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Tobacco Smoke and Asthma among Adults at the National and State Levels: Do Smoke-Free Laws and Regulations Affect Smoking Rate among those with Asthma?

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ABSTRACT

Background: Asthma is a chronic lung disease that inflames and narrows the airways. This results in recurrent episodes of coughing, wheezing, shortness of breath, and chest tightness. Although the causes of asthma are poorly understood, genetic and environmental factors have been implicated in the development and exacerbation of the disease. Among environmental risk factors, cigarette smoke is a well-known risk factor to trigger asthma symptoms. Exposure to secondhand smoke irritates the airways and may trigger an attack in adults with asthma. Smoke-free laws and regulations in the United States differ by state. The enforcement of smoke-free legislation has been related to asthma rates as it has been shown that they lead to a sustained drop in emergency hospital admission for asthma among adults. These laws and regulations are also necessary in reducing smoking rate and secondhand smoke exposure.

Objective: The purpose of this thesis is to examine the association between tobacco smoke rates and asthma status among adults at the national and state levels and to evaluate the effects of state smoke-free laws and regulations on tobacco smoking rate among adults with asthma.

Methods: The Centers for Disease Control and Prevention's 2009–2010 Behavioral Risk Factor Surveillance System data was used for the analyses. SAS-callable SUDAAN (version 10.0.0, RTI International, NC) was utilized to account for the complex sampling design of the BRFSS, and sample weights were used to produce estimates that were generalizable to the state and U.S. adult population. In addition to calculating descriptive statistics, chi-square tests and multivariate logistic regression were used to test for group differences and association between variables of interest. State level smoking rates were ranked to identify states that are in the lower and upper 20th percentiles and compare them with states' smoke-free laws and regulation status.

Results were considered significantly different if 95% confidence intervals (CIs) did not overlap or if statistical testing at $p < 0.05$ was applicable.

Results: Asthma prevalence rates are higher among adults that smoke cigarettes (10.5%, [aPR] =1.2) compared to non-smokers (7.8%, [aPR] =1.0). Of the 869,519 adult respondents in the survey, 8.5% reported having asthma. Nearly one-fifth (17.2%) of adults without asthma smoked cigarettes, while (21.7 %) of adults with asthma smoked. Females (10.5%) had higher asthma prevalence rates than males (6.4%). Black persons (10.0%), persons of American Indian (13.0%) descent had higher, and those of Hispanic (6.7%) descent had lower asthma rates than white persons (8.6%). Adults with a high

school education or less (9.1%) had higher asthma rates than those with an education level that was equivalent to a 4 year college or more (7.3%) , and those with low income (<\$15,000) had higher rates (13.3%) than those with high income (6.8%). Percentage of male (23.4%) and females (20.7%) with asthma who smoke are higher than those that do not smoke (19.3% and 15.1%, respectively).

Asthma prevalence rates and smoking rates vary by geographic location. Smoking rates among adults with asthma was highest in the South (LA, AL, SC, TN, OK, MS, AR, WV, KY) and a couple of Midwest states (OH, IN,). Evaluating the association between the 2008 State of Tobacco Control Report and smoking rate among adults with asthma by state showed a statistically significant relationship between smoking rate among adults with asthma and smoke-free policy and regulation at the state and national level. On average, states with the lowest smoking rate among persons with asthma (smoking rates less than 20th percentile) had significantly higher smoke-free policy grades (mean grade [sd]=7.2 [1.99]) than states with a high smoking rate (smoking rate of 80th percentile or more) (mean grade [sd]=2.0 [2.00]) (p-value < 0.00001).

Conclusion: Although most U.S. state smoke-free policies and regulations are relatively new, it is evident that these laws are effective in promoting cessation among adults and reducing nonsmokers' exposure to secondhand smoke. The study found that smoke-free laws may improve health by lowering asthma prevalence and smoking rates among adult smokers. Also, these policies in turn protect non-smokers from the harmful health effects of secondhand smoke.

Key Words: asthma, asthma prevalence, cigarette smoke, adults, smoke free policy

**TOBACCO SMOKE AND ASTHMA AMONG ADULTS AT THE NATIONAL
AND STATE LEVEL: DO SMOKE-FREE LAWS AND REGULATIONS AFFECT
SMOKING RATE AMONG THOSE WITH ASTHMA?**

By

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Georgia State University
Institute of Public Health

A Thesis Submitted to the Graduate Faculty of Georgia State University in Partial
Fulfillment of the Requirements for the Degree
Master of Public Health

Atlanta, GA
2013

APPROVAL PAGE

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SMOKING RATE AMONG THOSE WITH ASTHMA?

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DEDICATION PAGE

The following thesis document is dedicated to my family for their constant support and love.

ACKNOWLEDGEMENTS

I am thankful to God who has guided me through each step of my life. I thank the Lord for all His blessings.

I would like to thank my dear parents, my sister and brother and the rest of my family for all their support and love. You always believe in me.

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To my classmates at Georgia State University and Emory University: I am thankful for your help, friendship and encouragement.

AUTHORS'S STATEMENT

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CHAPTER I

INTRODUCTION

1.1 Background

Asthma is a respiratory disease that attacks the airways in the lungs; this chronic inflammatory condition affects more than 25 million Americans (Centers for Disease, 2011). Trends in current asthma prevalence rates have been on the rise with an estimated 16.5% increase from 7.3% in 2001 to 8.5% in 2010 (Centers for Disease, 2011). The previous Centers for Disease Control and Prevention (CDC) reports indicate significant differences in asthma prevalence between many population subgroups. While differences exist between the demographic subgroups- children, adults, males, females, whites, blacks, and Hispanics- there has been a rising trend in asthma prevalence across all subgroups over the past few decades. For instance, analyses of trends for 2001-2010 for current asthma prevalence for population-based rates show that in 2010, females had higher prevalence than men (9.7% compared with 5.7%); this prevalence rate has been increasing at a rate of 1.8% per year among males and females (Centers for Disease, 2011). The rise in the asthma prevalence rates creates a substantial health and economic burden on individuals and society (Hahn, 2010). It is estimated that the mean annual per capita healthcare expenditure for asthma related cases in 2006 was \$5,322 (in 2010 US dollars) (Rank, 2012); total cost estimate of asthma to the United States was \$56 billion in 2007 (Barnett, 2011).

Science has not found a cure for asthma. The causes of asthma are poorly understood as multiple factors (genetic predisposition, environment, gene-environment interactions) play a role

in the development and worsening of asthma symptoms (Grassi, 2006;Ober, 2005;Willemsen, 2008;Yang, 2007). However, the rise in asthma in recent years cannot be attributed to changes in genetic factors. Instead, intervention efforts to halt the increasing asthma prevalence should be redirected to focus on environmental factors.

Cigarette smoking and secondhand smoke are among the main environmental triggers of asthma. Even though it is known that smoking aggravates asthma symptoms, a higher percentage of adults with asthma smoke when compared to the percentage of adults without asthma that smoke (Table 3). The combination of asthma and active cigarette smoking results in more pronounced asthma symptoms, accelerated decline in lung function, and reduce the body's response to corticosteroid treatment (asthma medication) (Levy, 2002).

Smoke-free laws effectively reduce smoking and secondhand smoke exposure. In addition, smoke free legislation is shown to improve indoor air quality, increase the likelihood of smoke cessation and lower the rates of asthma exacerbations (Hahn, 2010;Jill S. Rumberger, 2010;Levy, 2002). It has been noted that aside from the health outcomes, smoke free legislation does not negatively affect employment or business profits (Haw, 2007;Prevention, 2007). Smoke free legislation is an important step that needs to be taken into account by public officials as a public health intervention strategy that reduces the long-term health and financial burden of cigarette/tobacco use. Asthma is a long-term disease that cannot be cured but through policy changes it can be relatively controlled and managed in order for individuals to live normal, active lifestyles (Centers for Disease, 2011).

1.2 Purpose of Study

The purpose of this study is to investigate the relationship between the prevalence of smoking and secondhand smoking exposure among adults with asthma by analyzing data from the 2009-2010 Behavioral Risk Factor Surveillance System. In addition, the impact of smoke-free policies on smoking prevalence among adults with asthma was examined by studying The American Lung Association State of Tobacco Control 2008 report that tracks progress on key tobacco control policies at the state and federal level and assigns grades to tobacco control laws and regulation enacted as of January 1, 2009 (Association, 2012). The results of this study bring light to whether smoke-free policies are cost effective in terms of reducing the annual direct healthcare cost of asthma and the indirect cost measured by lost work days (Association, 2012).

1.3 Research Questions

The purpose of this study is to gather data from the Behavioral Risk Factor Surveillance System (BRFSS) and the American Lung Association State of Tobacco Control report in order to answer the following questions:

- I. What is the current asthma prevalence rate and factors associated with it?
 - II. What is the percentage of smokers by asthma status, selected demographic characteristics and state?
 - III. What is the impact of state wide smoke-free laws on smoking rates among persons with asthma?
-

CHAPTER II

REVIEW OF THE LITERATURE

2.1 State of Asthma Defined

Asthma continues to be a major public health concern in the United States and worldwide. Asthma is a chronic and life-threatening disease that affects 25 million people in the U.S. and 235 million people around the world, according to the World Health Organization, 2012(To, 2012). In the U.S. asthma affects nearly 8% of the adult population and 9.5% of children aged 0-17 years. Projections speculate that by year 2025, the number of people worldwide with asthma will increase to 400 million, a 70% surge, as countries become more urbanize (To, 2012). The increase in prevalence poses a substantial concern in terms of the health and financial burden that the disease has on the general population and the country. In order to tackle the complexities of asthma, additional research is needed to understand the causes of asthma.

In asthma patients the airways in the lungs become inflamed which makes the airways sensitive; certain irritants cause the airways to tighten, become smaller and produce extra mucus; when the airways become obstructed the air flow to the lungs decreases making it harder to breath (Piipari, 2004). Asthma presents itself with the following respiratory symptoms: wheezing, coughing, nocturnal chest tightness, shortness of breath or attacks of breathlessness following activity, and chest tightness particularly at night or early in the morning (Cerveri, 2012). The airway obstruction that characterizes asthma is reversible and patients with this disease respond to corticosteroid therapy whether inhaled or orally (Cerveri, 2012).

At present, there is no known cure for asthma but people with asthma can be symptom free for days, even years with an affective asthma management and control (National Asthma, 2007 #111). The National Asthma Education and Prevention Program (NAEPP) was established in 1989 to address the asthma-related prevalence and mortality rates and to enhance the lives of persons with asthma. NAEPP convenes a panel of expert to perform a systematic review of all published scientific literature and report new finding or confirm that current asthma guidelines reflect scientific advances. This panel of 18 experts is independently appointed by the NAEPP and prepares a report, Expert Panel Report 3-Guidelines for the Diagnosis and Management of Asthma (EPR-3), that examines the up-to-date scientific progress made in the field of asthma control and management (National Asthma, 2007). Based on EPR-3, an affective asthma management consists of four components:

1. *Assessment of asthma severity and control*, there are two domains (National Asthma, 2007):

- a. *Assessment of impairments*

With regards to assessing asthma severity, physicians are required to examine the symptoms and lung function of current impairment. In evaluating asthma impairment, the following symptoms are taken into consideration: daytime symptom, awakening at night, the need for inhaled Short-Acting Beta₂-Agonists (SABA) for quick relief of symptoms, missing work or school days, the inability to engage in normal day-to-day activity and a quality-of-life assessment (National Asthma, 2007).

In terms of lung function, a spirometer classifies severity by measuring the forced expiratory volume in 1 second (FEV₁), the forced expiratory volume in six seconds (FEV₆) in adults, and the forced vital capacity (FVC) (Fuhlbrigge, 2001). The use of this tool has been found effective in cross-sectional and longitudinal studies of children that provided proof of an association between severity of airflow obstruction and the risk of exacerbations (Fuhlbrigge, 2001). These studies report FEV₁ to be useful in indicating risk for exacerbations; while FEV₁/FVC is a more accurate measure of severity in the impairment domain (National Asthma, 2007).

Aside from examining the effects of asthma on the quality of life and current functional capacity on an ongoing basis, we also need to assess the risks asthma presents for adverse events in the future.

b. *Assessment of risk for future exacerbation* (National Asthma, 2007)

The second part of assessing asthma severity and controlling it is to measure the future risks that asthma may present. These adverse events include exacerbations of asthma presented by episodes of progressively deteriorating shortness of breath, wheezing, chest tightness, and cough- and the risk of death.

Patients found to be at a high risk of future exacerbations are closely monitored and will need to undergo periodic assessments by their physicians. Depending on the frequency and intensity of exacerbations (measured by hospitalization, ICU admission, or unscheduled clinical care), several interventions are to be considered (discussed in the following three asthma management

components). To reduce risk, clinicians focus on avoiding frequently occurring exacerbations of asthma and reducing the need for ED visits or hospitalizations (National Asthma, 2007). Also, they work to stop the progressive loss of lung function especially in children. Finally, they present patients with options of prescriptive medicine with minimal or no side effects.

2. Education for people with asthma and those that provide asthma care (National Asthma, 2007)

EPR-3 provides recommendation on asthma self-management education at multiple points of care, tools for asthma self-management, and provider education. It is believed that specialized training in self-management is essential in creating behavior that modifies the outcomes of chronic illnesses such as asthma. Educating persons with asthma in self-management skills of ‘self-assessment, use of medications, and actions to prevent or control exacerbations’ will result in ‘reduction in urgent care visits and hospitalizations, reduction of asthma-related care costs, and improvement in health status’ (National Asthma, 2007, p.96)

3. Control of environmental factors and co-morbid conditions that affect asthma (National Asthma, 2007)

Evidence has shown a strong association, in people who have asthma, between exposure to allergens and an increase in asthma symptoms and exacerbation of the disease (EPR-3). EPR-3 notes that reducing exposure to indoor and outdoor inhalant allergens to which the patient is sensitized can improve asthma control. The report recommends an action plan for effective indoor and outdoor allergen avoidance in a multifaceted approach.

4. Appropriate medical treatment (National Asthma, 2007)

Among medical treatment there are two types of asthma medications: quick relief medications provide prompt relief of symptoms and long-term control medications prevents symptoms by reducing inflammation. Quick-relief meds are to be taken as needed when asthma symptom exacerbations occur, they do not provide long-term asthma control. On the other hand, to control asthma symptoms long-term patients need to take long-term control medication daily regardless of experience in symptoms. Corticosteroids are the first and most common choice of long-term controller medications (National Asthma, 2007).

Corticosteroids is an anti-inflammatory treatment that suppresses the inflamed airways 'by inducing the recruitment of the nuclear enzyme histone deacetylase 2 (HDAC2) to multiple activated inflammatory genes, which leads to deacetylation of the hyperacetylated genes, thereby suppressing inflammation' (Barnes, 2008, p.7). Several studies confirm that the regular administration of corticosteroids have been found to reverse the airway obstruction and inflammation caused by the disease (Fuhlbrigge, 2001;Piipari, 2004).

2.2 Risk Factors

Asthma is a condition that is complex with genetic as well environmental components. Researchers believe that exposure to a number of factors (genetic, environmental) and interactions between these factors are association with the exacerbation of asthma-like symptoms and the development of the disease (Grassi, 2006;Ober, 2005;Willemsen, 2008;Yang, 2007).

Other host-related factors have also been found to be associated with the occurrence of asthma in adults and children. Elements that are considered host-related include the age of the patient, their sex, race, ethnicity, smoking status, and obesity. Co-morbid conditions such as gastro-esophageal reflux disease [GERD] and chronic sinus disease have been shown to have a link to asthma (Pearlman, 2009). These host-related factors are classified as modifiable (education, income, health insurance status, smoking, obesity, and co-morbid conditions) (Beane, 2007;Gwynn, 2004;Services, 2006;Strine, 2004) and non-modifiable (e.g., heredity, age, sex, race, and ethnicity) (Gwynn, 2004;Strine, 2004;Willemsen, 2008).

Previous studies show that differences in life experiences (e.g. family, social, and economic environment), lifestyle choices (smoking, obesity, leisure time physical activities), and exposure to adverse indoor and outdoor environment factors (e.g., mold, pollens, house dust mites, cockroaches, rodents, animal allergens, environmental tobacco smoking [ETS], and other air pollutants) may account for some of the racial and ethnic differences in asthma prevalence (Committee on the Assessment of Asthma and Indoor Air, 2000;Gwynn, 2004;Strine, 2004). Even though previous studies have identified potential predictors of asthma, specific information about the predictive factors among racial and ethnic groups has been limited. These study findings were limited to either a few major race/ethnic groups (e.g., white vs. non-white or whites vs. blacks, Hispanics, and other race) or predictive factors were examined by controlling for the effects of race/ethnicity.

The potential determinants of asthma vary on a broad spectrum that ranges from genetic history to environmental factors.

Genetic

While genetic predisposition may result in the development of allergies (eczema or hay fever) and asthma, the genotype of asthma is multifactorial and positive family history does not necessarily predict the presence of the disease (Pope, 2002). Gene studies have found the presence of age-specific genetic effects (Dijk, 2013;Forno, 2012;Henderson, 2009). Several published studies and meta-analysis show genome-wide association for childhood and adult asthma, obtaining genetic associations at a genome-wide significant level, either in the original cohort, the replication cohort, or in a combined analysis. It is estimated that at least 40 genes were found to be associated with the development of asthma; replication studies in independent populations confirm these findings (Forno, 2012).

In the US, a study on an original population of Non-Hispanic whites found that the presence of the gene, PDE4D, had a strong association with the development of asthma in children and adults (Himes, 2009). The PDE4D sequence works to constrict the airways smooth-muscle cells, leading to asthma. The same study was replicated using African-Americans, European Americans, US Hispanics, and British and resulted in the same finding. However, the presence of PDE4D in the African-American population had no association with the development of asthma in adults or children (Forno, 2012;Himes, 2009).

Other findings in gene variants are essential in understanding the role of genetics in asthma pathophysiology and the effectiveness of certain gene inhibitor medications. Henderson et al. confirms that in order to find solutions to questions about the cause of asthma, we have to maximize our knowledge on genome-wide linkage to asthma (Dijk, 2013). To increase our

understanding of the etiology and natural history of asthma, ‘it is important to consider the impact of precise classification of phenotypes on issues such as replication of associations, misclassification bias and harmonization of outcomes across populations large enough to achieve sufficient statistical power, particularly for the detection of gene-gene and gene-environment interactions’ (Henderson, 2009).

Demographic

Predictors of asthma differ by sex, race/ethnicity, age, education, income, and smoking status. Asthma disproportionately affects certain demographic groups, and the disproportionate impact has persisted over time (Frieden, 2011;Prevention, 2007;Centers for Disease, 2011).

Current asthma prevalence is higher among women than men and among children (9.5%) than adults (7.7%) (Subbarao, 2009). In women, asthma prevalence is 9.2% compared to 7.0% for men. Subbarao et al., summarizes the findings of several research papers by reporting that in women airway hyper responsiveness increases in female in adulthood compared to men making them more susceptible to developing asthma. At puberty the incidence and prevalence of asthma starts to switch from being higher in boys to becoming higher in girls (Subbarao, 2009). Studies have been unable to find any scientific evidence that pubertal development is a resultant of asthma occurrence. This shift in the occurrence of asthma appears between the sexes during the first and second decades of life and during the fifth or sixth decades (Subbarao, 2009). In other words, before the age of 10 or mid-teen years, boys have higher frequency of asthma than girls. While no scientific evidence shows interference between puberty and asthma occurrence, after

puberty girls asthma rates starts to rise surpassing the rates of boys. However, investigations found that in general occurrence of asthma decreases with age in both sexes.

Aside from the gender differences in asthma prevalence, other selected characteristics affect the rates of asthma. For instance, asthma prevalence differs by race, ethnicity, poverty status, geographic region and urbanicity. Trends in population-based asthma prevalence by race and ethnicity show the interaction between prevalence rates and how they relate to these two factors. In the United States, the average annual 2008-2010 asthma prevalence was higher among blacks (11.1%), American Indian or Alaska Native (AI/AN) (8.8%) , persons of multiple race, and lower among Asian (5.3%) and Hispanics (6.3%) than whites (7.3%) (Gwynn, 2004;National Asthma, 2007;Prevention, 1999;Prevention, 2000;Smith, 2005;Strine, 2004). Compared with non-Hispanic white and non-Hispanic black persons, current asthma prevalence is higher among Puerto Rican and lower among Mexican persons.

The National Health Interview Survey (NHIS) shows that family income along with geographic region and urbanicity play an important role on asthma prevalence. Those with family income below the federal poverty threshold (11.2%) have higher current asthma prevalence rates compared to persons with family income at or above the federal poverty line (8.5%) (Akinbami, 2012). Combining this finding with the examination of geographic region, analysts found that prevalence is higher in the Northeast (8.8%) than in the West (8.0%) and South (7.6%) and in the Midwest (8.7%) compared to the South (7.6%) (Akinbami, 2012). However, when prevalence rates for urbanicity were examined, current asthma prevalence did not differ between metropolitan and nonmetropolitan areas.

Environmental

Among the factors that affect the current asthma status are seasonal and non-seasonal allergens such as cockroaches, dust mites, mold, pets and animals, pollen, and other air pollutants and asthma irritants such as cigarette smoke (Huss, 2001). The National Institutes of Health (NIH) and the Environmental Protection Agency (EPA) funded a 2-year study that found a strong relationship between modestly increasing the levels of air pollution and elevated frequency of asthma symptoms and lower lung function (Pope, 2002). These environmental factors affect a person's life differently and an individual's reaction to them may change over time.

Indoor air pollution: (smoke, mold, cigarette, noxious fumes, cleaning products, paints) have been found to help in the development of allergic reactions and asthma, especially in children. Exposure to certain air pollutants such as O₃, PM₁₀, CO, SO₂ and NO₂ have been linked to an increase in asthma symptoms (Agrawal, 2012;Huss, 2001;Mishra, 2002). Studies have found that a main source of indoor pollution is cooking smoke, which is the product of biomass and solid fuel combustion used in developing countries. Exposure to cooking smoke and tobacco smoke indoors increases the severity of asthma attacks, reduces the efficacy of treatments, and worsens the decline of lung functions (Agrawal, 2012;Huss, 2001;Mishra, 2002).

The most commonly research topics that are thought to be related to the onset of asthma are exposure to house dust mites, cockroach and pet-derived allergens. Exposure to mite and cockroach allergens early in life and in homes causes children to become increasingly sensitized. A study that examined the found that 88% of patients that were exposed furred or feathered pets

in the house tested positive for allergy skin tests which have been associated with asthma morbidity (Huss, 2001). In the United States, the strongest risk factors associated with the development of asthma are sensitization to dog and cat allergens (Huss, 2001). Studies have found that avoidance of allergens early in life helps prevent sensitization.

Outdoor air pollution: the two commonly described air pollutants that can affect health and are monitored by the U.S. Environmental Protection Agency (EPA) are particulate matter and ozone (Girardot, 2006;McConnell, 2002). Particulate matter are solid or liquid particles suspended in the air and can be man-made (e.g., smoke, fumes, soot, and combustion by products) or natural (e.g., windblown dust, volcanoes, agricultural crops, forest fires, pollen, and sea salt) (Brook, 2010;Dockery, 2009;Pope, 2006). The relationship between exposure to concentrated outdoor particulate matter and adverse health effects includes lung cancer, premature death, exacerbation of respiratory and cardiovascular disease (e.g., asthma and COPD), and increased risks for cardiovascular morbidity (e.g., myocardial infarction and arrhythmia) (Brook, 2010;Dockery, 2009;Pope, 2006).

Ozone, the second well-characterized air pollutant, is the most abundant photochemical oxidant (Hoppe, 2003). This atmospheric pollutant when inhaled exacerbates asthma and lessens the ability of the lungs to eliminate infectious agents and toxins (Girardot, 2006;McConnell, 2002). At ground level, ozone appears as urban smog and affects people with outdoor occupations, athletes and sensitive groups (children, elderly, asthmatics or people with other respiratory or cardiovascular disease) (Girardot, 2006;Hoppe, 2003;Triche, 2006).

Research on the topic of asthma development provides no consensus on whether environmental factors directly result in the development of asthma or only trigger asthma symptoms (Beane, 2007;Institute of Medicine, 2000;Frieden, 2011). Different approaches to prevent and treat asthma continue to be studied.

2.3 Smoking and Asthma

Tobacco smoke is one of well-known risk factors for asthma. Persons with asthma are more sensitive to the toxic chemicals and irritants that are brought about by cigarette smoke. Piipari et al reported that the risk of developing asthma was significantly higher among current smokers and among former smokers compared with those who have never smoked (OR, 1.33 and 1.49 respectively) (Piipari, 2004). Sufficient evidence supports that exposure to secondhand tobacco smoke, also known as environmental tobacco smoke (ETS) causes exacerbation of asthma symptoms among preschool age children (Institute of Medicine, 2000).

Similar to asthma the common symptoms of smoking are wheezing, coughing, shortness of breath, nasal congestion, and burning or watery eyes. Among the environmental factor of asthma, cigarette smoking and secondhand smoke are important triggers (Cerveri, 2012;Stapleton, 2011;Piipari, 2004). Tobacco smoke increases inflammation and mucus secretion in the airways which leads to worsening asthma symptoms, more airway inflammation, and reduces response to inhaled corticosteroid treatment, and an accelerated decline in lung function (Institute of Medicine, 2000;Piipari, 2004). It is well documented that exposure to

tobacco smoke or prolonged periods in asthmatic patients contributes to a decline (estimated 18%) in lung function (Institute of Medicine, 2000;Piipari, 2004).

Furthermore, studies have found that patients with asthma that smoke or are exposed to second hand smoke respond poorly to corticosteroid treatments. In cases where patients had severe asthma, increasing the dosage of inhaled or oral corticosteroids failed to suppress inflammation (Barnes, 2008). This is the result of corticosteroid resistance that is believed to be linked to impaired nuclear enzyme histone deacetylase 2 (HDAC2) functions (Barnes, 2008). As noted, the prevalence of smoking among adults has introduced additional complications to a chronic disease that continues to affect the lives of a growing population.

Tobacco smoke contains more than 4,000 toxic substances (Spira, 2004). Irritating substances settle in the moist lining of the airways (tar, carbon monoxide, nicotine, and more) causing inflammation and increasing mucus secretion (Spira, 2004). Very few studies have been done in humans to establish the effects of smoking on epithelial cells of the pulmonary airways (Spira, 2004). Spira et al. has found that cigarette smoking decreased the functionality of several tumor suppressor genes and genes that regulate airway inflammation. Another adverse outcome of smoking is that it damages cilia in the airways. Cilia are microscopic hair-like projections lining the epithelial cells that sweep dust and mucus out of the airways keeping them free from infection; a process called mucociliary clearance (Leopold, 2009). The harmful toxins present in tobacco smoke paralyzes the cilia cells allowing dust and mucus to accumulate in the airways, as a result, more mucus can build up in the airways, triggering an asthma attack (Krieger, 2001;Leopold, 2009) Leopold et al. has shown that smoking not only inhibits the movement of

cilia cells, but is also associated with the average shortening of airway epithelial cilia (by 13%) which further reduces efficacy (Krieger, 2001). Long-term smoking will result in the progressive destruction of cilia and thus a decrease in mucociliary clearance making smokers more vulnerable for developing smoking-induced lung disease. However, depending on prior smoking habits, the lungs have the ability to heal and smoking cessation allows the cilia to re-grow and resume functioning within 2 years. Studies that examined effects of smoking cessation have found that genes serving metabolizing and antioxidant functions reverted to normal levels within 2 years, while tumor suppressor genes failed to return to never smoker levels after quitting. Leopold et al. concludes that this finding may provide the rationale for ‘the continued risk for developing lung cancer many years after individuals have ceased to smoke’ (Leopold, 2009).

2.4 Smoke free Legislation- *health and economic outcomes*

Effective smoke-free legislation is among the most fundamental public health intervention measures that could reverse the adverse health effects of smoking. Research has shown that when a comprehensive smoke free law is effectively implemented, the entire population gains in terms of health benefits. Studies conducted in a number of countries (Canada, Italy, Ireland) have shown a significant reduction in the levels of people reporting exposure to second hand smoke. Also found as a result of smoke free legislation was a decline in respiratory/sensory symptoms, emergency department visits for asthma among children and adults (18% for

children, 24% for adults). Interestingly, in Ireland, emerging research has found that 1 year after implement a smoking ban in the workplace, the risk for preterm birth declined by 25% and the rate of maternal smoking decrease by 12% (Kabir, 2009).

Aside from the positive health outcomes of smoke-free legislation, the American Lung Association release a study conducted by researchers at Penn State University that analyzed the nationwide cost-benefit of smoking cessation treatments. The researchers found that smoking costs the nation a combined annual \$301 billion in lost productivity (\$67.5 billion), premature death (\$117 billion), and health care expenditure (\$116 billion) (Jill S. Rumberger, 2010). The researchers concluded that depending on the smoking cessation counseling method, the return on investment for the state could be up to 87% (Jill S. Rumberger, 2010).

Several strategies have been implemented and studied in an effort to reduce smoking prevalence. The most common intervention is a comprehensive smoke free-state wide program, banning smoking in public places, increasing the tax on cigarette sales. In Washington State, Dilley et al. examined the health effects associated with tobacco control interventions (program, policy, and price intervention for tobacco) to quantify the return on investment (Dilley, 2012). This study was the first of its kind to investigate the association between multiple specific health conditions (Ischemic heart disease hospitalizations, cerebrovascular disease hospitalizations, chronic respiratory disease hospitalizations, esophageal cancer incidence, larynx cancer incidence, oral cancer incidence, and lung cancer incidence) and multiple proven tobacco control interventions (comprehensive state program, state policy banning smoking in public places and price increases) (Dilley, 2012). The findings of the study concluded that the tobacco control

interventions worked to lower hospitalization rates and would save the state of Washington an estimated \$1.5 billion in hospitalization.

Smoke-free legislation

National Health Service (NHS) statistics analyzed by researchers in Scotland found that the introduction of smoke-free legislation resulted in a reduction to exposure to secondhand smoke among nonsmokers living in nonsmoking household (Haw, 2007). Haw et al. measured the amount of cotinine, an alkaloid found in tobacco, concentrations by collecting saliva samples from smokers and nonsmokers. Then, the researchers compared the cotinine measurements in the respondents prior to the smoke-free legislation and a year following it (Haw, 2007). The overall geometric mean cotinine for nonsmokers living in nonsmoking households fell by 49% (P-value less than 0.001). This significant reduction in exposure to secondhand smoke observed in Scotland, leads the researchers to believe that smoke-free legislations may produce immediate health gains as well as a reduce morbidity and mortality related to second-hand smoke(Haw, 2007). What the study adds to research in the topic of smoke-free legislation effects on the population is that the main beneficiaries of the legislation are non-smokers living in non-smoking homes as exposure to secondhand smoke was reduced in all public places and workplaces but not in homes and private cars.

Cigarette tax

In the ongoing debate to lower smoking prevalence, cigarette tax is the most commonly advocated means to reduce smoking. Legislative interventions to increase the price of cigarettes have been found to discourage young adults from starting smoking as well as reducing the

average cigarette consumption among current smokers (Services, 2005; CDC, 2000; Medicine, 2007) In 2009, the United States placed a 15% federal tax increase on the average pack of cigarette price with a proposed additional average increase of 34%. Researchers at the University of Illinois at Chicago measured the immediate impact of excise tax increase on youth through May 2009 by using the Monitoring the Future survey, a national representative survey of 8th, 10th, and 12th grade students (Huang, 2012). Huang et al. found that the 62 cent-a-pack increase resulted in an estimated average drop of 11.5%.

Martire et al. performed a study in Australia that compares the possible impacts of proposed price increases on smoking prevalence in both the United States (U.S.) and Australia. The researchers examine the effects of the proposed tax increases (U.S. - 34%, Australia- 49%) as well as the probable effect of financial stress (Martire, 2011). The findings of the paper suggest that the increase in tobacco tax will potentially lower the prevalence in both countries. Implementation of tax increases was found to decrease smoking in lower socioeconomic status groups by 2% in the U.S. and 8% in Australia. However, by taking into account the effects of financial stress, Martire et al. found that smoking prevalence was reduced by 0.13% in the U.S. and 0.36% in Australia (Martire, 2011). Therefore, the researchers believe that instead of tacking this issue from one standpoint of taxing cigarettes, instead ‘policy makers need to interrupt the negatively reinforcing cycle of smoking and financial stress’ by ‘providing free or subsidized smoking cessation resources to reduce the economic burdens borne by financially stressed smokers and facilitate quitting.’ (Martire, 2011, p.628)

CHAPTER III

METHODOLOGY

3.1 Data Source

The Centers for Disease Control and Prevention's 2009–2010 Behavioral Risk Factor Surveillance System (BRFSS) data was used for the analyses. SAS-callable SUDAAN (version 10.0.0, RTI International, NC) was utilized to account for the complex sampling design of the BRFSS and sample weights were used to produce estimates that were generalizable to the state and U.S. adult population.

BRFSS is a joint partnership project with the Centers of Disease Control and Prevention (CDC) and U.S. states and territories (William J. Curry, 2010). BRFSS is an ongoing state-based, random-digit dialed telephone survey of the non-institutionalized U.S. population, aged 18 years of older. The main purpose of the BRFSS is to gather uniform and state-specific data on preventive health practices and risk behaviors that are linked to chronic diseases, injuries, and preventable infectious diseases in the adult population. The data collection process of the BRFSS is administered by state health departments with guidelines provided by CDC. The 2009 and 2010 BRFSS was conducted in the 50 states, the District of Columbia (DC), Puerto Rico (PR), the U.S. Virgin Islands, Guam, American Samoa, and Palau (CDC, 2011). However, we limited our analysis to 50 states and, the District of Columbia (DC). This survey is the world's largest ongoing survey with over half a million interviews conducted annually.

In order to have a representative sample of the population, BRFSS uses a statistical method called post stratification (CDC, 2011). In post stratification, after data has been collected, data is compare it to an auxiliary data set representation of the population to make sure that the

distributions of demographic characteristics (age, race and ethnicity, sex, geographic region within a population.) are within a few percentage points (Gelman, 2000). If the collected data differ by more than the accepted range of percentage points, the sample data is adjust to conform to the population's parameters.

BRFSS measures personal behaviors that put an individual's health at risk. To determine current cigarette smoking, respondents are asked, "Have you ever smoked at least 100 cigarettes in your entire life?" and "Do you now smoke cigarettes every day, some days, or not at all?" Current smokers are defined as those who reported having smoked >100 cigarettes during their lives and who currently smoked every day or some days (CDC, 2011). Because BRFSS data is state-specific, median values rather than a national average are reported. Topics included in the BRFSS questionnaire that has to do with demographic data include: age, sex, ethnicity, race, marital status, education level, employment status, income, county of residence, pregnancy status and children less than 18 years in household (CDC, 2011).

Response rate is an outcome rate with the number of complete and partial interviews in the numerator and an estimate of the number of eligible units in the sample in the denominator. To account for respondents accurately, the BRFSS uses the Council of American Survey Research Organizations (CASRO) rates (CDC, 2011). Response Rate calculation assumes that the unresolved numbers contain the same percentage of eligible households as the records whose eligibility or ineligibility are determined. Therefore, the BRFSS uses proportions of unknown households in each of the states to estimate the total number of households from those whose eligibility is undetermined (CDC, 2011) In 2009, the median response rate was 52.48% (does

not include Guam, Puerto Rico or the Virgin Islands) and in 2010 the median response rate was 54.60% (CDC, 2011).

To assess smoke-free policies, we used the American Lung Association State of Tobacco Control 2008 report that follows state and federal policy advancements on important tobacco control policies and allocates grades to tobacco control laws and regulation enacted as of January 1, 2009. The ALA State of Tobacco Control accounts for laws enacted by January of 2009, so when individuals were surveyed in 2009 and 2010 and asked about their smoking status their answers were dependent on the effects of existing laws, which was put into effect in the prior year. The federal government along with all 50 states and the District of Columbia are evaluated and graded to establish whether or not tobacco control laws are effective in protecting citizen's health against tobacco use and reducing the economic cost of tobacco on society (CDC, 2012). The American Lung Association State of Tobacco Control report evaluates both state and federal policies in four main areas: smoke-free air, tobacco prevention and control funding, cigarette taxes and cessation coverage (Association, 2012). The calculation of state grades for tobacco prevention and control spending is based on the Centers for Disease Control and Prevention's (CDC) published Best Practices for Comprehensive Tobacco Control Programs (Association, 2012). The smoke-free air laws grading is based on criteria developed by an advisory committee convened by the National Cancer Institute with some modification to reflect the current policy environment. The grading for state cigarette excise tax is based on the average of all state taxes; the grades are adjusted annually to reflect the change in the average tax increase. Finally, the state cessation coverage grading system is based on the U.S. Department of Health

and Human Services' published Clinical Practice Guideline on Treating Tobacco Use and Dependence.

3.2 Study Measures

Consistent with previous CDC reports, two asthma prevalence questions are being used by BRFSS as a measure of asthma data. First, an affirmative response to the question "Have you ever been told by a doctor {nurse or other health professional} that you have asthma?" defines a status of lifetime asthma (CDC, 2011). Second, current asthma is defined as an affirmative response to the previous question followed by an affirmative response to the following question, "So you still have asthma?" Responses to these two questions are tabulated for adults for various demographic groups.

To provide more meaningful results that are specific to unique groups, the analyses were stratified related to current health conditions (asthma) and risk factors according to the following characteristics: gender, age group, race, education, income and smoking status. We categorized gender into two groups (male/female); age into four groups (18-34/35-44/45-64/65+); race into six groups (White/Black/American Indian/Alaskan Native/Hispanic/Other race); education into three groups (high school education or less/some college/college 4 years or more); income into five groups (<15000/15000-24999/25000-49999/50000-74999/≥7500); and smoking into three groups (current smoker/former smoker/ non-smoker) (Table 1). These categories were chosen because of the assurance of a reasonable sample size in each category. The data were analyzed specifically for adults with asthma and adults without asthma.

The American Lung Association State of Tobacco Control report evaluates both state and federal policies by comparing them against targets based on the most current, recognized criteria for effective tobacco control measures, and translating each state's relative progress into a letter grade of A through F (CDC, 2012). A grade of "A" is assigned for excellent tobacco control policies while an "F" indicates inadequate policies. The American Lung Association (ALA) tracks four components to grade states:

1. Tobacco Prevention and Control Spending
2. Smokefree Air
3. Cigarette Excise Tax
4. Cessation Coverage

For this study we used three of the four components because 'Tobacco Prevention and Control Spending' did not change much among states. To examine the association between state of tobacco control efforts and smoking rate among those with asthma, we summed for each of the three components for each state to obtain overall policy grade for each state as shown in Table 5. The overall policy grade was used because it accounts for the cumulative effects of all three important laws selected:

1. Smoke-free air for its effects on reducing secondhand smoke in homes, work or restaurants,
 2. Increased cigarette excise tax for discouraging people who smoke as well as reducing exposure to secondhand smoke.
-

3. Smoking cessation programs for encourage people who smoke to quite will improve smokers' health and also will reduce exposure to secondhand smoke. As evident in literature, the adverse health outcomes of smoking such as worsening asthma symptoms, emphysema, chronic bronchitis, and risk of cancer can be substantially reduced the sooner individuals quit smoking.

3.3 Analysis

The BRFSS dataset was obtained electronically and downloaded into SAS-callable SUDAAN (version 10.0.0, RTI International, NC) for analysis. Alpha levels of <0.05 was used for all statistical significance testing. Multivariate analyses were performed to reveal descriptive statics regarding the study population. These analyses categorized and identified frequencies and central tendencies around age distribution, race/ethnicity, education level of respondents, income as well as smoking status (Table 2).

Multivariate logistic regression (dependent variable is Asthma- Yes/No) analysis was also used to examine the association between demographic characteristics (age, sex, race, ethnicity, education, income), smoking status, and asthma status (Table 2). We performed a similar analysis to examine the association between smoking status and selected survey respondents characteristics by asthma status and presented the results in Table 3. Percentage of smokers among adults with and without asthma was estimated for each state by, adjusting for sex, age, and race and results are presented in Table 4.

We tested for and found no multicollinearity among independent variables. For instance, the two independent variables, education and income, did not have a high correlation. P-value of

0.05 or less and not overlapping confidence interval (Gelman, 2000) are considered to be statistically significant.

For the purposes of this analysis, demographic variables were re-coded in a variety of ways that differed from the original coding of the data. The recoding structure is illustrated in Table 1. Specifically, age, race, education level, household income, and smoking status were re-coded to form more condensed and representative groups found within the study population.

Table 1 *Initial and Re-coded Demographic Information*

Characteristics	Initial Coding	Re-coded
Age	18-34- 18-24 25-34- 25-34 35-44- 35-44 45-54- 45-54 54-64- 54-64 64-99- 64 or older BLANK- Not asked or missing	1- 18-34 2- 35-44 3- 45-54 4- 55-64 5- 65+
Race	1- White 2- Black or African American 3- Asian 4- Native Hawaiian or Other Pacific Islander 5- American Indian, Alaska Native 6- Other 7- Don't know/Not sure 8- Multiracial but preferred race not asked 9- Refused BLANK-Not asked or missing	1- White 2- Black 3- AI/AN 4- Asian/Pacific Islander 5- Hispanic 6- Other
Hispanic/Latino	1- Yes 2- No 7- Don't know/Not sure 9- Refused	

Education level	1- Never attended school or only kindergarten 2- Grades 1 through 8 (Elementary) 3- Grades 9 through 11 (Some high school) 4- Grades 12 or GED (High school graduate) 5- College 1 year to 3 years (Some college or technical school) 6- College 4 years or more (College graduate) 9- Refused BLANK- Not asked or Missing	1- High School (HS) graduate or less 2- Some college 3- College 4 years or more
Household income	1- Less than \$10,000 2- Less than \$15,000 (\$10,000 to less than \$15,000) 3- Less than \$20,000 (\$15,000 to less than \$20,000) 4- Less than \$25,000 (\$20,000 to less than \$25,000) 5- Less than \$35,000 (\$25,000 to less than \$35,000) 6- Less than \$50,000 (\$35,000 to less than \$50,000) 7- Less than \$75,000 (\$50,000 to less than \$75,000) 8- \$75,000 or more 77- Don't know/Not sure 99- Refused BLANK- Not asked or Missing	1- <15,000 2- 15,000-24,999 3- 25,000-49,999 4- 50,000-74,999 5- ≥75,000
Smoking status	1- Current smoker- now smokes every day 2- Current smoker- now smokes some days 3- Former smoker 4- Never smoked 9- Don't know/ Refused/ Missing	1- Current smoker 2- Former smoker 3- Non-smoker
Asthma status	Lifetime Asthma:	Combined:

	1- No 2- Yes 9- Don't know/Refused/ Missing	1- No 2- Yes
	Current Asthma: 1- No 2- Yes 9-Don't know/Refused/ Missing	

CHAPTER IV

RESULTS

Results from the BRFSS dataset and ALA analysis are explained below. The results presented address the three research questions posed at the onset of the study and outlined in chapter one of this paper.

4.1 Characteristics of survey respondents

After recoding the demographic variables from initial categories, frequency statistics were run on the following demographic markers: gender, age, race, highest level of education and income and on smoking status. Table 2 presents the overall demographic characteristics of the survey participants, as well as the current asthma prevalence by demographic and socioeconomic variables. For the period 2009-2010, a total of 869,519 respondents from 50 states and the District of Columbia (DC) were included in the analyses. Of those, 51% were female, mostly white (69%), 35% had an education level that was equivalent to a 4 year college or more, and nearly 18% were smokers.

4.2 Asthma prevalence and factors associated with asthma

Consistent with the previous CDC reports, our findings indicate that asthma status is significantly associated with all selected demographic and socioeconomic characteristics of the survey respondents (Table 2). The p-values for the chi-square test for independence to determine whether there is a significant relationship between two categorical variables was less than 0.0001 in all the categories analyzed. Results of frequency statistics are further depicted in Table 2.

Average annual current asthma prevalence was 8.5%. Consistent with the previous CDC reports, asthma prevalence rate was higher in females than males (10.5% compared with 6.4%). The current asthma prevalence for each age group were significantly higher than the reference age group (aged 65+ years), however the highest was among adults aged 18-34 (9.3%; adjusted prevalence rate ratio [aPR]=1.4) (Table 2). Among race/ethnic groups, current asthma prevalence was high among other races (13.5%), followed by American Indian/ Alaskan Native (13.0%), Black (10.0%), White (8.6%), Hispanic (6.7%), and finally Asian (4.8%). Also, persons with some college degree (9.3%; [aPR]=1.1) and those in the lowest income bracket (13.3%; [aPR]=1.9) had the highest current asthma prevalence in their respective categories. Smoking status is significantly associated with current asthma status. Asthma prevalence was higher among both current (10.5%; [aPR]=1.2) and former smokers (8.8%; [aPR]=1.2) compared with non-smokers (7.8%; [aPR]=1.0).

Table 2 Characteristics of adult survey respondents— 2009–2010 Behavioral Risk Factor Surveillance System data

	Total	Characteristics of Survey Respondents	Current Asthma Prevalence¹	Prevalence Rate Ratio
	Sample size ²	% (95% CI)	% (95% CI)	aPR (95% CI) ³
Total	869519	100 (0–0)	8.5 (8.4–8.6)	
Sex			$p < 0.0001$ ⁴	
Male	329080	48.7 (48.5–48.9)	6.4 (6.3–6.6)	1.0 (reference)
Female	540439	51.3 (51.1–51.5)	10.5 (10.3–10.7)	1.6 (1.6–1.7)
Age, year range			$p < 0.0001$	

18–34	95841	28.2 (28.0–28.5)	9.3 (9.0–9.6)	1.4 (1.3–1.4)
35–44	116615	20.2 (20.1–20.4)	7.8 (7.6–8.1)	1.2 (1.2–1.3)
45–54	170300	19.5 (19.3–19.6)	8.4 (8.2–8.7)	1.3 (1.2–1.3)
55–64	194791	14.7 (14.6–14.8)	9.0 (8.8–9.2)	1.3 (1.2–1.3)
65+	284209	17.4 (17.2–17.5)	7.8 (7.6–7.9)	1.0 (reference)
Race(non-Hispanic)/ Ethnicity			<i>p</i> <0.0001	
White	690022	69.2 (69.0–69.4)	8.6 (8.5–8.8)	1.0 (reference)
Black	68024	10.2 (10.0–10.3)	10.0 (9.6–10.5)	1.0 (1.0–1.1)
AI/AN⁵	11961	1.1 (1.0–1.1)	13.0 (11.7–14.5)	1.3 (1.2–1.5)
Asian/Pacific Islander	15899	3.8 (3.7–3.9)	4.8 (4.2–5.5)	0.6 (0.5–0.7)
Hispanic	53732	13.6 (13.4–13.8)	6.7 (6.3–7.1)	0.7 (0.6–0.7)
Other race	18973	2.2 (2.1–2.3)	13.5 (12.5–14.5)	1.4 (1.3–1.6)
Education level			<i>p</i> <0.0001	
High School (HS) graduate or less	340602	38.4 (38.2–38.6)	9.1 (8.9–9.3)	1.0 (1.0–1.1)
Some college	232036	26.3 (26.1–26.5)	9.3 (9.1–9.6)	1.1 (1.1–1.1)
College 4 years or more	293834	35.3 (35.1–35.5)	7.3 (7.1–7.5)	1.0 (reference)
Household Income			<i>p</i> <0.0001	

<\$15000	86389	10.4 (10.2–10.5)	13.3 (12.8–13.8)	1.9 (1.8–2.0)
\$15000– \$24999	133940	15.8 (15.6–16.0)	10.3 (9.9–10.6)	1.5 (1.4–1.6)
\$25,000– \$49999	205802	24.6 (24.4–24.8)	8.1 (7.9–8.4)	1.2 (1.1–1.2)
\$50000– \$74999	120544	16.0 (15.9–16.2)	7.7 (7.4–8.0)	1.1 (1.0–1.2)
≥\$75000	202340	33.2 (32.9–33.4)	6.8 (6.6–7.0)	1.0 (reference)
Smoking status			<i>p</i> <0.0001	
Current smoker	139086	17.6 (17.4–17.8)	10.5 (10.2–10.9)	1.2 (1.2–1.3)
Former smoker	263614	24.9 (24.7–25.1)	8.8 (8.6–9.0)	1.2 (1.2–1.2)
Non-smoker	461190	57.5 (57.3–57.7)	7.8 (7.7–8.0)	1.0 (reference)

¹Includes persons who answered “yes” to the questions: “Have you ever been told by a doctor, nurse, or other health professional that you had asthma?” and “Do you still have asthma?”

²Sample size (unweighted) for the corresponding subpopulations

³Prevalence rate ratio (95% confidence interval) adjusted for age, sex, race, ethnicity, education, income, smoking.

⁴P-values for the chi-square test for independence to determine whether there is a significant relationship between two categorical variables.

⁵American Indian/ Alaskan Native

4.3 Smoking rate by selected characteristics among adults with and without asthma

Following the establishment of baseline demographic characteristics of BRFSS respondents and asthma status among them, we further examined smoking rate among those with and without asthma and identify the factors associated with high smoking rate (Table 3). Cross comparison between adults with asthma and adults without asthma, smoking rate was higher among those with asthma (21.7%) than those without asthma (17.2%). Comparison between the

different sub-group within adults with asthma and adults without asthma indicate that same demographic characteristics have the high smoking rate. Regardless of asthma status, smoking rates are higher among males than females, ages 18-34, 35-44, 45-54, 55-64 than ages 65+; American Indian/Alaska Native (AI/AN) and other race than whites; adults with high school or less education and adults with some college education than adults with 4 years or more college education, and among all adults with income of less than \$75,000 than adults with higher income (\geq \$75,000) (Table 3). Worth noting that, smoking rates are lower among blacks, Asian/Pacific Islander, and Hispanic for both adults with asthma and without asthma, compared with whites. For example, percentage of smoking was 23.4% for adult males with asthma and 19.3% for adult males without asthma. Both rate for males were higher than rates for females (20.7% vs. 15.1%) (Table 3).

Table 3 *Percentage of smokers by asthma¹ status and selected demographic characteristics—2009–2010 Behavioral Risk Factor Surveillance System data*

	Adults with asthma (n=78,159)		Adults without asthma (n=783,417)	
	Percentage of smokers (n=15,597)		Percentage of smokers (n=123,090)	
	% (95% CI)	aPR (95% CI) ²	% (95% CI)	aPR (95% CI)
Total	21.7 (21.1-22.4)		17.2 (17.0-17.4)	
Sex				
Male	23.4 (22.2-24.7)	1.0 (reference)	19.3 (19.0-19.6)	1.0 (reference)
Female	20.7 (20.0-21.4)	0.9 (0.8-0.9)	15.1 (14.9-15.3)	0.8 (0.8-0.8)
Age, year range				

18–34	26.1 (24.5-27.7)	3.0 (2.8-3.3)	21.5 (21.1-22.0)	3.1 (3.0-3.2)
35–44	22.9 (21.5-24.4)	3.2 (2.9-3.5)	17.7 (17.3-18.1)	3.0 (2.9-3.2)
45–54	25.1 (23.9-26.3)	3.2 (2.9-3.4)	19.6 (19.2-19.9)	3.1 (3.0-3.2)
55–64	19.6 (18.6-20.7)	2.4 (2.2-2.6)	16.2 (15.9-16.5)	2.5 (2.4-2.5)
65+	9.9 (9.3-10.6)	1.0 (reference)	8.2 (8.0-8.4)	1.0 (reference)
Race(non-Hispanic)/ Ethnicity				
White	21.4 (20.7-22.1)	1.0 (reference)	17.5 (17.3-17.7)	1.0 (reference)
Black	22.9 (20.9-25.0)	0.8 (0.7-0.9)	19.2 (18.6-19.9)	0.8 (0.8-0.9)
AI/AN³	40.4 (34.7-46.4)	1.4 (1.2-1.6)	31.9 (29.7-34.2)	1.3 (1.2-1.4)
Asian/Pacific Islander	9.7 (6.2-14.8)	0.6 (0.4-0.9)	9.4 (8.3-10.6)	0.6 (0.5-0.7)
Hispanic	19.2 (17.0-21.7)	0.6 (0.5-0.7)	14.4 (13.7-15.1)	0.5 (0.5-0.5)
Other race	31.1 (27.5-34.9)	1.1 (1.0-1.3)	24.8 (23.3-26.4)	1.2 (1.1-1.2)
Education level				
High School (HS) graduate or less	31.3 (30.1-32.4)	2.6 (2.3-2.8)	24.4 (24.1-24.8)	2.5 (2.4-2.5)
Some college	22.2 (21.0-23.4)	2.0 (1.8-2.2)	19.1 (18.7-19.5)	2.0 (1.9-2.0)
College 4 years or more	8.3 (7.6-9.0)	1.0 (reference)	8.2 (8.0-8.5)	1.0 (reference)
Household Income				
<\$15000	36.7 (34.8-38.7)	3.1 (2.8-3.5)	27.2 (26.4-27.9)	2.3 (2.2-2.4)
\$15000–\$24999	31.8 (30.1-33.6)	2.9 (2.5-3.2)	25.4 (24.8-26.0)	2.2 (2.1-2.2)

\$25,000–\$49999	22.6 (21.2-24.1)	2.2 (1.9-2.5)	20.0 (19.6-20.5)	1.7 (1.6-1.8)
\$50000–\$74999	14.7 (13.3-16.1)	1.5 (1.3-1.7)	15.3 (14.8-15.7)	1.3 (1.3-1.4)
≥\$75000	8.4 (7.6-9.3)	1.0 (reference)	10.3 (10.0-10.5)	1.0 (reference)

¹Includes persons who answered “yes” to the questions: “Have you ever been told by a doctor, nurse, or other health professional that you had asthma?” and “Do you still have asthma?”

²Prevalence rate ratio (95% confidence interval) adjusted for age, sex, race, ethnicity, education, income, smoking.

³American Indian/ Alaskan Native

4.4 Adjusted smoking rate by asthma status for each state

After assessing the asthma and smoking status by demographic and socioeconomic variables, the percentage of smokers among adults by asthma status and state was examined. A sample size of 869,519 respondents was collected and the percent smokers for each state was estimated by adjusting for sex, age and race. Overall, the analysis of all 50 states shows that the percentage of adult smokers who have asthma is significantly higher than the percentage of adult smokers without asthma. The U.S. total includes all 50 states plus DC and excludes the three territories (Guam, Virgin Island, and Puerto Rico). Of all the states, 24 were found to have higher rates of adults with asthma who smoke than adults without asthma who smoke. The states that were found to have higher rates of smoking adults with asthma are indicated with an asterisk in the ‘Adults with asthma who smoke’ percentage column (Table 4). Among states, the lowest percentage of smokers among adults with asthma was in Utah at 11.2% . Of the 50 states, Alabama, Arkansas, Indiana, Kentucky, Louisiana, Ohio, Oklahoma, Mississippi, South Carolina, Tennessee, and West Virginia had the highest smoking rate as seen in Map as top 20%. Arkansas (+9.1%), Kentucky (+9.6%), Mississippi (+7.6%), South Carolina (+8.3%), and

Tennessee (+9.0%) had the largest positive percentage difference between adults with asthma who smoke and adults without asthma who smoke (Table 4).

Table 4 Percentage of smokers among adults by asthma¹ status and states— 2009–2010
Behavioral Risk Factor Surveillance System data

	Total			Adults <u>with</u> asthma who smokes (n=15,597)		Adults <u>without</u> asthma who smokes (n=123,090)	
	Sample size ²	% ³	(95% CI) ³	% ³	(95% CI) ³	% ³	(95% CI) ³
U.S. Total⁴	869519			*21.4	(20.8-22.1)	17.3	(17.1-17.5)
AL	14457	22.1	(21.0-23.2)	26.2	(22.6-30.1)	22.0	(20.9-23.2)
AK	4396	17.2	(15.5-19.0)	23.4	(16.7-31.8)	20.1	(18.2-22.2)
AZ	11231	15.7	(14.4-17.1)	16.3	(12.5-20.9)	15.6	(14.2-17.1)
AR	8036	21.8	(20.3-23.4)	*30.6	(25.1-36.8)	21.5	(20.0-23.2)
CA	35170	13.3	(12.7-13.9)	*16.2	(14.2-18.4)	12.2	(11.6-12.7)
CO	23619	16.2	(15.4-17.0)	15.4	(12.8-18.4)	16.6	(15.8-17.5)
CT	13272	14.5	(13.5-15.5)	16.1	(12.8-20.1)	14.2	(13.2-15.4)
DE	8607	17.7	(16.5-19.0)	*23.2	(18.8-28.2)	17.3	(16.0-18.7)
DC	7880	14.9	(13.8-16.1)	15.2	(12.1-18.9)	15.6	(14.4-16.9)
FL	47164	18.1	(17.3-19.0)	19.6	(16.8-22.8)	17.0	(16.1-17.9)
GA	11684	17.0	(16.0-18.2)	21.2	(17.0-26.1)	17.5	(16.3-18.7)
HI	13235	16.5	(15.4-17.8)	16.6	(13.8-19.8)	14.8	(13.8-15.8)
ID	12399	15.5	(14.5-16.5)	17.8	(14.7-21.4)	15.9	(14.9-17.0)

IL	11047	17.8	(16.7-18.9)	18.5	(14.9-22.6)	17.6	(16.5-18.8)
IN	19507	21.6	(20.7-22.6)	*26.9	(23.9-30.2)	21.8	(20.8-22.8)
IA	12126	16.5	(15.5-17.5)	*22.4	(18.8-26.4)	16.2	(15.2-17.2)
KS	27482	17.0	(16.3-17.7)	*21.5	(18.8-24.4)	17.0	(16.3-17.8)
KY	17713	24.6	(23.4-25.8)	*34.0	(30.2-38.2)	24.4	(23.1-25.7)
LA	15914	21.1	(20.1-22.1)	25.8	(21.5-30.7)	21.8	(20.8-22.8)
ME	16214	17.7	(16.8-18.6)	20.8	(18.1-23.6)	17.5	(16.6-18.4)
MD	17776	15.1	(14.2-16.0)	*20.1	(16.7-24.0)	14.9	(14.0-15.8)
MA	33042	14.7	(14.0-15.3)	*18.9	(16.8-21.2)	14.0	(13.3-14.7)
MI	18118	18.8	(18.0-19.7)	21.0	(18.3-24.0)	19.1	(18.2-20.1)
MN	14579	15.3	(14.4-16.4)	16.1	(12.6-20.4)	15.8	(14.8-16.9)
MS	19283	22.3	(21.3-23.3)	*30.3	(26.8-34.0)	22.7	(21.7-23.8)
MO	10486	21.4	(20.2-22.8)	25.2	(20.6-30.5)	21.8	(20.5-23.2)
MT	14922	16.9	(15.9-18.0)	*24.1	(20.2-28.5)	17.3	(16.2-18.5)
NE	32377	16.6	(15.6-17.6)	17.5	(14.2-21.3)	17.0	(16.0-18.1)
NV	7753	21.4	(19.8-23.2)	23.0	(18.0-28.8)	21.6	(19.8-23.4)
NH	12035	15.9	(14.9-16.9)	17.3	(14.6-20.5)	16.1	(15.0-17.2)
NJ	24828	15.7	(15.0-16.5)	16.0	(13.7-18.5)	15.1	(14.3-15.9)
NM	15834	18.2	(17.2-19.4)	20.9	(17.3-25.1)	18.0	(16.9-19.1)
NY	15877	17.3	(16.5-18.3)	*21.6	(18.7-24.8)	16.4	(15.4-17.3)
NC	25416	19.5	(18.6-20.4)	23.5	(20.5-26.9)	19.8	(18.9-20.8)
ND	9530	16.9	(15.8-18.0)	20.6	(16.8-25.0)	17.7	(16.5-19.0)

OH	19631	21.0	(20.1-21.9)	*26.5	(23.2-30.0)	20.9	(20.0-21.9)
OK	15590	22.7	(21.7-23.6)	*29.9	(26.7-33.4)	24.0	(23.0-25.0)
OR	9360	15.7	(14.6-17.0)	*23.4	(19.2-28.1)	15.8	(14.6-17.1)
PA	20415	19.6	(18.7-20.4)	*24.2	(21.4-27.2)	18.8	(17.9-19.7)
RI	12895	15.3	(14.3-16.3)	16.0	(13.2-19.3)	15.4	(14.4-16.5)
SC	19293	20.0	(18.9-21.2)	*28.4	(23.5-34.0)	20.1	(18.9-21.3)
SD	13552	15.4	(14.4-16.4)	17.6	(14.4-21.2)	16.4	(15.3-17.5)
TN	11346	20.8	(19.6-22.1)	*29.5	(24.6-35.0)	20.5	(19.2-21.8)
TX	29685	17.4	(16.5-18.4)	*22.2	(19.0-25.8)	16.6	(15.7-17.6)
UT	20334	08.8	(08.2-09.4)	11.2	(9.2-13.6)	9.3	(8.7-10.0)
VT	13462	15.9	(15.0-16.8)	*21.3	(18.3-24.7)	15.6	(14.7-16.6)
VA	10576	18.5	(16.9-20.2)	24.2	(19.7-29.5)	18.4	(16.7-20.2)
WA	39922	14.7	(14.1-15.2)	*18.6	(16.6-20.7)	14.7	(14.1-15.3)
WV	9218	26.1	(24.9-27.3)	*32.8	(28.5-37.4)	25.6	(24.3-26.9)
WI	9334	18.3	(17.0-19.8)	*25.0	(20.1-30.6)	18.3	(16.9-19.9)
WY	11897	18.9	(17.9-20.0)	21.1	(17.7-25.0)	19.7	(18.5-20.8)

*Percentage of smokers among adults with asthma was greater than the percentage of smokers among adults who does not have asthma

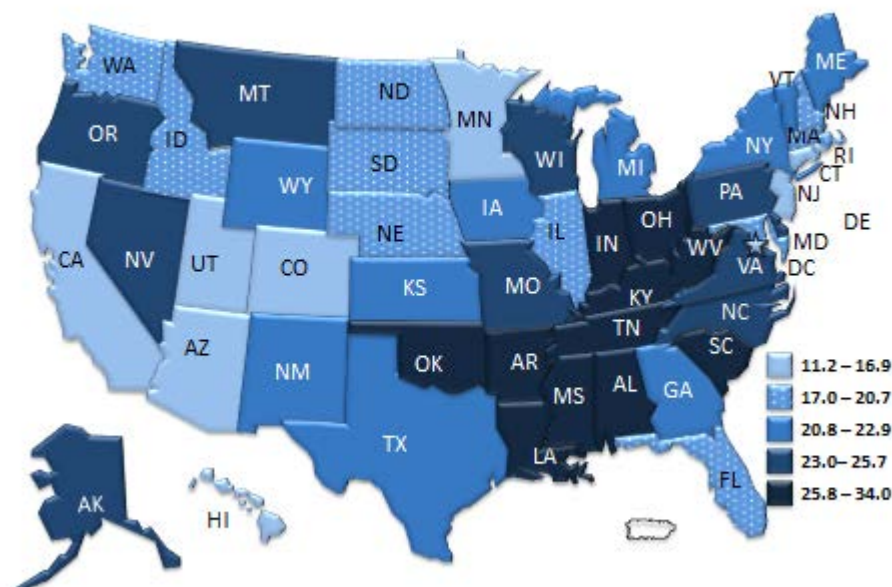
¹Includes persons who answered “yes” to the questions: “Have you ever been told by a doctor, nurse, or other health professional that you had asthma?” and “Do you still have asthma?”

²Sample size (unweighted) for the corresponding subpopulations

³Adjusted for sex, age, and race/ethnicity

⁴U.S. Total includes 50 states plus DC and excludes the three territories

Map1. Percentage of smokers among adults with asthma by state



4.5 Adjusted smoking rate among adults with asthma and level of tobacco control by states

After examining the percentage of smokers among adults by asthma status and state, the smoking rate among adults with asthma as it relates to different measure of tobacco control was analyzed by state. Data from the ALA 2008 report that tracks progress on key tobacco control policies at the state level were used to create Table 5. In Table 5, for each state, we presented adjusted (by age, sex, and race) smoking prevalence rate among adults with asthma and data from the ALA 2008 report on the three policies- smoke-free air (total score, grade), cigarette excise tax (tax rate per pack of 20 cigarettes, grade), and cessation coverage (total score, grade). We calculated, the ‘Overall Policy Grade’ column, which is the cumulative grade of all policies implemented by using the scores for grades as A=4, B=3, C=2, D=1, F=0. Tables 6 and 7 take a

closer look at the states with the highest and lowest smoking rates for adults with asthma and overall smoke-free policy grade as indication of state of tobacco control efforts to examine the association between them.

Table 5 *Percentage of adults with asthma who smoke and level of tobacco control by state.*

		Level of tobacco control						
		Smokefree Air Grading ¹		Cigarette Excise Tax overview ¹		Cessation Coverage Grading ¹		Overall Policy Grade
State	% Adults with asthma who smoke ²	Total Score	Grade	Tax Rate (per pk.of 20)	Grade	Total Score	Grade	
AL	26.2	20	F	\$0.425	F	17	F	0
AK	23.4	21	F	\$2.00	B	19	F	3
AZ	16.3	46	A	\$2.00	B	26	D	8
AR*	30.6	36	B	\$0.59	F	28	D	4
CA	16.2	40	A	\$0.87	D	28	D	6
CO	15.4	40	A	\$0.84	D	21	F	5
CT	16.1	39	C	\$2.00	B	5	F	5
DE*	23.2	46	A	\$1.15	D	15	F	5
DC	15.2	36	A	\$2.00	B	28	D	8
FL	19.6	41	B	\$0.339	F	21	F	3
GA	21.2	31	C	\$0.37	F	3	F	2
HI	16.6	43	A	\$2.00	B	21	F	7

ID	17.8	36	B	\$0.57	F	21	F	3
IL	18.5	48	A	\$0.98	D	29	C	7
IN*	26.9	7	F	\$0.995	D	30	C	3
IA*	22.4	42	A	\$1.36	C	18	F	6
KS*	21.5	16	F	\$0.79	D	18	F	1
KY*	34	3	F	\$0.30	F	18	F	0
LA	25.8	36	B	\$0.36	F	16	F	3
ME	20.8	42	A	2.00	B	29	C	9
MD*	20.1	42	A	\$2.00	B	14	F	7
MA*	18.9	42	A	\$2.51	A	29	C	10
MI	21	17	F	\$2.00	B	21	F	3
MN	16.1	41	A	\$1.504	C	35	B	9
MS*	30.3	11	F	\$0.18	F	25	D	1
MO	25.2	16	F	\$0.17	F	4	F	0
MT*	24.1	35	I ³	\$1.70	C	18	F	2
NE	17.5	18	I ⁴	\$0.64	D	30	C	3
NV	23	37	B	\$0.80	D	32	C	6
NH	17.3	33	D	\$1.33	C	22	F	3
NJ	16	41	A	\$2.575	A	24	F	8
NM	20.9	40	A	\$0.91	D	33	B	8
NY*	21.6	43	A	\$2.75	A	24	F	8
NC	23.5	6	F	\$0.35	F	24	F	0

ND	20.6	32	C	\$0.44	F	33	B	5
OH*	26.5	44	A	\$1.25	C	23	F	6
OK*	29.9	34	D	\$1.03	D	24	F	2
OR*	23.4	45	A	\$1.18	D	34	B	8
PA*	24.2	36	C	\$1.35	C	32	C	6
RI	16	41	A	\$2.46	A	36	B	11
SC*	28.4	10	F	\$0.07	F	19	F	0
SD	17.6	30	F	\$1.53	C	15	F	2
TN*	29.5	34	C	\$0.62	D	6	F	3
TX*	22.2	9	F	\$1.41	C	18	F	2
UT	11.2	41	A	\$0.695	D	21	F	5
VT*	21.3	36	A	\$1.99	B	22	F	7
VA	24.2	13	F	\$0.30	F	23	F	0
WA*	18.6	47	A	\$2.025	B	24	F	7
WV*	32.8	7	F	\$0.55	F	21	F	0
WI*	25	16	F	\$1.77	C	33	B	5
WY	21.1	0	F	\$0.60	D	19	F	1

*Percentage of smokers among adults with asthma was greater than the percentage of smokers among adults who does not have asthma

¹SOURCE: American Lung Association. State of Tobacco Control 2008 <http://www.lung.org/about-us/publications/index.html#presidents-report>

²Adjusted for sex, age, and race/ethnicity

³Montana gets an "I" for Incomplete because they passed a smokefree law in 2005, but parts of it were delayed from taking effect until October 1, 2009.

⁴Nebraska gets an "I" for Incomplete because they passed a smokefree law in 2008, but it does not take effect until June 1, 2009.

4.5 Evaluating the association between the level of tobacco control and smoking rate among adults with asthma by states

We examined the association between the smoking rate among adults with asthma and measure of state of tobacco control efforts among states. Smoking rates for states were divided into five quintiles and overall policy grade, mean, minimum, and maximum values of overall tobacco control policy grade for states in each 5 quintile were calculated (Table 6). To show the impact that tobacco control had on smoking rate, we performed t-test to examine if mean tobacco control policy grades for states in quintile 1(Q1) to Q4 were significantly different than the mean tobacco control policy grade for states with the highest smoking rate (Q5=highest 20%). The mean tobacco control policy grade was calculated based on the three tobacco control laws. We assigned a grade point by giving 4 points for grade A, 3 points for B, 2 points for C, 1 point for D, and 0 points for F. Then the points for the 3 components were summed to derive the total points (overall policy grade) for each state. The total points ranges from zero to 12. The higher the overall policy grade, the better the tobacco control effort in the state. The mean tobacco control policy grade for states in Q1, Q2, and Q3 groups were significantly higher than the grade for states in Q5 with a p-value less than 0.05.

Table 6 Percentiles of smoking rate (adjusted for sex, age, and race) among adults with asthma and corresponding grades for tobacco control status

	Quintiles of smoking rate among adults with asthma (20% of states in each quintile based on smoker percentages)				
Smoking Rate	Q1 (lowest = 11.2%-16.7%)	Q2 (second = 17.0%-20.7%)	Q3 (middle = 20.8%-22.9%)	Q4 (fourth = 23.0%-25.7%)	Q5 (highest = 25.8%-34.0%)
Overall Policy Grade	72	50	47	35	22
Range (min-max grade)	5-11	2-10	1-9	0-8	0-6
Mean grade (std dev)	7.2 (1.99)	5.0 (2.62)	4.7 (3.20)	3.5 (2.92)	2.0 (2.00)
T-test	Q1 vs. Q5	Q2 vs. Q5	Q3 vs. Q5	Q4 vs. Q5	
P-value	0.0000*	0.0096*	0.0369*	0.1925	

*Mean grades for the two groups are significantly different (t-test: p-value <0.05)

In addition to measuring the effects of tobacco control measures nationwide, the states with the lowest and highest smoking rates among adults with asthma were identified and listed in Table 7. Table 7 shows that none of the states in the lowest quintile have an asterisk next to their name, indicating that the percentage of adult smokers with asthma is not greater than the percentage of adult smokers who do not have asthma. On the other hand, nine out of the eleven states listed in the highest quintile column have an asterisk next to their name. The states in the lowest quintile (5-11) had an overall policy grade range that was significantly higher than the states in the highest quintile (0-4) (t-test: p-value <0.00001). Also, the ranges of smoking rates

in the lowest (11.2-16.6) and highest quintiles (25.8-34.0) were found to be statistically significant.

Table 7 *Evaluating the tobacco control status of states with the lowest and highest smoking rates (adjusted for sex, age, and race) among adults with asthma*

Note: none of the lowest states have asterisk next to the name.

The states with the lowest smoking rates among adults with asthma (below 20 th percentile of 17.0%)			The states with the highest smoking rates among adults with asthma (80 th percentile of 25.8% and above)		
State	% Smokers	Overall Policy Grade	State	% Smokers	Overall Policy Grade
UT	11.2	5	LA	25.8	3
DC	15.2	8	AL	26.2	0
CO	15.4	5	OH*	26.5	6
NJ	16.0	8	IN*	26.9	3
RI	16.0	11	SC*	28.4	0
CT	16.1	5	TN*	29.5	3
MN	16.1	9	OK*	29.9	2
CA	16.2	6	MS*	30.3	1
AZ	16.3	8	AR*	30.6	4
HI	16.6	7	WV*	32.8	0
			KY*	34.0	0
Total grade		72			22

Mean (std dev)*		7.2 (1.99)			2.0 (2.0)
Range	(11.2-16.6)	(5-11)		(25.8-34.0)	(0-4)

*Mean grades for the two groups are significantly different (t-test: p-value <0.00001)

CHAPTER V

DISCUSSION AND CONCLUSION

5.1 Discussion

The first effort to educate the public on the harmful effects of second hand smoking was in 1971 when Surgeon General Jesse Steinfeld suggested that low-dose exposure to cigarette smoke may have a potential public health risk for nonsmokers (Institute, 2000). Fifteen years later, in 1986, Surgeon General Dr. Koop released a report *The Health Consequences of Involuntary Smoking*. The report included the following:

“Involuntary smoking is a cause of disease, including lung cancer, in health nonsmokers.

The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.

The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke.”(Institute, 2000)

After the publication of Dr. Koop’s report, federal agencies started taking steps to educate the public on the dangers of tobacco smoke to smokers as well as non-smokers. One of the most significant steps taken by a federal agency came in 1990 when the U.S. Environmental Protection Agency (EPA) released a publication *Assessment on Environmental Tobacco Smoke (ETS)* in which they classify tobacco smoke as a Group A carcinogen that can cause lung cancer

in nonsmokers (Institute, 2000). Since then, a multitude of reviews conducted by medical and governmental organizations leave no doubt that environmental tobacco smoke causes disease in smokers and non-smokers. The health effects associated with exposure to ETS include: developmental (fetal growth and sudden infant death syndrome), respiratory (acute lower respiratory tract infections in children, asthma induction and exacerbation in children, chronic respiratory symptoms in children, eye and nasal irritation in adults, and middle ear infections in children), carcinogenic effects (lung cancer and nasal sinus cancer), and cardiovascular effects (heart disease mortality and acute and chronic coronary heart disease morbidity) (Institute, 2000).

Multiple factors play a role in the development of asthma and the exacerbation of asthma symptoms. We examined the predictive ability of selected factors on asthma prevalence in the 2009 and 2010 BRFSS data. We found that all selected potential predictive factors were significant predictors of current asthma for the overall study population. These findings are consistent with those of previous research (Zahran, 2012; Strine, 2004; Gwynn, 2004). Examining data from the ALA Tobacco Control Report of 2008 along with smoking rate in states, our findings indicate that states with better tobacco control policies/laws had lower smoking rate among adults with asthma than states that do not have.

In order to combat this public health concern, the public health community has been involved in policies that have a more comprehensive approach designed to change environmental and social norms. The findings of this paper show evidence of significant association between smoking and asthma status and lower smoking prevalence rate among adults with asthma in

states with three tobacco control interventions. This study was unusual in specifically linking national reduced smoking prevalence among adults with asthma with an increasing tobacco control measures (smoke-free environment, cigarette excise tax and cessation coverage) as opposed to specific community based efforts (Dockery, 1993;Juster, 2007;Head, 2012;Roberts, 2012;Huang, 2012) and specific tobacco control policies (Juster, 2007;Roberts, 2012;Huang, 2012;Wakefield, 2008;Kabir, 2009;Jill S. Rumberger, 2010;Dilley, 2012). The study also furnished great detail on the link between smoking prevalence and specifically asthma rates as it relates to policy which is lacking in research, most literature reviewed discuss the link between smoking prevalence and policy discounting the effect smoking has on asthma rates (Juster, 2007;Roberts, 2012;Head, 2012;Huang, 2012;Wakefield, 2008;Kabir, 2009;Jill S. Rumberger, 2010;Dilley, 2012). While there is a wealth of research on cessation outcomes related to specific legislation measure, there is less research on population-level smoking prevalence.

Although, the association between lower smoking rate and the implementation of the tobacco control policies and measures is based on state level evaluation (not based on individual data), nonetheless the findings clearly indicate a strong association, which brings the importance of government role in reducing smoking rate and related harmful health effects.

Policy measures

Among the three policy measures discussed in this study, Cigarette excise tax is the most controversial. President Obama states that his proposal for a 94-cent federal tax increase ‘would have substantial public health benefits, particularly for young America’ (Budget, 2013). This is

following the 62 cent tax increase in 2009 which effectively dropped cigarette sales by an estimated 10% (Huang, 2012). This topic is controversial for two reasons. First, sales taxes are viewed as regressive because the higher income groups invest a smaller share of their income than the poor. Second, since the prevalence of smoking is higher among the poor, cigarettes are disproportionately consumed by the poor. While excise tax could have some sort of discriminatory selection because it is targeting the people who cannot afford to buy cigarettes. However, it is this sector of the community that is most affected by smoking rate and asthma based on statistics (Table 2 and 3). Public health policy aims to treat behavioral change, the extent to which higher taxes cause smokers to quit or cut back on smoking.

If the poor cut back enough relative to the rich, the smoking and asthma rates should be balanced in a way that will promote the policy's impact as possible effective preventive measure. Some may argue that putting this additional burden on low income populations could also lead to some other unfavorable results like cutting back from their expense on food and other essential necessities and using the money to buy cigarettes which will create financial stress (Martire, 2011). However, if higher cigarette tax forces the poor in society to cut back on their consumption of cigarettes that will meet the goal of reducing overall smoking rate, exposure to second hand smoke as well as lessen the economic burden on the health care system brought on by smoking related diseases.

It is important to note that, as demonstrated by Martire et al., the financial stress on responses to price increases may lower the quit rates among certain smokers. Not all smokers among the poor population will respond the same to the increase in cigarette tax, some may

choose to quite in so doing they eliminate the financial burden of cigarette taxes. Others may lessen their daily consumption and be left where they started spending the same amount yet consuming less. The final group is that which will continue to smoke the same amount, spending more than they did previously and experiencing additional financial stress. An approach that may prove beneficial is to create policy that will support smokers that are struggling to quit by providing them with free or subsidized smoking cessation resources that will reduce their economic burdens. This approach has been implemented and appropriately funded in eight states as of 2011; according to the CDC's *Best Practices for Comprehensive Tobacco Control Programs* the per capita recommended level of investment ranges from \$9.23 to \$18.02 based on state's specific characteristics. The report confirms that if states were to continue the level of investment for a 5 year time span, there would see the number of smokers decline by 5 million.

An additional advantage to implementing and funding tobacco cessation programs is that fewer young adults (particularly in the 8th through 12th grade range) would smoke. Tauras et al., published a study in the American Journal of Public Health in which they conclude that if states invested the CDC-recommended minimum level for tobacco control, the estimates for youth smoking prevalence would have been lowered by 3.5- 13.5% nationwide (Tauras, 2005).

5.2 Study Limitations

The use of BRFSS dataset is a great strength as it has been applied to research and evaluated by the CDC and participating states since 1984 (Prevention, 2011). The content of the surveillance system which includes: designs of the questionnaire, survey questions, interviewing

methods, procedures for data collection and processing has been progressively developed over the past 29 years to improve data quality. At large, statistics from the BRFSS is relied on and accurate. However, there were several limitations in this study.

First, the BRFSS data is collected by telephone interviews. Individuals who live in households without a residential telephone (1.7%) are not included (Prevention, 2011). Therefore, the BRFSS might exclude persons of lower socioeconomic status or households with cellular phones only (24.9%) (CDC, 2011). Second, the survey is based on populations that are not institutionalized; thereby excluding persons residing elsewhere, such as nursing homes or long-term care facilities, this may affect the findings for older adults. Third, the BRFSS data are self-reported by respondents and smoking status was not validated by biochemical tests. As a result, smoking maybe underreported because of social desirability bias. Fourth, the sampling frame of the BRFSS is the entire state; therefore, some rural areas might be represented by relatively few interviews. Finally, the study assumes that the full benefits of the tobacco control measures will be accrued within a year of implementation. However, in reality, it may take years before individuals decide to change their smoking habits and for society to start reaping the economic and health benefits of lower smoking rates.

5.3 Conclusion

Our findings show that predictors of asthma differed by demographic characteristics. As in the results for whites and blacks, all selected potential factors (sex, age, education, income, and smoking status) were significant predictors of asthma prevalence in the overall adult population.

Also, shown in this study is the link between smoking status and asthma status nationwide as it affects different demographic groups in the population. Finally, we were able to show the link between smoking rate and state of tobacco control efforts. The study findings indicate that comprehensive policy measures have a significant impact on the smoking rate nationwide. Although most U.S. state smoke-free policies and regulations are relatively new, it is evident that that these laws are effective in promoting cessation among adults and reducing nonsmokers' exposure to secondhand smoke. The study found that smoke-free laws will improve health by lowering smoking rates among adult smoker that will in turn reduce harmful health effects due to smoking and exposure to secondhand smoke, including exacerbation of asthma symptoms.

Further studies are warranted to track impact of interventions expected to affect smoking on the general population. Observational studies with longitudinal design are needed to employ clear definitions of policy components and careful control for confounding. These studies will be important in developing targeted interventions to reduce the health and economic impact of smoking among disproportionately affected segments of the United States population.

REFERENCES

- Agrawal, S. "Effect of Indoor Air Pollution from Biomass and Solid Fuel Combustion on Prevalence of Self-Reported Asthma among Adult Men and Women in India: Findings from a Nationwide Large-Scale Cross-Sectional Survey." *J Asthma* 49, no. 4 (2012): 355-65.
- Akinbami, L. J., J. E. Moorman, C. Bailey, H. S. Zahran, M. King, C. A. Johnson and X. Liu. "Trends in Asthma Prevalence, Health Care Use, and Mortality in the United States, 2001-2010." *NCHS Data Brief*, no. 94 (2012): 1-8.
- Asthma in Adults Fact Sheet. <http://www.lung.org/lung-disease/asthma/resources/facts-and-figures/asthma-in-adult.html>.
- Barnes, P. J. "Immunology of Asthma and Chronic Obstructive Pulmonary Disease." *Nat Rev Immunol* 8, no. 3 (2008): 183-92.
- Barnett, S. B. and T. A. Nurmagambetov. "Costs of Asthma in the United States: 2002-2007." *J Allergy Clin Immunol* 127, no. 1 (2011): 145-52.
- Beane, J., P. Sebastiani, G. Liu, J. S. Brody, M. E. Lenburg and A. Spira. "Reversible and Permanent Effects of Tobacco Smoke Exposure on Airway Epithelial Gene Expression." *Genome Biol* 8, no. 9 (2007): R201.
- Brook, R. D., S. Rajagopalan, C. A. Pope, 3rd, J. R. Brook, A. Bhatnagar, A. V. Diez-Roux, F. Holguin, Y. Hong, R. V. Luepker, M. A. Mittleman, A. Peters, D. Siscovick, S. C. Smith, Jr., L. Whitsel, J. D. Kaufman, Epidemiology American Heart Association Council on, Council on the Kidney in Cardiovascular Disease Prevention, Physical Activity Council on Nutrition and Metabolism. "Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement from the American Heart Association." *Circulation* 121, no. 21 (2010): 2331-78.
- Budget, Office of Management and. *Fiscal Year 2014 Budget of the U.S. Government*. Washington, DC, 2013.
- CDC 2011. Centers for Disease Control and Prevention. "Vital Signs: Asthma Prevalence, Disease Characteristics, and Self-Management Education: United States, 2001--2009." *MMWR Morb Mortal Wkly Rep* 60, no. 17 (2011): 547-52.
-

- Cerveri, I., L. Cazzoletti, A. G. Corsico, A. Marcon, R. Niniano, A. Grosso, V. Ronzoni, S. Accordini, C. Janson, I. Pin, V. Siroux and R. de Marco. "The Impact of Cigarette Smoking on Asthma: A Population-Based International Cohort Study." *Int Arch Allergy Immunol* 158, no. 2 (2012): 175-83.
- Institute of Medicine 2000. Committee on the Assessment of Asthma and Indoor Air, Division of Health Promotion and Disease Prevention, Institute of Medicine. *Clearing the Air: Asthma and Indoor Air Exposures*: The National Academies Press, 2000.
- Dijk, F. N., J. C. de Jongste, D. S. Postma and G. H. Koppelman. "Genetics of Onset of Asthma." *Curr Opin Allergy Clin Immunol* 13, no. 2 (2013): 193-202.
- Dilley, J. A., J. R. Harris, M. J. Boysun and T. R. Reid. "Program, Policy, and Price Interventions for Tobacco Control: Quantifying the Return on Investment of a State Tobacco Control Program." *Am J Public Health* 102, no. 2 (2012): e22-8.
- Dockery, D. W. "Health Effects of Particulate Air Pollution." *Ann Epidemiol* 19, no. 4 (2009): 257-63.
- Dockery, D. W., C. A. Pope, 3rd, X. Xu, J. D. Spengler, J. H. Ware, M. E. Fay, B. G. Ferris, Jr. and F. E. Speizer. "An Association between Air Pollution and Mortality in Six U.S. Cities." *N Engl J Med* 329, no. 24 (1993): 1753-9.
- Forno, E., J. Lasky-Su, B. Himes, J. Howrylak, C. Ramsey, J. Brehm, B. Klanderman, J. Ziniti, E. Melen, G. Pershagen, M. Wickman, F. Martinez, D. Mauger, C. Sorkness, K. Tantisira, B. A. Raby, S. T. Weiss and J. C. Celedon. "Genome-Wide Association Study of the Age of Onset of Childhood Asthma." *J Allergy Clin Immunol* 130, no. 1 (2012): 83-90 e4.
- Frieden, T. R., Control Centers for Disease and Prevention. "Forward: Cdc Health Disparities and Inequalities Report - United States, 2011." *MMWR Surveill Summ* 60 Suppl, (2011): 1-2.
- Fuhlbrigge, A. L., B. T. Kitch, A. D. Paltiel, K. M. Kuntz, P. J. Neumann, D. W. Dockery and S. T. Weiss. "Fev(1) Is Associated with Risk of Asthma Attacks in a Pediatric Population." *J Allergy Clin Immunol* 107, no. 1 (2001): 61-7.
- Gelman, Andrew and John B. Carlin. *Poststratification and Weighting Adjustments*: Wiley, 2000.
-

- Girardot, S. P., P. B. Ryan, S. M. Smith, W. T. Davis, C. B. Hamilton, R. A. Obenour, J. R. Renfro, K. A. Tromatore and G. D. Reed. "Ozone and Pm2.5 Exposure and Acute Pulmonary Health Effects: A Study of Hikers in the Great Smoky Mountains National Park." *Environ Health Perspect* 114, no. 7 (2006): 1044-52.
- Grassi, M., M. Bugiani and R. de Marco. "Investigating Indicators and Determinants of Asthma in Young Adults." *Eur J Epidemiol* 21, no. 11 (2006): 831-42.
- Gwynn, R. C. "Risk Factors for Asthma in Us Adults: Results from the 2000 Behavioral Risk Factor Surveillance System." *J Asthma* 41, no. 1 (2004): 91-8.
- Hahn, E. J. "Smokefree Legislation: A Review of Health and Economic Outcomes Research." *Am J Prev Med* 39, no. 6 Suppl 1 (2010): S66-76.
- Haw, S. J. and L. Gruer. "Changes in Exposure of Adult Non-Smokers to Secondhand Smoke after Implementation of Smoke-Free Legislation in Scotland: National Cross Sectional Survey." *BMJ* 335, no. 7619 (2007): 549.
- Head, P., B. E. Jackson, S. Bae and D. Cherry. "Hospital Discharge Rates before and after Implementation of a City-Wide Smoking Ban in a Texas City, 2004-2008." *Prev Chronic Dis* 9, (2012): E179.
- Henderson, J., R. Granell and J. Sterne. "The Search for New Asthma Phenotypes." *Arch Dis Child* 94, no. 5 (2009): 333-6.
- Himes, B. E., G. M. Hunninghake, J. W. Baurley, N. M. Rafaels, P. Sleiman, D. P. Strachan, J. B. Wilk, S. A. Willis-Owen, B. Klanderman, J. Lasky-Su, R. Lazarus, A. J. Murphy, M. E. Soto-Quiros, L. Avila, T. Beaty, R. A. Mathias, I. Ruczinski, K. C. Barnes, J. C. Celedon, W. O. Cookson, W. J. Gauderman, F. D. Gilliland, H. Hakonarson, C. Lange, M. F. Moffatt, G. T. O'Connor, B. A. Raby, E. K. Silverman and S. T. Weiss. "Genome-Wide Association Analysis Identifies Pde4d as an Asthma-Susceptibility Gene." *Am J Hum Genet* 84, no. 5 (2009): 581-93.
- Hoppe, P., A. Peters, G. Rabe, G. Praml, J. Lindner, G. Jakobi, G. Fruhmann and D. Nowak. "Environmental Ozone Effects in Different Population Subgroups." *Int J Hyg Environ Health* 206, no. 6 (2003): 505-16.
-

- Huang, Jidong and Frank J. IV Chaloupka. "The Impact of the 2009 Federal Tobacco Excise Tax Increase on Youth Tobacco Use." no. April (2012).
- Huss, K., N. F. Adkinson, Jr., P. A. Eggleston, C. Dawson, M. L. Van Natta and R. G. Hamilton. "House Dust Mite and Cockroach Exposure Are Strong Risk Factors for Positive Allergy Skin Test Responses in the Childhood Asthma Management Program." *J Allergy Clin Immunol* 107, no. 1 (2001): 48-54.
- Institute, National Cancer. "State and Local Legislative Action to Reduce Tobacco Use: Smoking and Tobacco Control." monograph. Bethesda, Maryland: National Institutes of Health, 2000.
- Jackson, T., C. Roberts and D. N. Pearlman. "Adults with Asthma Who Smoke--a Neglected Population?" *Med Health R I* 94, no. 10 (2011): 306-8.
- Jill S. Rumberger, Christopher S. Hollenbeak, David Kline. *Potential Costs and Benefits of Smoking Cessation: An Overview of the Approach to State Specific Analysis*. Pennsylvania: Pennsylvania State University, 2010.
- Juster, H. R., B. R. Loomis, T. M. Hinman, M. C. Farrelly, A. Hyland, U. E. Bauer and G. S. Birkhead. "Declines in Hospital Admissions for Acute Myocardial Infarction in New York State after Implementation of a Comprehensive Smoking Ban." *Am J Public Health* 97, no. 11 (2007): 2035-9.
- Kabir, Z., V. Clarke, R. Conroy, E. McNamee, S. Daly and L. Clancy. "Low Birthweight and Preterm Birth Rates 1 Year before and after the Irish Workplace Smoking Ban." *BJOG* 116, no. 13 (2009): 1782-7.
- Korrick, S. A., L. M. Neas, D. W. Dockery, D. R. Gold, G. A. Allen, L. B. Hill, K. D. Kimball, B. A. Rosner and F. E. Speizer. "Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers." *Environ Health Perspect* 106, no. 2 (1998): 93-9.
- Krieger, Gary R; Sullivan, John B. *Clinical Environmental Health and Toxic Exposures*. Philadelphia, Pa: London : Lippincott Williams & Wilkins 2001 2001.
- Leopold, P. L., M. J. O'Mahony, X. J. Lian, A. E. Tilley, B. G. Harvey and R. G. Crystal. "Smoking Is Associated with Shortened Airway Cilia." *PLoS One* 4, no. 12 (2009): e8157.
-

- Levy, D. T. and K. Friend. "Examining the Effects of Tobacco Treatment Policies on Smoking Rates and Smoking Related Deaths Using the Simsmoke Computer Simulation Model." *Tob Control* 11, no. 1 (2002): 47-54.
- Martire, K. A., R. P. Mattick, C. M. Doran and W. D. Hall. "Cigarette Tax and Public Health: What Are the Implications of Financially Stressed Smokers for the Effects of Price Increases on Smoking Prevalence?" *Addiction* 106, no. 3 (2011): 622-30.
- McConnell, R., K. Berhane, F. Gilliland, S. J. London, T. Islam, W. J. Gauderman, E. Avol, H. G. Margolis and J. M. Peters. "Asthma in Exercising Children Exposed to Ozone: A Cohort Study." *Lancet* 359, no. 9304 (2002): 386-91.
- Medicine, Institute of. *Ending the Tobacco Problem: A Blueprint for the Nation*, Edited by Richard J. Bonnie. Washington, DC: The National Academies Press, 2007.
- Mishra, Vinod. "Effect of Indoor Air Pollution from Biomass Combustion on Prevalence of Asthma in the Elderly." *Environmental Health Perspectives* 111, no. 1 (2002): 71-77.
- Moorman, J. E., R. A. Rudd, C. A. Johnson, M. King, P. Minor, C. Bailey, M. R. Scalia, L. J. Akinbami, Control Centers for Disease and Prevention. "National Surveillance for Asthma--United States, 1980-2004." *MMWR Surveill Summ* 56, no. 8 (2007): 1-54.
- National Asthma, Education and Program Prevention. "Expert Panel Report 3 (Epr-3): Guidelines for the Diagnosis and Management of Asthma-Summary Report 2007." *J Allergy Clin Immunol* 120, no. 5 Suppl (2007): S94-138.
- Ober, C. and E. E. Thompson. "Rethinking Genetic Models of Asthma: The Role of Environmental Modifiers." *Curr Opin Immunol* 17, no. 6 (2005): 670-8.
- Pearlman, A. N., R. K. Chandra, D. Chang, D. B. Conley, A. Tripathi-Peters, L. C. Grammer, R. T. Schleimer and R. C. Kern. "Relationships between Severity of Chronic Rhinosinusitis and Nasal Polyposis, Asthma, and Atopy." *Am J Rhinol Allergy* 23, no. 2 (2009): 145-8.
- Piipari, R. and J. J. Jaakkola. "Smoking and Asthma in Adults." *Eur Respir J* 24, no. 5 (2004): 734-9.
-

- Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston. "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." *JAMA* 287, no. 9 (2002): 1132-41.
- Pope, C. A., 3rd and D. W. Dockery. "Health Effects of Fine Particulate Air Pollution: Lines That Connect." *J Air Waste Manag Assoc* 56, no. 6 (2006): 709-42.
- Pope, C. A., 3rd, M. Ezzati and D. W. Dockery. "Fine-Particulate Air Pollution and Life Expectancy in the United States." *N Engl J Med* 360, no. 4 (2009): 376-86.
- CDC, 1997. Centers for Disease Control and Prevention. "From the Centers for Disease Control and Prevention. State-Specific Prevalence among Adults of Current Cigarette Smoking and Smokeless Tobacco Use and Per Capita Tax-Paid Sales of Cigarettes--United States, 1997." *JAMA* 281, no. 1 (1999): 29-30.
- CDC, 2000. Centers for Disease Control and Prevention. *Reducing Tobacco Use: A Report of the Surgeon General*. Atlanta, GA, 2000.
- CDC, 2014. Centers for Disease Control and Prevention. "Reduced Secondhand Smoke Exposure after Implementation of a Comprehensive Statewide Smoking Ban--New York, June 26, 2003-June 30, 2004." *MMWR Morb Mortal Wkly Rep* 56, no. 28 (2007): 705-8.
- CDC BRFSS. Centers for Disease Control and Prevention. *Behavioral Risk Factor Surveillance System 2010 Summary Data Quality Report*. 2011.
- Rank, M. A., J. T. Liesinger, J. Y. Ziegenfuss, M. E. Branda, K. G. Lim, B. P. Yawn, J. T. Li and N. D. Shah. "Asthma Expenditures in the United States Comparing 2004 to 2006 and 1996 to 1998." *Am J Manag Care* 18, no. 9 (2012): 499-504.
- Roberts, C., P. J. Davis, K. E. Taylor and D. N. Pearlman. "The Impact of Rhode Island's Statewide Smoke-Free Ordinance on Hospital Admissions and Costs for Acute Myocardial Infarction and Asthma." *Med Health R I* 95, no. 1 (2012): 23-5.
- Services, The Task Force on Community Preventive. *The Guide to Community Preventive Services: What Works to Promote Health?* New York, NY, 2005.
-

- Services, US Department of Health and Human. "The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General." Atlanta (GA), 2006.
- Smith, L. A., J. L. Hatcher-Ross, R. Wertheimer and R. S. Kahn. "Rethinking Race/Ethnicity, Income, and Childhood Asthma: Racial/Ethnic Disparities Concentrated among the Very Poor." *Public Health Rep* 120, no. 2 (2005): 109-16.
- Spektor, D. M., M. Lippmann, G. D. Thurston, P. J. Liou, J. Stecko, G. O'Connor, E. Garshick, F. E. Speizer and C. Hayes. "Effects of Ambient Ozone on Respiratory Function in Healthy Adults Exercising Outdoors." *Am Rev Respir Dis* 138, no. 4 (1988): 821-8.
- Spira, A., J. Beane, V. Shah, G. Liu, F. Schembri, X. Yang, J. Palma and J. S. Brody. "Effects of Cigarette Smoke on the Human Airway Epithelial Cell Transcriptome." *Proc Natl Acad Sci U S A* 101, no. 27 (2004): 10143-8.
- Stapleton, M., A. Howard-Thompson, C. George, R. M. Hoover and T. H. Self. "Smoking and Asthma." *J Am Board Fam Med* 24, no. 3 (2011): 313-22.
- Stephen J. Blumberg, Ph.D., and Julian V. Luke. *Wireless Substitution: Early Release of Estimates from the National Health Interview Survey, January - June 2010*. 2010.
- Strine, T. W., E. S. Ford, L. Balluz, D. P. Chapman and A. H. Mokdad. "Risk Behaviors and Health-Related Quality of Life among Adults with Asthma: The Role of Mental Health Status." *Chest* 126, no. 6 (2004): 1849-54.
- Subbarao, P., P. J. Mandhane and M. R. Sears. "Asthma: Epidemiology, Etiology and Risk Factors." *CMAJ* 181, no. 9 (2009): E181-90.
- Tauras, J. A., F. J. Chaloupka, M. C. Farrelly, G. A. Giovino, M. Wakefield, L. D. Johnston, M. O'Malley P, D. D. Kloska and T. F. Pechacek. "State Tobacco Control Spending and Youth Smoking." *Am J Public Health* 95, no. 2 (2005): 338-44.
- To, T., S. Stanojevic, G. Moores, A. S. Gershon, E. D. Bateman, A. A. Cruz and L. P. Boulet. "Global Asthma Prevalence in Adults: Findings from the Cross-Sectional World Health Survey." *BMC Public Health* 12, (2012): 204.
-

- Triche, E. W., J. F. Gent, T. R. Holford, K. Belanger, M. B. Bracken, W. S. Beckett, L. Naeher, J. E. McSharry and B. P. Leaderer. "Low-Level Ozone Exposure and Respiratory Symptoms in Infants." *Environ Health Perspect* 114, no. 6 (2006): 911-6.
- Wakefield, M. A., S. Durkin, M. J. Spittal, M. Siahpush, M. Scollo, J. A. Simpson, S. Chapman, V. White and D. Hill. "Impact of Tobacco Control Policies and Mass Media Campaigns on Monthly Adult Smoking Prevalence." *Am J Public Health* 98, no. 8 (2008): 1443-50.
- Willemsen, G., T. C. van Beijsterveldt, C. G. van Baal, D. Postma and D. I. Boomsma. "Heritability of Self-Reported Asthma and Allergy: A Study in Adult Dutch Twins, Siblings and Parents." *Twin Res Hum Genet* 11, no. 2 (2008): 132-42.
- William J. Curry, M.D. and Myron R. Schwartz, M.A.,. *Analyses of Behavioral Risk Factor Surveillance System Data for Rural Health Outcomes*. Pennsylvania State University, 2010.
- Yang, I. A., S. Savarimuthu, S. T. Kim, J. W. Holloway, S. C. Bell and K. M. Fong. "Gene-Environmental Interaction in Asthma." *Curr Opin Allergy Clin Immunol* 7, no. 1 (2007): 75-82.
- Zahran, H. S., C. J. Person, C. Bailey and J. E. Moorman. "Predictors of Asthma Self-Management Education among Children and Adults--2006-2007 Behavioral Risk Factor Surveillance System Asthma Call-Back Survey." *J Asthma* 49, no. 1 (2012): 98-106.
-