.

Studies employing more advanced research designs to account for potential confounders find a similar relationship between cigarette taxes and infant health. Using exogenous variation in excise taxes on cigarettes by states, a series of papers have found a positive relationship between tax hikes and rising birth weight in children (Evans, 2000; Ringel and Evans 1995; Ringel and Evans 2000). They find that cigarette taxes have contributed to rising birth weight for children that are born to women who were induced to quit as cigarette taxes rose by nearly 200 to 400 grams (Lien and Evans, 2005).

Despite the broad evidence linking maternal smoking and health in infancy, limited research has rigorously examined outcomes after infancy. One notable study by David Simon (2015) employs a novel event study framework using a differences-in-differences research designed to examine the impact of in-utero exposure to cigarette smoke on child health. Using variation in excise taxes on cigarettes, he concludes increases in cigarette taxes led to reductions in hospitalizations for asthma, and increased school attendance, thereby providing indirect evidence in showing the relationship between early smoke exposure and childhood asthma. The study shows changes in infant health as a main causal pathway for the long lasting health effects in childhood.

2.3 Estimating Early Life Smoke Exposure

2.3.1 How do Smokers respond to Cigarette Taxes?

The role of cigarette taxes on smoking behavior has been extensively studied on both the extensive margin of whether individuals decide to smoke (Chaloupka, 1991; Levy et. al 2002; Chaloupka et al, 2010; Chaloupka et al, 2012), and the intensive margin of how much individuals

smoke (Mullahy, 1997; Tauras, 2006). The general findings of the literature are negative effects on both intensive and extensive margins with only disputes on the magnitudes of the effect sizes. Increases in cigarette taxes do induce behavioral responses such as substitution towards high tar cigarettes (Evans and Farrelly, 1998), and changes in inhalation patterns (Adda, 2006), but the net effect on smokers suggests that they are responsive to cigarette taxes and as prices of cigarettes increase due to taxes, then consumption of cigarettes falls.

Although elasticities for the whole population for smokers are interesting, the more pertinent elasticities for the purposes of this study are elasticities for maternal smoking. The research on maternal responsiveness to cigarette taxes has been less explored than general elasticities, however considerable evidence (Decicca and Smith, (2012); Gruber and Zinman (2000); Markowitz et. al, (2011)) finds that pregnant women are similar to other smokers and do respond to higher prices[4]. The elasticities for maternal smoking range from -.81 on the high end to about -.14 on the low end, with additional variation based on age, race, socioeconomic status, and by years of analyses. Similar work (Bharadwaj, Johnsen, and Loken, 2014) exploring how women respond to higher costs of smoking such as through smoking bans, reveals that reductions in smoking have substantive impacts on child health.

In figure four, I present the large declines in smoking prevalence in the United States. During this study's time period, smoking prevalence declines from approximately 32 percent to about 18 percent. Figure five shows that considerable variation in cigarette taxes exists during the study time period. For much of the 1980s cigarette tax hikes were infrequent, and the real value of the cigarette tax was much lower than during 1990s. Beginning in the late 1990s with

[4] Simon (2014) reviews the literature on cigarette taxes and maternal smoking in detail.

the Master Tobacco Settlement of 1998, large rises in cigarette taxes occur affecting nearly all states. Concurrently, large federal tax hikes on cigarette also occur during this time period.

2.3.2 The National Survey of Children's Health

The National Survey of Children's Health (NSCH) is a representative cross sectional study of children conducted in three waves in 2003, 2007, and 2012 with an overall large sample size of 290,725 over the three waves. The survey attempts to measure health of children from 0 to 17 years old. The NCSH includes large samples of both Hispanic and African-American children in addition to children from very poor SES households. Measures of children's health are obtained by asking a member of the household to respond to the survey via telephone. Mothers were most likely to provide answers on their child, as around 78% of the respondents were the mothers and 20% being fathers, with other relatives at roughly 2% respectively. The large sample provides enough statistical power to capture small differences in prevalence of diseases which is important since often the prevalence of most childhood illnesses are low.

The survey respondent provided demographic information to NCSH that described the race of the child, and the selected child's race was identified as white, black, mixed race, Hispanic, or other. The responding adult also provided information on age, measured in years, and the gender of the child. Asthma, behavior or conduct issues, hearing issues, and whether the household contains a smoker are binary variables. For asthmatic children, the responding parent also provides the severity of asthma on a three point Likert scale. The responses for health status are on a five point Likert scale.

The NSCH also contains household characteristics on income relative to poverty level. The survey does not directly indicate income, but a variable exists for that reveals family income based on the poverty level. Since calculations of poverty level include family size, the income

level available in the data reflects both income and family size. The poverty level data are characterized as deviations from the poverty level, and are calculated based on the relevant poverty benchmark per the survey year. I use this measure of the poverty level to create three categories. Low SES are households that are at or below 100% of the federal poverty level, mid SES are households that are between 100% and 300% above the poverty level, and high SES households are 300% above the poverty level.

Descriptive statistics from the NCSH, tax data, and the BLS are shown in table 13. Overall, the asthma prevalence rate in the full sample is roughly fourteen percent. Table 14 displays the dependent variables based on race and SES and shows the large differences in asthma prevalence by both SES and race. As the graphs in figures six and seven show, the incidence of asthma rises with the age of children. Such a profile is common with childhood diseases, as diagnoses of disease increase as children age. The race gap between blacks, Hispanics, and whites begins at birth and also grows as children age. The gap between Hispanics and whites is considerably smaller than the gap between blacks and whites. Measures of severity, as shown in figures eight and nine, reflects similar gaps as minority children have self-reported case of more severe asthma, but such differences do not change with age. Asthma severity remains at roughly the same level throughout childhood.

Similar differences in asthma prevalence and severity are visible by measures of socioeconomic status. Figures ten and eleven show the variation in both the prevalence and severity of asthma between low, middle, and high SES households. Analyzing the prevalence of asthma by socioeconomic status reveals a differential pattern. Children from the poorest households have substantial higher rates of asthma than the non-poor households. The prevalence of asthma for children from the middle SES group tends to be between the poor and

non-poor children. Severity of asthma shown in figure four for each group follows a consistent pattern. Unlike the prevalence of asthma, severity remains stable around the mean, with children from poorer households having a case of more severe asthma. Even though severity asthma shows variation by race and SES, the level of severity does not change with increasing age.

2.3.3 Estimation Strategy

In order to estimate the impact of exposure to smoke during the formative early years of childhood, in this study I exploit plausibly exogenous variation in state cigarette taxes during the prenatal period for the child. Equation 11 shows the differences-in-differences model with the identifying variation arising from changes in cigarette tax rates across states within years. In the regression model Y_{ist} represents the dependent variables such as asthma, severity of asthma, and health status. The model contains state fixed effects denoted by φ_s to account for time invariant state specific attributes, a full set of age dummies to account for the nonlinearity in disease prevalence with age denoted by α_i, and time fixed effects denoted by τ_t to account for temporal shocks that may influence asthma. I use the cigarette tax for the child's state during the year of the prenatal period. The vector X_{ist} contains demographic characteristics such as race and the gender of the key selected child to improve precision of the estimates, and local macroeconomic conditions[5] such as log of per capita income and local unemployment rates. The coefficient of interest is on the cigarette taxes, β, measured in 2007 dollars.

$$Y_{ist} = \alpha_s + \tau_t + Cigtax\beta + X_{ist} + \varepsilon \quad (11)$$

The responsiveness to cigarette taxes varies by different socioeconomic groups, so I further estimate the model based by SES groups and race to explore if cigarette excise taxes have

[5] State per-capita income begins in 1986, whereas the first cohort in this study is born in 1985.

a heterogeneous impact on health outcomes. Children from lower SES households are presumably more likely to be affected by cigarette taxes since the poor tend to reside in smaller households, are more sensitive to taxation, and have higher rates of parental smoking. Similarly, I estimate separately by race to assess the potential differential responses in children's health from changes in cigarette taxes. Such racial differences are likely to capture underlying socioeconomic differences. Since the unit of variation is at state by age by survey year, I also estimate models with state by year fixed effects to account for possible changes in trends in a non-parametric manner.

After estimating the DD empirical model, I use an Oaxaca-Blinder decomposition (Oaxaca, 1973; Blinder, 1974) to decompose the SES gap or the gradient and the race gap using cigarette taxes to reflect how much of the gap would occur in a counterfactual world without tobacco control policy such as cigarette taxes. The decomposition allows for a counterfactual comparison of the gap by showing that given the sets of endowments what the gap in health status would be by both SES and race had cigarette taxes not been implemented. Since the identifying variation in cigarette taxes arises from exogenous variation in cigarette tax increases then the decomposition has a causal interpretation.

Although the estimates in this paper are intention to treat (ITT), elasticities on maternal smoking can be used to scale up ITT coefficient to obtain a range of the treatment effect on the treated (TOT). Thus, the reduced form estimates of the intention to treat coefficient provides a lower bound for the impact of in-utero smoke exposure on child health. The effect sizes are considerably larger for children of women who were induced to quit as result of changes in cigarette taxes. Furthermore, data limitations which result in the omission of local state tax rates such as county and city tax rates that are common in certain metropolitan areas create concerns

of measurement error. Such taxes are a concern especially with rising rates of local taxes at both the county and city levels during the 1990s, and these omissions can produce downward biases provided the measurement error is classical.

The identifying assumptions for the difference-in-difference model is that trends in states that experience tax increases are the same as in states that do not experience tax changes for the relevant measures of health. I assess for the validity of DD assumptions by using pre-lag cigarette taxes to evaluate whether states that increase cigarette taxes versus states that do not increase cigarette taxes have similar time trends. The macroeconomic controls also address threats from changes in composition if macroeconomic conditions influence cigarette taxes and thereby influence fertility decisions, but come with a cost of reducing the sample size because of data limitations. Lastly, the inclusion of state by year fixed effect serves as an additional check to account for the potential threat of changes in state trends over time.

Besides the main identification assumption of parallel trends, another potential threat to identification would arise if cigarette tax increases are correlated with other attempts to improve children's health. Such contemporaneous investments are highly unlikely, because most revenues from cigarette taxes contribute directly to the state's general funds. Furthermore, to provide auxiliary support for the empirical framework, I also conduct placebo tests on other prevalent measures of children's health such as bone injuries and behavioral or conduct issues as diagnosed by a doctor. Since exposure to smoke can affect birth weight, choosing the diseases for the placebo tests that satisfy the exclusion restriction of the test is challenging.

The analysis in this study may suffer from concerns that could have downward bias on the coefficient of interest on cigarette taxes. First, because the in-utero period is measured with error, and since this error is much more likely to be classical, then estimates of the effect of in-

utero smoke exposure on child health are likely to be attenuated. The early years are seen as critical period with potentially differential impacts based on exposure during different time periods and it might be interesting to separate effects from in-utero to post birth exposure. As a result of the imprecise information on the timing of birth, I cannot separate the effect of cigarette taxes into a prenatal versus a postnatal period, and thus I interpret the coefficient to be the effect of cigarette taxes in early life[6].

2.4 The Effects of Early Life Smoke Cigarette Taxes on Childhood Outcomes

2.4.1 Associations between Smoking and Child Outcomes

Regression estimates in table 15 compare health and well-being outcomes of children from smoking and non-smoking households. As shown in column 1, for the whole sample, children from smoking households tend to perform poorer on many measures of well-being as they tend to have higher rates of asthma by two percentage points, a more severe version of self-reported asthma by 3 percentage points, worse health status by 3 percentage points than children from non-smoking households. All of these differences are statistically different from zero at the conventional levels of significance and the inclusion of macroeconomic controls and the state by year fixed effects in column 2 does not affect the coefficients.

Additionally, analyzing the association between contemporary household smoking and child outcomes based on race and SES in table 16 reveals mostly similar effects based on race but considerable heterogeneity based on SES. The association between household smoking and

[6] Cigarette tax increases happen infrequently to differentiate between effects in potentially in-utero period or variation that happens by each year. Even in specifications that include pre-birth and post-birth taxes, the identifying variation is arising from much fewer observations than in the whole sample.

childhood asthma is similar in significance and magnitude across racial categories at higher incidence of asthma by roughly 2 percentage points with somewhat higher rates for blacks than whites. However, notable differences arise in self-reported severity and health status of asthma. White children in smoking households, report worse severity of asthma by a significant 7 percentage points and worse health status by a statistically significant .183 percentage points. Whereas for black children, smoking households have a small significant effect of more severe asthma by 6 percentage points, and worse health status by .11 percentage points. Unlike blacks and whites, for Hispanics the association between household smoking and asthma severity is statistically indistinguishable from zero. Such an outcome is likely because asthma severity is conditional on having asthma and the sample sizes for Hispanics are very small. The inclusion of macroeconomic controls and state by year fixed effects does not have any substantive effect on the significance or magnitude of the associations between household smoking and child outcomes.

On the other hand, the analysis based on SES in table 16 shows a sharper gradient for each dependent variable. The relationship between household smoking and childhood asthma ranges from 3, 2, and 1.5 percentage points respectively for low, middle, and high SES households. A similar pattern exists for severity of asthma with worse rates for middle SES children than high SES children, however, the severity of asthma is not significantly worse for children from low SES smoking households. The heterogeneity for asthma rates can likely be explained by stylized facts of health economics which suggest that higher rates of smoking occur in lower SES households and that these smokers are more responsive to higher tax rates[7].

[7] Cigarette price elasticities of demand by income status suggest higher responsiveness by lower income groups. See Farrelly, Pechachek, and Chaloupka (2003); and Chaloupka and Warner (2000) for a review.

Alternatively, other work suggests that medication patterns vary based on SES which could contribute to the asthma gap (Zutshi, 2007). Health status, on the other hand, is one variable that has an inconsistent pattern based on SES. Children from low SES smoking households have better health status than their non-smoking counterparts, but the effects for middle SES and high SES households are consistent at 8 and 10 percentage points, respectively. The differences for all SES groups remain unaffected by the inclusion of controls for macroeconomic variables and state by year fixed effects.

2.4.2 Early Life Exposure and Measures of Asthma

The higher rate of asthma and the lower health status for children from households with smokers versus households with non-smokers raise concerns that there could exist some alternative attributes other than smoking itself that could be causal mechanisms. For example, smoking households may engage in different behaviors than non-smoking households which may affect childhood health development. Such differential investments may occur because smoking households tend to differ from smoking households on observable attributes such as education and SES. More importantly a bigger concern is unobservable factors that could be correlated with both household smoking and poor health outcomes for children that could produce a spurious link between smoking and child outcomes. The DD model overcomes the threat of a spurious relationship provided that the identification assumptions hold. Estimates from the DD model exploit exogenous variation to generate the reduced form intention to treat estimates of maternal smoking on childhood health.

The DD estimates are presented in table 17. A one-dollar increase in constant 2007 dollars produces an economically and statistically significant reduction in the prevalence of asthma by 1.7 percentage points. Inclusion of macroeconomic controls and state by year fixed

effects does not influence magnitude of the effect changing it to 1.5 percentage points and the effect remains highly statistically significant. Despite differences in self-reported severity of asthma that follows a pattern similar to the prevalence of asthma, the impact of cigarette taxes on the severity of asthma conditional on having asthma produces effect sizes that are small and statistically indistinguishable from zero, however. But the effect of early life taxes on health status is also fairly small and statistically indistinguishable from zero.

To provide auxiliary evidence in support of the differences in differences research design, I also use the tax rates before the prenatal period. The value of these estimates are that they provide some evidence that the result does not capture time trends toward improvements in children's health. I find the terms of the pre-trend regressions to have solid economically and statistically insignificant zeros across different specifications in columns three to six. The large magnitude on the coefficient on the early life tax suggests large important effects as would attest despite the loss of statistical significance because of the strong correlation between the pre-birth and early life tax.

The addition of a post birth tax in columns five and six also does not reach statistical significance, which suggests the largest effects on health are during the prenatal period. The coefficient in column 3 for the in-utero tax is smaller and still negative and significant at 1.2 percentage points. The addition of macroeconomic controls reduces both the size and significance. It is still possible that post-natal exposure has adverse health consequences, however the effect size is likely to be small and the current study would be underpowered to detect such an effect. Similar to column 3, without controls and the addition of pre-birth and post birth taxes, the coefficient on in-utero taxes is negative and significant at 1.4 standard deviations. All coefficients on asthma severity and health status are statistically indistinguishable from zero.

2.4.3 Does the Effect of Cigarette Taxes on Child Health vary based on Race and SES?

Estimating the impact of cigarette taxes in table 18 based on SES and race produces considerable heterogeneity in the effect on asthma[8]. First beginning by race, an increase in the cigarette tax by $1 in constant 2007 dollars produces a negative and statistically significant reduction in asthma prevalence for white children by 1.6 percentage points. Slightly larger effects are found for a dollar increase in tax increase for Hispanic children by about twenty percent. The largest estimates are for black children at nearly 2.4 percentage point reductions in asthma prevalence. The impact of in-utero cigarette taxes on measures of severity and health status are small and statistically insignificant for blacks and whites, however, small reductions in severity and worse health are found for Hispanic children but these reductions are not statistically distinguishable from zero. The coefficients by race remain largely unchanged in size however significance levels change for blacks and Hispanics. Despite these changes, the new coefficients are statistically indistinguishable from the coefficients in the regression which excludes macroeconomic controls and state by year fixed effects.

Unlike estimates based on race, estimates based on SES show substantially stronger variation in the effect of an in-utero cigarette tax on health in childhood. For children from the lowest SES level, a $1 increase in the cigarette tax has a large statistically significant reduction in asthma prevalence by 3.1 percentage points. But for children in the middle SES group, the impact of cigarette taxes remains large and statistically significant effects and produces reduction in asthma prevalence by 1.9 percentage points. For children that are in the highest income levels cigarette taxes have very small and weak economically and statistically significant impact of

[8] Chaloupka and Pacula (1999) examine heterogeneity by race and gender in responsiveness to cigarette taxes.

about 1.1 percentage point reduction in asthma. The impact of in-utero cigarette taxes on measures of severity and health status are small and statistically insignificant for all SES groups. The coefficients by SES remain statistically indistinguishable in specifications that include state by survey year fixed effects and macroeconomic controls.

2.4.4 Assessing the Identification Strategy: Placebo Tests

Alternatively, states may have enacted policies that aim to improve children's health concurrently with tobacco control policies. If cigarette taxes are correlated with other measures that improve children's health, then the effects in this study maybe capturing include the effects of tobacco control policies and state improvements in children's health. Such a scenario is highly unlikely since a large portion of cigarette tax revenues are not invested in health investments. Nevertheless, to assess if other conditions in childhood that should not be influenced by cigarette taxes are affected by cigarette taxes, I conduct a few placebo tests. Since exposure to cigarette smoke affects birth weight which is a strong predictor of health and cognitive abilities, satisfying the exclusion restrictions for the placebo tests is challenging. The NSCH contains rich information on alternative diseases, and I choose diseases that have a high occurrence but that are unlikely to be affected by birth weight such as hearing issues and behavior or conduct issues as diagnosed by a physician.

The results in table 19 show the findings from the placebo tests. First, beginning with behavior and conduct, the impact of a $1 increase in-utero cigarette taxes on produces a very precise zero effect. In the next two specifications the addition of the pre-birth tax rate, post-birth tax rate, and the state by survey year fixed effects does not alter the coefficients as the effect of cigarette taxes on behavior and conduct issues remains economically and statistically insignificant. Next, the placebo test on hearing issues produces similar findings as with behavior

or conduct issues. The effect of cigarette taxes during the in-utero period of life produce effects that are statistically indistinguishable from zero. The zero effects on the in-utero period for both diseases provides some supports for the claim that cigarette taxes are driving the variation in reducing asthma rates instead of alternative investments in improving children's health.

2.4.5 Gender and Variation in Asthma Outcomes

The work on the fetal-origins hypothesizes that gender can influence the impact of an in-utero shock (Almond and Currie, 2011), because male fetuses maybe more sensitive than female fetuses. In table 20, I analyze the differences in outcomes between boys and girls to in-utero smoke exposure. A $1 increase in the cigarette tax reduces the prevalence of asthma in boys by a statistically significant 2.1 percentage points (off of a mean of roughly fifteen percent for boys) as shown in column 1. After accounting for macroeconomic conditions and inclusion of state by year fixed effects during childhood, the coefficient for males is smaller by about twenty percent but nonetheless remains significant at 1.7 percentage points. The effects for girls on the other hand, are robust to specifications with or without macroeconomic controls at about a significant negative 1.2 to 1.3 percentage points (off a mean of roughly 11 percent for women).

2.4.6 Does the Effect on Asthma change by State Prevalence and the Intensity of Taxation?

The substantial variation in smoking prevalence and the intensity of cigarette taxes provide additional opportunities to assess the role of cigarette taxes in reducing childhood asthma. The relative increases in taxes are higher for states that have low taxes and as a result in inducing the marginal smoker to quit would be higher than in states with high taxes. In table 21, I compare outcomes between high and low tax states. For high tax states an increase in the

cigarette tax during the prenatal period has a modestly negative coefficient on childhood asthma in both specifications with or without controls. In low tax states, however, an increase in the cigarette taxes has a large statistically significant negative effect of 5.6 percentage point reduction in asthma. Inclusion of macroeconomic variables and state by year fixed effect reduces the size to 3.6 percentage point reduction in asthma prevalence which is within the error bounds of the earlier estimate. Changes in cigarette taxes in the utero period of life have no statistically distinguishable effects on either severity of asthma or health status in both high and low tax states.

Alternatively, the marginal smoker will also be present in states with high smoking versus low smoking prevalence states. Incidentally, the association between high smoking prevalence and low cigarette taxes states is high. I present the results in table 22 which compare the effects of in-utero exposure between the ten highest states with smoking prevalence and the ten lowest states. Because higher prevalence states should have more responsive smokers, then effects should be larger than in low prevalence states. For high prevalence states, a statistically significant reduction in both asthma prevalence and severity of asthma is seen at 3.9 and 11 percentage points. In low smoking states, the coefficient on asthma is negative and a modestly sized at 1 percentage point, however, it is only marginally significant. Again, the effects are robust to the inclusion of macroeconomic controls and state by year fixed effects. Lastly, the effect on health status is small and statistically indistinguishable from zero in both high and low smoking prevalence states.

2.4.7 Do Cigarette Taxes Influence Racial and Socioeconomic Inequality in Childhood Asthma?

In this section, I decompose the gaps in health for asthma prevalence by both SES and race using an Oaxaca-Blinder decomposition using cigarette taxes. Since the variation in cigarette taxes is plausibly random, the decomposition allows for a causal counterfactual comparison of the asthma gap based on race and SES. The decomposition separates the impacts of cigarette taxes into a coefficient effect or people's responsiveness to cigarette taxes, the unexplained portion of the gap, and an endowment effect which is the explained portion. I present the decomposition results in table 23.

First beginning with SES, I classify children coming from the lowest income level as being poor, and the children from the two highest income levels as non-poor. As shown on table 11, poor children tend to come from states that have slightly higher cigarette taxes on the whole, and this small difference is statistically distinguishable from zero. The difference in the cigarette tax endowment has a negligible effect on the asthma gap between poor and non-poor children. A notable and large statistically significant difference occurs in how poor and the non-poor respond to cigarette taxes. The poor tend to be very responsive to cigarette taxes, and in the absence of cigarette taxes the asthma gap between the poor and poor have been significantly larger. Therefore, the heterogeneous responses indicate that children from the poorest households have benefited from the changes in cigarette taxes.

In contrast, decomposing the asthma gap produces negligible impact of cigarette taxes based on race. Again in table 23 I decompose the gap in asthma between whites and non-whites. The non-white category includes Blacks, Hispanics, and multi-racial individuals. The outcomes of the decomposition are robust to exclusion of multi-racial individuals. The decomposition shows that White and Non-White gap cannot be explained by cigarette taxes. Both the coefficient and endowment effects are very small and statistically indistinguishable from zero.

Thus, doing the decomposition by race implies that racial differences are likely socioeconomic differences because the racial gap reflects an underlying difference in behavioral responses by SES. Thus, cigarette taxes and tobacco control policies do not have a significant impact on the asthma gap between whites and non-whites.

2.5. Cigarette Taxes and Public Policy

It is an established fact of human development that in-utero shocks have adverse effects on adult outcomes, yet very few studies have examined the causal impact of in-utero shocks on outcomes in childhood. This study is one of the few that investigates how adverse conditions in early life affect health in childhood, an intermediate phase to adulthood. By using exogenous variation in cigarette taxes during the in-utero stage of life, this study credibly estimates the effect of early life smoke exposure on children's health, notably asthma. The findings demonstrate that a $1 increase in cigarette taxes produces sizeable reductions in asthma prevalence of 1.7 percentage points. Since poorer households are more likely to be responsive to changes in cigarette taxes, the effect of cigarette taxes on asthma varies considerably based on SES and race. As a result, increases in cigarette taxes produce larger improvements in health for children from the poorer households.

Besides improving health outcomes, the empirical findings from this study raise several implications for public policy, one of which involves the contentious discussions on optimal tax policy for cigarettes. The usual cost-benefit analysis for cigarette tax analysis focuses on the costs and benefits to the smoker and ignores the secondary effects of smoking on child health. An omission of such a large intergenerational spillover that arises from maternal smoking in governmental cost-benefit calculations would then understate the benefits from cigarette tax hikes and result in less optimal tax policy.

In addition to tax policy considerations, reducing asthma prevalence can also have social returns in childhood through reductions in public spending on health expenditures. The value of reduction in asthma, a benefit that arises from changes in cigarette taxes, reveals the social gain is substantial. A child without asthma incurs about $608 in medical expenditures per annum, whereas a child with asthma incurs costs of over $1,042 per annum, respectively a seventy percent increase from non-asthmatic children (PediatricAsthma.org). The total cost to treat asthma per annum in the United States is 3.2 billion dollars, and a fifteen percent reduction would provide social value of approximately $480 million dollars a year. Such an estimate is clearly a lower bound because it neglects the value of improved leisure from better health for children in addition to the dynamic improvements for future health and income. The results from this study suggest that the impact of cigarette tax hikes on children's health can be substantial especially if they persist over the life cycle of the children. Thus, enacting smoking reduction health interventions then also double as human capital interventions that can raise both the production of health and human capital in children.

Lastly, the results from this study also have implications for health disparities and inequality. The differential impacts of cigarette taxes on asthma prevalence by SES illustrate that cigarette taxes have identified one main dimension of childhood health inequality through asthma. Addressing child health can also be an important approach to reducing inequality. Decomposing the health gap by SES and race provides a counterfactual world in which if tobacco policy is absent, i.e. if cigarette tax hikes did not occur, then inequality between children from low SES and high SES would be higher; similarly, the health gap between non-whites and white would also be higher. Thereby cigarette taxes can raise social welfare and child well-being, along with reducing inequality by both SES and race.

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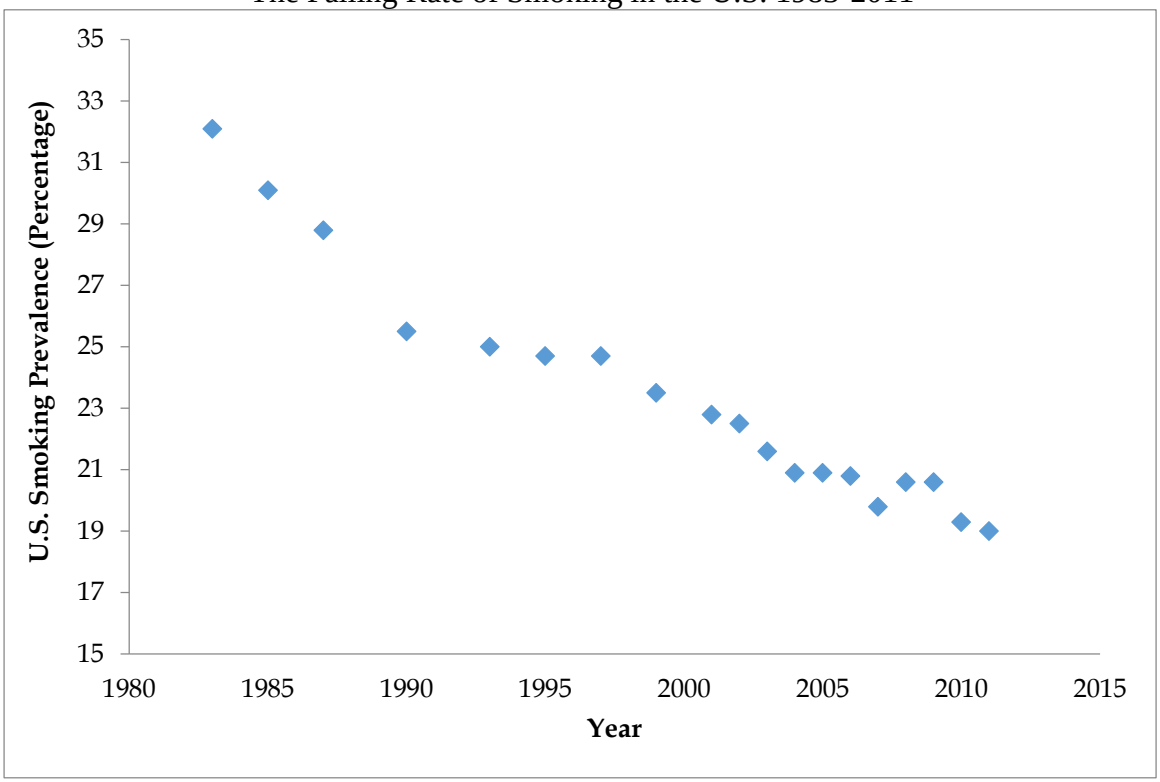
2.7 Figures and Tables

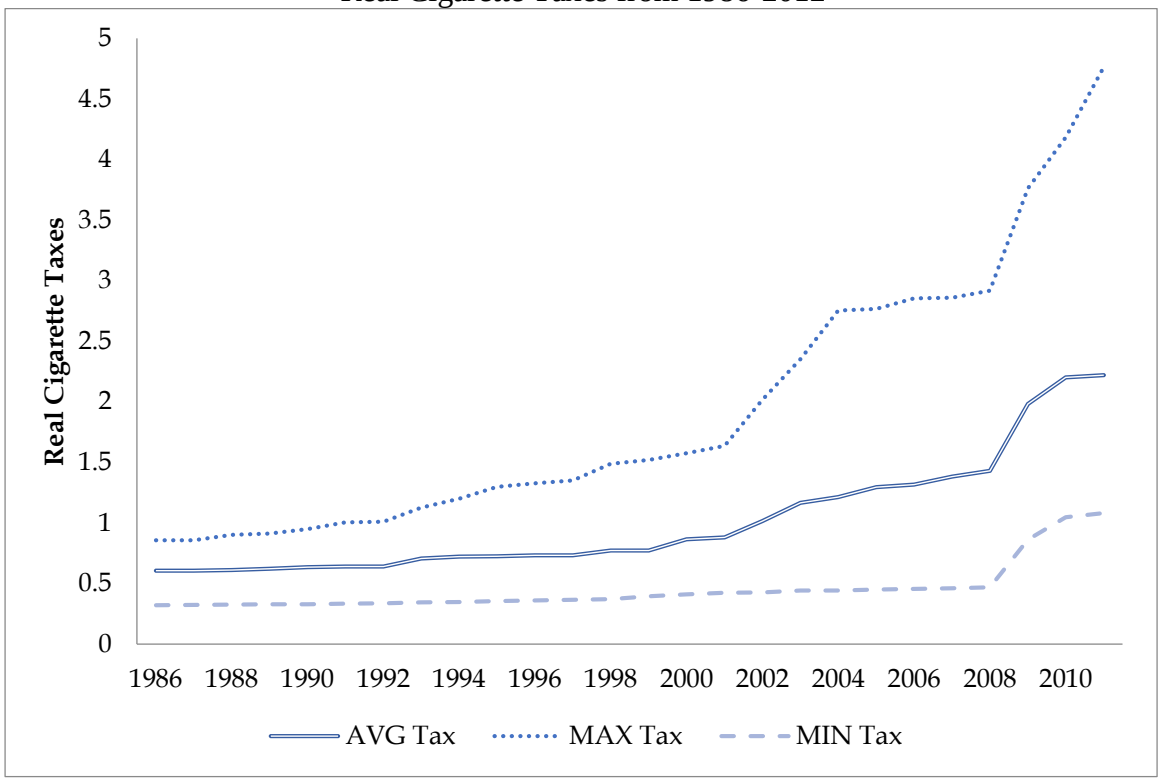
2.7.1 Figures

Figure 4
The Falling Rate of Smoking in the U.S. 1983-2011



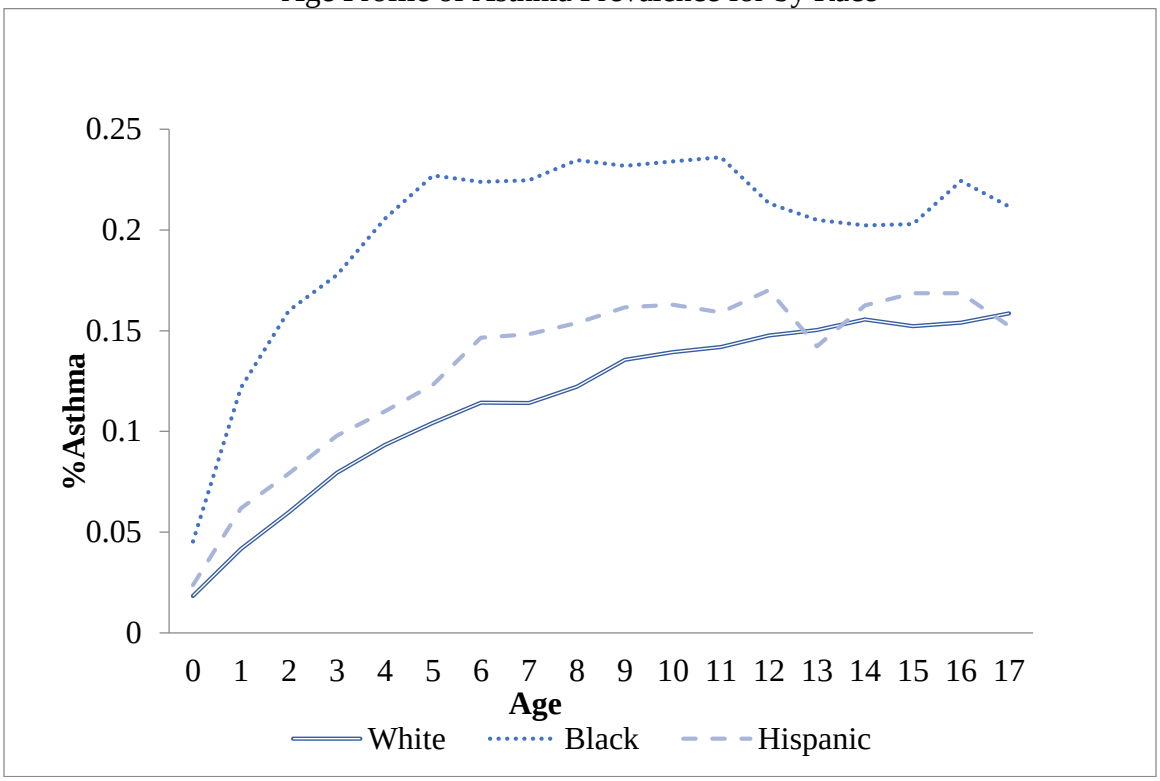
Source: Centers for Disease Control (2015).

Figure 5
Real Cigarette Taxes from 1986-2012



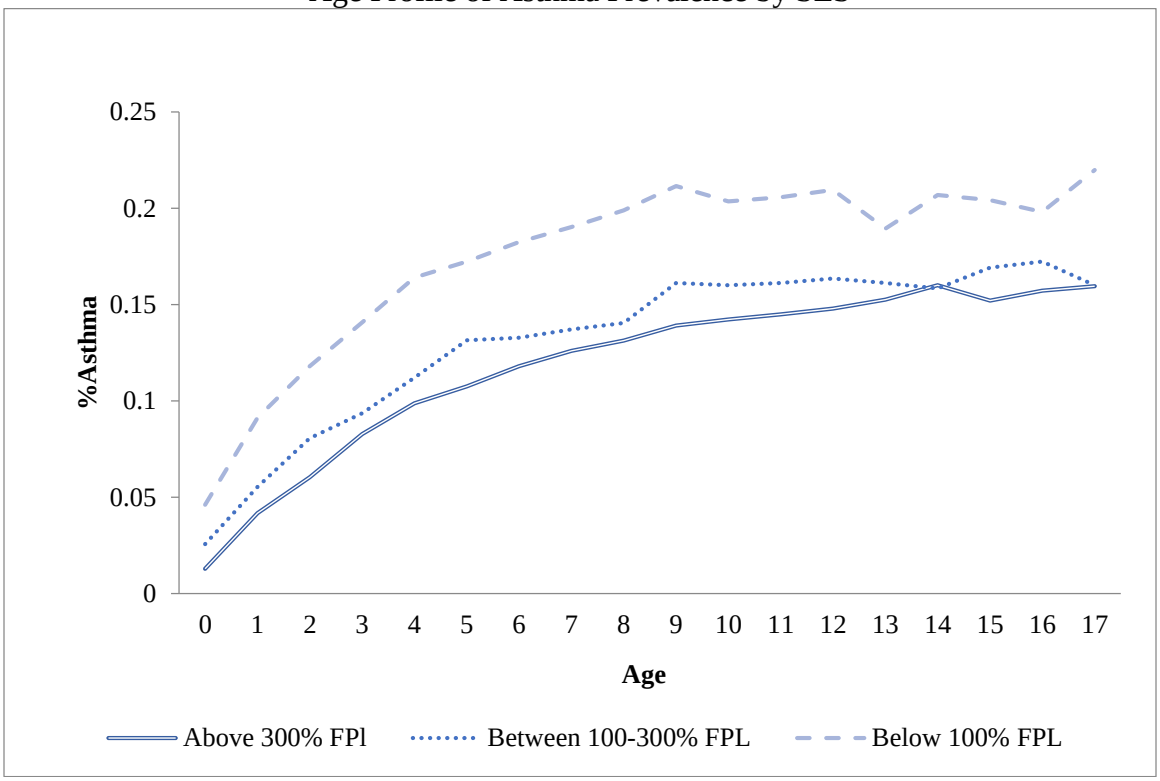
Source: Impacteen (2015) and the author's calculations. Cigarette taxes are measured in constant 2007 dollars.

Figure 6
Age Profile of Asthma Prevalence for by Race



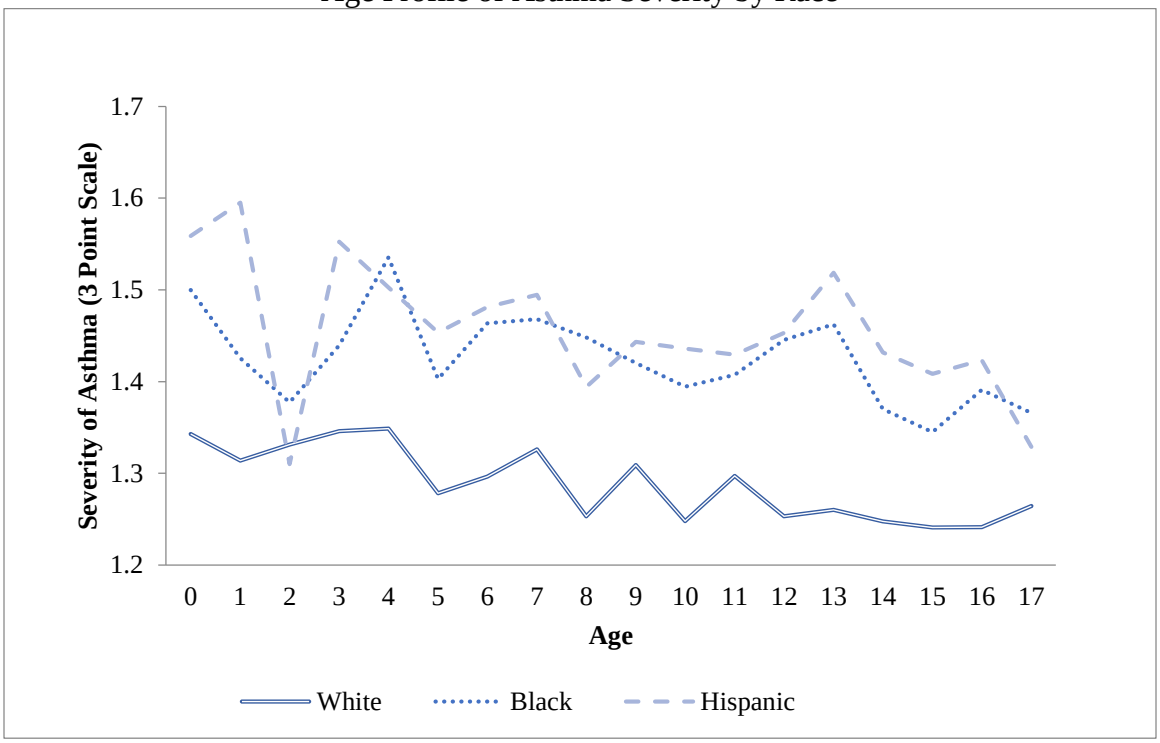
Source: National Survey of Children's Health (2003, 2007, and 2012).

Figure 7
Age Profile of Asthma Prevalence by SES



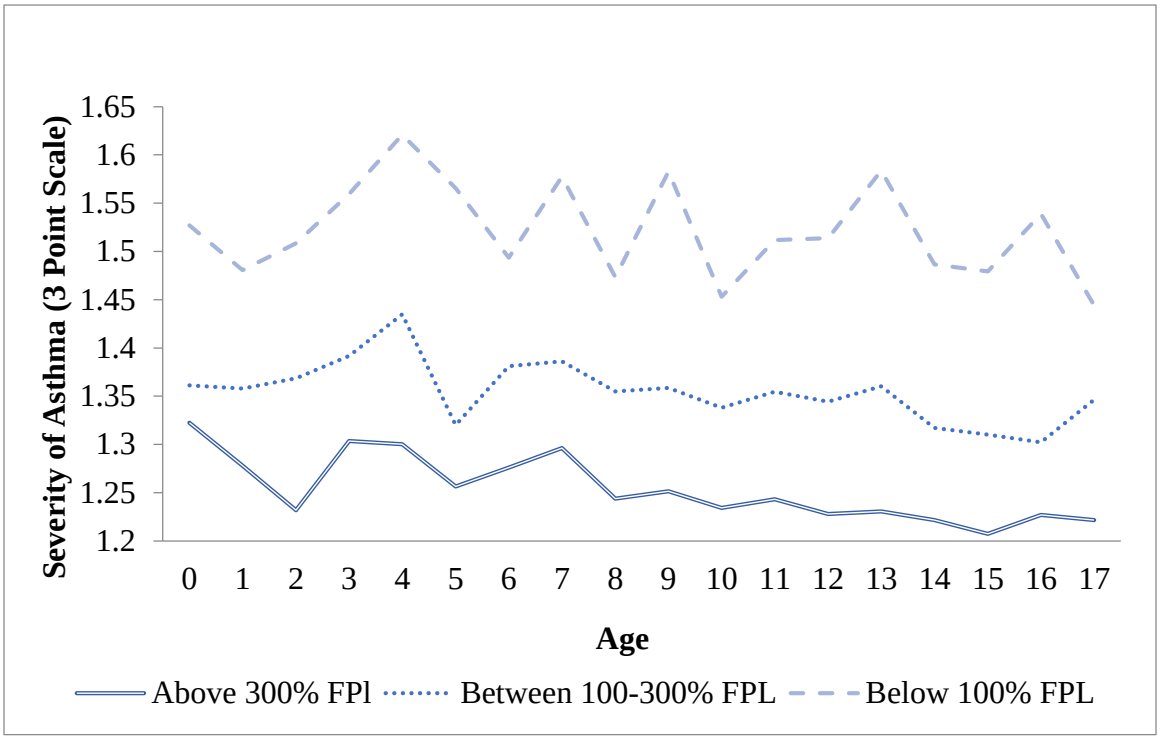
Source: National Survey of Children's Health (2003, 2007, and 2012).

Figure 8
Age Profile of Asthma Severity by Race



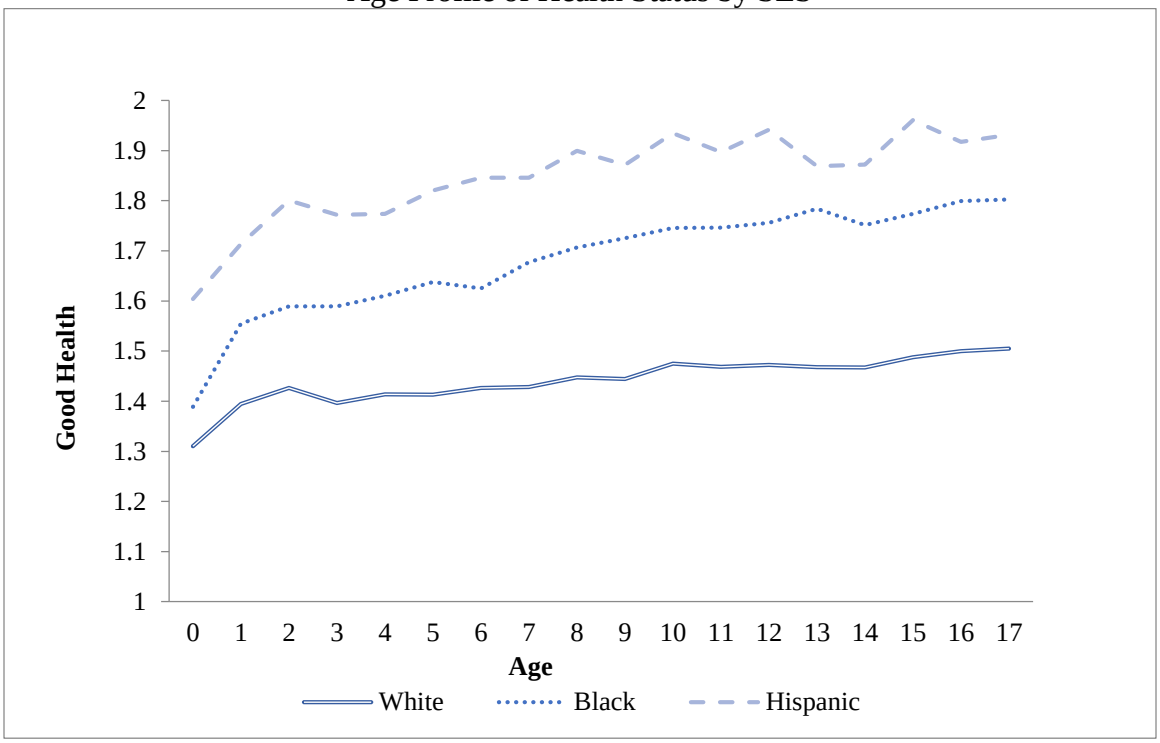
Source: National Survey of Children's Health (2003 ,2007, and 2012). Lower values of severity represent a less severe form of asthma

Figure 9
Age Profile of Asthma Severity by SES



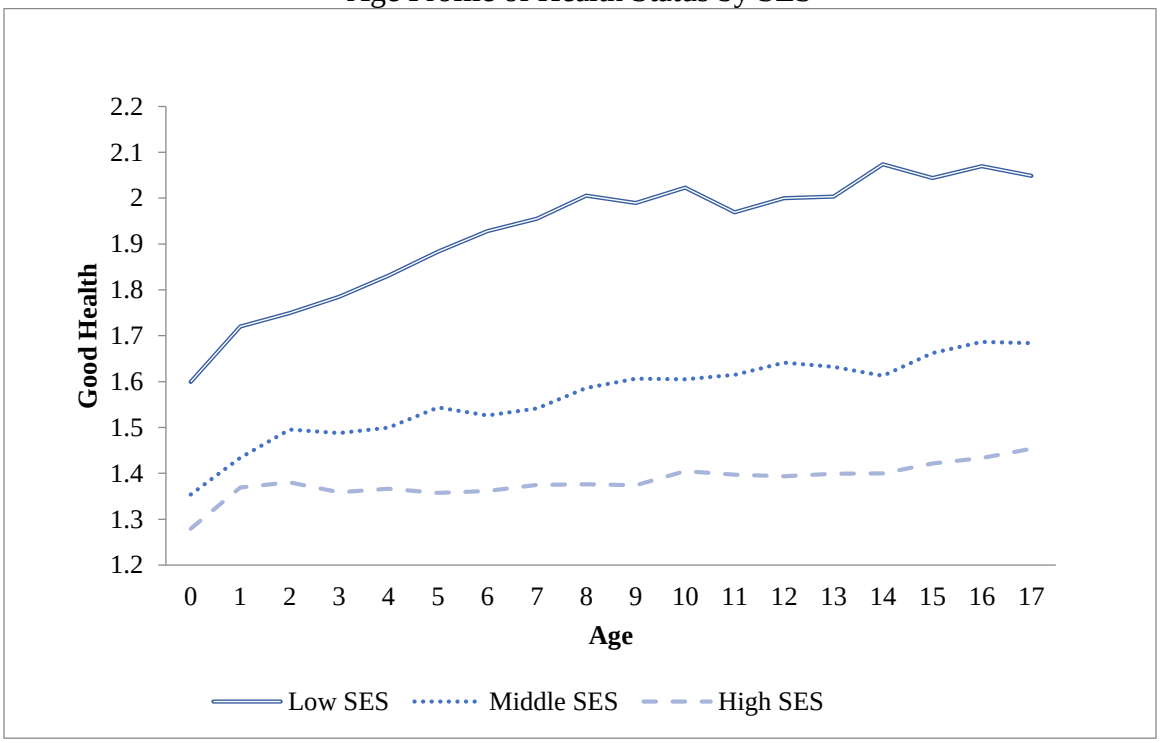
Source: National Survey of Children's Health (2003 ,2007, and 2012). Lower values of severity represent a less severe form of asthma

Figure 10
Age Profile of Health Status by SES



Source: National Survey of Children's Health (2003, 2007, and 2012). Lower values for health status represent better health.

Figure 11
Age Profile of Health Status by SES



Source: National Survey of Children's Health (2003, 2007, and 2012). Lower values for health status represent better health.

Table 13
Descriptive Statistics for the NCHS Whole Sample

Variables	Mean	STD
Demographics		
Low SES	0.117	0.321
Middle SES	0.330	0.470
High SES	0.460	0.498
Male	0.514	0.500
Age	8.930	5.310
White	0.732	0.443
Black	0.100	0.300
Hispanic	0.129	0.335
Household Smoker	0.260	0.439
Dependent Variables		
Asthma	0.137	0.344
Severity	1.324	0.552
Health Status	2.109	0.969
Hearing	0.032	0.177
Behavior or Conduct	0.046	0.210
Cigarette Taxes		
In Utero Tax	0.858	0.483
Pre-Birth Tax	0.815	0.430
Post-Birth Tax	0.544	0.451
Economic Conditions		
Log Per-Capita Income	10.490	0.371
Unemployment	5.38	1.620

Sources: National Survey of Children's Health (2003, 2007, and 2012), Impacteen (2015), and Bureau of Labor Statistics (2015). Notes: Standard deviations are next to the mean of the observations. *N*=289,210

Table 14
Descriptive Statistics of Outcomes by Race and SES

	White		**Black**		**Hispanic**	
	Mean	STD	Mean	STD	Mean	STD
Asthma	0.120	0.324	0.201	0.401	0.129	0.335
Severity	1.286	0.519	1.421	0.613	1.446	0.642
Health Status	2.040	0.935	2.241	1.007	2.396	1.084
Hearing	0.033	0.177	0.034	0.181	0.033	0.178
Behavior	0.046	0.211	0.050	0.219	0.042	0.200
	Low SES		**Mid SES**		**High SES**	
	Mean	STD	Mean	STD	Mean	STD
Asthma	0.170	0.376	0.131	0.337	0.132	0.338
Severity	1.524	0.676	1.350	0.563	1.243	0.479
Health Status	2.509	1.083	2.198	0.959	1.922	0.887
Hearing	0.037	0.189	0.032	0.175	0.031	0.173
Behavior	0.047	0.213	0.048	0.213	0.045	0.206

Source: National Survey of Children's Health (2003, 2007, and 2012). Notes: Standard deviations are next to the mean of the observations. *N*=289,21

Table 15
Examining the Association between having a Smoker in the Household and Child Outcomes

Dependent Variable: Health and Well-Being		
	1	2
Asthma	0.027***	0.027***
	(0.002)	(0.002)
Severity	0.065***	0.062***
	(0.007)	(0.008)
Health Status	0.139***	0.137***
	(0.007)	(0.007)
State and Year	Yes	Yes
State by Year	No	Yes
Controls	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012) Notes: Household smoking is a binary variable of whether anyone in the household smokes. All regressions include full set of age, sex, state dummies, and regression with controls include log per capita income and the local unemployment rate at year of birth. Huber White standard errors clustered at the state level. * $p<0.05$, ** $p<.01$, *** $p<.01$

Table 16
Examining the Association between having a Smoker in the Household and Child Outcomes by Race and SES

Dependent Variable: Measures of Asthma						
	1	2	3	4	5	6
By Race	**White**		**Black**		**Hispanic**	
Asthma	0.025***	0.025***	0.022**	0.024**	0.026***	0.025***
	(0.002)	(0.002)	(0.007)	(0.007)	(0.007)	(0.007)
Severity	0.074***	0.070***	0.062**	0.056*	0.020	0.025
	(0.009)	(0.009)	(0.021)	(0.022)	(0.028)	(0.028)
Health Status	0.183***	0.182***	0.118***	0.114***	-0.023	-0.024
	(0.005)	(0.005)	(0.017)	(0.016)	(0.016)	(0.016)
By SES	**Low**		**Middle**		**High**	
Asthma	0.034***	0.033***	0.020***	0.020***	0.016***	0.016***
	(0.006)	(0.006)	(0.003)	(0.003)	(0.002)	(0.002)
Severity	-0.032	-0.030	0.029*	0.027*	0.016***	0.016***
	(0.019)	(0.020)	(0.012)	(0.012)	(0.002)	(0.001)
Health Status	-0.046**	-0.047**	0.080***	0.080***	0.109***	0.106***
	(0.014)	(0.015)	(0.009)	(0.009)	(0.006)	(0.006)
State and Year	Yes	Yes	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes	No	Yes
Controls	No	Yes	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012). Notes: Household smoking is a binary variable of whether anyone in the household smokes. All regressions include full set of age, sex, year, and state dummies, and regression with controls include log per capita income and the local unemployment rate at the year of birth. Huber White standard errors clustered at the state level. * p<0.05, ** p<.01, *** p<.001

Table 17
The Effect of Early Life Cigarette Taxes on Asthma Prevalence and Health

Dependent Variable: Measures of Asthma						
	1	2	3	4	5	6
Does Child Have Asthma?						
In-Utero Tax	-0.017***	-0.015***	-0.014**	-0.010	-0.019**	-0.010
	(0.003)	(0.003)	(0.005)	(0.005)	(0.006)	(0.006)
Post-Birth Tax			-0.004	-0.007	-0.004	-0.007
			(0.006)	(0.006)	(0.006)	(0.006)
Pre-Birth Tax					0.005	-0.0002
					(0.006)	(0.006)
How Severe is Asthma?						
In-Utero Tax	0.002	-0.018	-0.009	-0.024	-0.056	-0.060
	(0.019)	(0.021)	(0.03)	(0.034)	(0.048)	(0.050)
Pre-Birth Tax			0.0007	0.003	0.004	0.006
			(0.031)	(0.03)	(0.031)	(0.032)
Post-Birth Tax					0.051	0.041
					(0.040)	(0.042)
Health Status						
In-Utero Tax	-0.004	-0.003	0.013	0.012	0.019	0.022
	(0.006)	(0.008)	(0.010)	(0.011)	(0.014)	(0.014)
Pre-Birth Tax			-0.024	-0.023	-0.025	-0.024
			(0.013)	(0.014)	(0.013)	(0.014)
Post-Birth Tax					-0.007	-0.011
					(0.013)	(0.013)
State and Year	Yes	Yes	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes	No	Yes
Controls	No	Yes	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012). Notes: Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables include log state per capita income and local unemployment rates at year of birth. Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. * p<0.05, ** p<.01, *** p<.001

Table 18
The Effect of Early Life Cigarette Taxes on Asthma Prevalence and Health by SES and Race

Dependent Variables: Measures of Asthma						
	1	2	3	4	5	6
By Race	**White**		**Black**		**Hispanic**	
Asthma	-0.017***	-0.017***	-0.025*	-0.021	-0.018*	-0.010
	(0.003)	(0.003)	(0.010)	(0.012)	(0.007)	(0.007)
Severity	0.002	-0.020	0.059	0.071	-0.100*	-0.113*
	(0.019)	(0.020)	(0.037)	(0.048)	(0.04)	(0.051)
Health Status	-0.004	-0.002	0.011	0.032	-0.022	-0.041
	(0.006)	(0.008)	(0.023)	(0.027)	(0.021)	(0.026)
By SES	**Low**		**Middle**		**High**	
Asthma	-0.032***	-0.032***	-0.019***	-0.013**	-0.012**	-0.012***
	(0.008)	(0.008)	(0.004)	(0.004)	(0.003)	(0.003)
Severity	-0.012	-0.030	-0.016	-0.044	0.010	0.018
	(0.038)	(0.041)	(0.027)	(0.031)	(0.023)	(0.026)
Health Status	-0.020	-0.031	0.002	0.006	-0.010	-0.009
	(0.020)	(0.022)	(0.009)	(0.012)	(0.006)	(0.007)
State and Year	Yes	Yes	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes	No	Yes
Controls	No	Yes	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012). Notes: Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables are log state per capita income and local unemployment rates at year of birth. Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. * $p<0.05$, ** $p<.01$, *** $p<.001$

Table 19
Placebo Estimates on the Effects of Early Life Cigarette Taxes on Other Childhood Ailments

Dependent Variables: Measures of Asthma						
	1	2	3	4	5	6
Does Child Have Hearing Issues?						
In-Utero Tax	0.001	0.001	0.003	0.002	-0.0004	-0.001
	(0.001)	(0.002)	(0.003)	(0.003)	(0.005)	(0.005)
Pre-Birth Tax			-0.003	-0.002	-0.003	-0.002
			(0.004)	(0.004)	(0.004)	(0.004)
Post-Birth Tax					0.004	0.004
					(0.003)	(0.003)
Does Child Have Behavior or Conduct Issues?						
In-Utero Tax	-0.001	-0.003	0.011*	0.009	0.011	0.008
	(0.002)	(0.002)	(0.005)	(0.005)	-0.007	-0.007
Pre-Birth Tax			-0.015*	-0.015*	-0.015*	-0.015*
			(0.005)	(0.006)	(0.006)	(-0.006)
Post-Birth Tax					0.001	0.001
					(0.005)	(0.005)
State and Year	Yes	Yes	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes	No	Yes
Controls	No	Yes	No	Yes	No	Yes

Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables are log state per capita income and local unemployment rates at year of birth. Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. Source: National Survey of Children's Health (2003, 2007, and 2012). *N*=289,216. Standard errors in parentheses * p<0.05, ** p<.01, *** p<.00

Table 20
Variation in the Effects of Early Life Cigarette Tax by Gender

Dependent Variables: Measures of Asthma				
	Male		**Female**	
	1	2	3	4
Asthma	-0.021***	-0.018***	-0.012**	-0.013**
	(0.003)	(0.004)	(0.004)	(0.004)
Severity	-0.007	-0.012	-0.011	-0.034
	(0.021)	(0.025)	(0.025)	(0.031)
Health Status	0.0004	0.004	-0.013	-0.012
	(0.007)	(0.008)	(0.010)	(0.012)
State and Year	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes
Controls	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012). Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables are log state per capita income and local unemployment rates at year of birth. Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. * $p<0.05$, ** $p<.01$, *** $p<.001$

Table 21
Variation in the Effect of Early Life Cigarette Taxes by Levels of Cigarette of Taxes

Dependent Variables: Measures of Asthma				
	High Tax		**Low Tax**	
	1	2	3	4
Asthma	-0.006	-0.004	-0.056***	-0.036*
	(0.005)	(0.005)	(0.011)	(0.012)
Severity	-0.002	-0.006	-0.018	-0.065
	(0.023)	(0.025)	(0.064)	(0.057)
Health Status	-0.008	-0.008	-0.071	-0.065
	(0.006)	(0.005)	(0.035)	(0.041)
State and Year	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes
Controls	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012). Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables are log state per capita income and local unemployment rates at year of birth. The high tax states include Alaska, Connecticut, Hawaii, Maine, Massachusetts, Michigan, New York, New Jersey, Rhode Island, and Washington. The low tax states include Alabama, Georgia, Kentucky, Mississippi, Missouri, North Carolina, South Carolina, Tennessee, Virginia, and Wyoming. Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. * $p<0.05$, ** $p<.01$, *** $p<.001$

Table 22
Variation in the Effect of Early Life Cigarette Taxes by Smoking Prevalence

Dependent Variables: Measures of Asthma				
	High Smoking		**Low Smoking**	
	1	2	3	4
Asthma	-0.039***	-0.033**	-0.009	-0.012
	(0.008)	(0.01)	(0.005)	(0.006)
Severity	-0.119*	-0.149**	-0.065*	-0.068
	(0.040)	(0.042)	(0.026)	(0.033)
Health Status	-0.024	-0.029	-0.005	-0.021
	(0.022)	(0.028)	(0.013)	(0.014)
State and Year	Yes	Yes	Yes	Yes
State by Year	No	Yes	No	Yes
Controls	No	Yes	No	Yes

Source: National Survey of Children's Health (2003, 2007, and 2012Each column represents a separate regression for the respective dependent variable. All regressions include full set of dummies for age, sex, year, and state. The control variables are log state per capita income and local unemployment rates at year of birth. The high prevalence states are the following Kentucky, W. Virginia, Arkansas, Tennessee, Louisiana, Indiana, Mississippi, Missouri Alabama, Michigan, Ohio, and Oklahoma. The low prevalence states are the following: New Hampshire, Washington, Arizona, Vermont, Idaho, Massachusetts, Maryland, New York, Connecticut, Utah Hawaii, California.) Huber-White Standard errors clustered at the state are included in parenthesis under the coefficients. * $p<0.05$, ** $p<.01$, *** $p<.001$

Table 23
Decomposing the Asthma Gap by Race and Socioeconomic Status

Asthma Gaps					
	Coefficient	S.E.		Coefficient	S.E.
By Socioeconomic Status			*By Race*		
Non-Poor	0.127***	(0.003)	White	0.123***	(0.003)
Poor	0.170***	(0.006)	Non-White	0.160***	(0.005)
Differences	-0.043***	(0.005)	Differences	-0.036***	(0.005)
Explained	0.005***	(0.001)	Explained	0.0001	(0.002)
Unexplained	-0.048***	(0.004)	Unexplained	-0.036	(0.004)
Explained			*Explained*		
Cigarette Tax	0.001***	(0.0003)	Cigarette Tax	0.0002	(0.0004)
Unexplained			*Unexplained*		
Cigarette Tax	0.016*	(0.007)	Cigarette Tax	0.004	(0.006)

Source: National Survey of Children (2003, 2007, and 2012). Selected coefficients presented for the asthma gap by SES and race. Both decompositions include controls for age, year, and gender of child. Huber-White standard errors clustered at the state. * p<0.05, ** p<.01, *** p<.001

3. VITA

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Fields

Major Fields	Health Economics, Labor Economics, and Economics of Education
Minor Fields	Public Economics and Political Economy

Education

2016	Ph.D. in Economics, University of Illinois at Chicago
2010	M.A. in Economics, University of Illinois at Chicago
2008	B.A. in Economics (with distinction), Political Science, minor in Psychology, University of Illinois at Chicago

Publications

Double Take: The Effect of Sibling Sex Composition on Women's Earnings, Schooling, and Labor Supply. Forthcoming at **Economics Letters**

Awards and Honors

2015 – 2016	CKF Dissertation Grant
2015	2nd Place, Illinois Economic Association PhD Paper Competition
2015	Student Fellow of the Public Choice Society
2014 – 2016	Daniel Searle Fellowship
2014 – 2015	Visiting Dissertation Fellow, Mercatus Center
2014, 2015	APEE Young Scholar Award
2014, 2015	Hayek Fund Award
2013 – 2014	Adam Smith Fellow, Mercatus Center
2013 – 2014	Humane Studies Fellowship

Teaching Experience

Instructor
Loyola University Chicago
Principles of Macroeconomics:
Spring 2014-60 students (3.9/5.0)
Spring 2015-51 students (4.4/5.0)

Principles of Microeconomics:
Fall 2014- 46 students (4.4/5.0)
Fall 2015- 49 students (4.4/5.0)

Daley College - City Colleges of Chicago
Principles of Macroeconomics:
Fall 2012 - 17 Students
Spring 2013 – 46 Students

Teaching Assistant
University of Illinois at Chicago
Spring 2014 – *Tobacco Control*
Spring 2014– *MBA Microeconomics*

Selected Presentations
University of Arkansas at Little Rock, 2016
APEE, 2015, 2016
Western Economic Association, 2016
Bureau of Economic Analysis, 2016
Society of Labor Economists, 2015, 2016
Southern Economic Association, 2015
International Sociological Association, 2015
UIC Economics Lunch 2013, 2015
Public Choice Society, 2014, 2015
Eastern Economic Association, 2015
Illinois Economic Association, 2014, 2015
Southern Regional Science Association, 2014
University of Illinois at Chicago Seminar, 2011

Selected Associations
American Economic Association
Society of Labor Economists
The National Economists Club, Washington DC
Southern Economics Association
Western Economic Association

Research Experience and Other Employment
Summer 2013 Pathways Intern, USDA Economic Research Service
2010 Staff Consultant/Intern, Robinwood Consulting

Languages
English (Native), Hindi (Conversant), Urdu (Conversant), Spanish (Intermediate)

Skills

Advanced: Stata, SAS, SPSS,
Intermediate: Matlab, R, ArcGIS

Professional Development

Summer school on Socioeconomic Inequality at the University of Chicago, July 2016
ICPSR Summer Workshop, January 2016
UC Berkeley Initiative for Transparency in the Social Sciences, June 2015
Art of Teaching Workshop, July, 2014
Liberty Fund: Topics in Public Choice, September, 2014
Public Choice Outreach, August, 2013