Prevalence of Asthma among Minority Population Children

Aged 0-17 Years in the United States

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ABSTRACT

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BACKGROUND: *"Asthma is a chronic inflammatory disorder of the airways....In susceptible individuals; this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyper responsiveness to a variety of stimuli"*

PURPOSE OF THE STUDY: This study will evaluate the impact of demographic and clinical predictors on the prevalence of childhood asthma among minority children aged 0-17 in United States.

METHODS: For this research, data from the 2011-2012 survey will be employed to conduct the required statistical tests using SAS SOFTWARE to answer the addressed research questions. A total of 95,677 NSCH interviews were completed nationally by parents/caregivers of children and youth approximately 1,876 in each state and the District of Columbia, ranging from 1,811 in South Dakota to 2,200 in Texas. The

Statistical analysis will begin with an estimation of the national prevalence of asthma among children aged 0-17 years and living in the United States. Characteristics of the sample population will then be compared with the general population to assess if there are differences or validate how the sample population represents the general population. The characteristics in Table 41 will show how levels asthma and severe asthma are distributed among children by their age, gender, race/ethnicity, SES, clinical predictors and insurance status.

RESULTS: Results have shown that being from low SES and minority race/ethnicity group (Hispanic, Non-Hispanic Black, and multi R racial/other Non-Hispanic) will increase the asthma and severe asthma rates among children living in the US.

DISCUSSION: Conducting a new study on a national scale to display the differences in childhood asthma prevalence among minority population living in United States is valuable for measuring the prevalence of this chronic disease and the discrepancy burden among US children; and for developing intervention programs for minority population living in US. This study will also find what is still confounding and unknown about childhood asthma and recommend areas for further research.

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This work is dedicated to my parents Abdulrazaq and Lameeah, my wife Hawraa and my two children Rayan and Farah whose love and support strengthened me to go through tedious days and nights of extensive reading in conducting this research and preparing this report.

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1.0 CHAPTER I INTRODUCTION

1.1 BACKGROUND

Based on the Asthma Diagnosis and Treatment Guidelines, the National Heart, Lung, and Blood Institute (NHLBI) in the National Institutes of Health (NIH) proposed the following definition of Asthma:

"Asthma is a chronic inflammatory disorder of the airways....In susceptible individuals; this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyper responsiveness to a variety of stimuli". [1]

Moreover, according to the "Family Guide to Asthma and Allergies" by the American Lung Association (ALA) in their definition of the significant characteristic of asthma, they states:

"Much has been learned about asthma in recent years, but nothing is more important than the observation that asthma is a disease of airway inflammation. By this, physicians mean that people with asthma have chronically inflamed airways that are ever prone to become twitchy and constricted after exposure to an asthma trigger. It is as if the airways of people with asthma are lying in wait for trouble. They stay poised at the edge of a cliff . . . ". And this implies that asthma is both a chronic and an occasional illness.[2]

There are two different forms of asthma. The first, recognized as allergic or extrinsic asthma, and is classified by attacks triggered by exposure to what is so-called "asthma

triggers," such as pet dander, negative tobacco smoke, dust mites, and mold spores. Usually, the onset of this form of asthma appears before the age of 30; actually, the majority of childhood asthma is allergic.

The second form is the non-allergic or intrinsic asthma, which demonstrates itself with the same symptoms as allergic asthma; but attacks of this form of asthma are not activated by recognizable allergens. While intrinsic asthma can begin at any age, the onset usually occurs in adulthood.[3]

Asthma is a chronic inflammatory illness of the airways.[4, 5] In 2011 Report "Global Strategy for Asthma Management and Prevention ", the Global Initiative for Asthma states that asthma is a chronic disease of both children and adults, resulting in 300 million individuals suffering worldwide.[6, 7] In addition, Asthma disease involving a number of factors or causes that has been associated with family, infective, allergens, socioeconomic, psychosocial, and environmental factors.[8, 9]

In the United States, asthma affects more than 22 million persons.[10, 11] According to the current asthma prevalence by the National Center for Health Statistics and the Centers for Disease Control and Prevention; it is considered one of the most common chronic illnesses of childhood, affecting more than 6 million children. However, there have been significant advances since the release of the first clinical practice guidelines by the National Asthma Education and Prevention Program (NAEPP) in 1991. For instance, the total of deaths due to asthma has dropped, even in the appearance of a rising prevalence of the illness; less number of asthmatic patients report limitations to activities; and an increasing percentage of asthmatic patients receive proper patient education.[10]

A part from this, Hospitalization rates have continued relatively constant over the past decade, with lesser percentages in some age groups but higher percentages among young children, ages 0–4 years. According to the literature, there is some suggestion that better perception of childhood asthma supplies these percentages. Nonetheless, the burden of preventable hospitalizations remains. Jointly, asthmatic persons have more than 497,000 hospitalizations yearly.[28] Likewise, racial/ethnic differences in asthma burden continue, with substantial impact on African American and Puerto Rican residents. The confront remains to support all asthmatic individuals, especially those at high risk, should obtain quality asthma care.

Health differences are the differences that arise by gender, race/ethnicity, education level, income level, disability, or geographic location. These differences exist amongst all age groups, involving children and adolescents. For instance, low-income families and children of color holdup behind their more comfortable White peer in terms of health status. Similarly, children lower in the socioeconomic hierarchy suffer extremely from nearly every disease and illustrate higher percentages of mortality than those above them.[4] Low-income children have higher percentages of mortality and are more liable to have greater severity of disability even with the same type of disability and to have multiple conditions.[8,9] The relationship between health status and socioeconomic status has been also seen when the education level and occupation of children's parents are considered.[6]

However, some health differences are unpreventable, such as health complications that are associated to a person's genetic structure. Though, most health differences are possibly preventable, particularly when they are associated to factors such as living in

low-income neighborhoods or having unsatisfactory access to health care. Reducing, if not eliminating, health differences is a vital goal for a sum of reasons. Childhood is a time of enormous physical, social and emotional growth. Children who experience health problems are more likely to miss school, to have lifetime health problems and to acquire high costs for health care. In addition to the effects for children and their families, health differences have social effects in terms of productivity in adulthood as well as costs associated with health care. Health differences are also an issue of fairness; all children deserve the chance to be healthy and successful.

1.2 GOALS AND OBJECTIVES

This study in the next chapters, is aiming to examine whether racial/ethnic differences in childhood asthma have been increasing or decreasing over a period of time. We focus on the recent 2011-2012 period and across several indicators of childhood asthma. The main goals of this study were to examine the association of race/ethnicity and family income with the prevalence of childhood asthma. Specifically, we assessed whether racial/ ethnic disparities in asthma prevalence vary by depth of poverty in order to explore how race/ethnicity and income may interact to result in childhood asthma disparities. Where we found a racial/ethnic difference in asthma prevalence, we examined whether it could be partly explained by clinically relevant variables. We hope that a refined understanding of racial/ethnic differences in childhood asthma will sharpen research, clinical, and advocacy efforts to reduce disparities.

This secondary perspective data analysis to the National Survey of Children's Health (NSCH) allows researchers to recognize how the overall childhood asthma among

minority US population is changing over time and whether the significant gap in race/ethnic differences across diverse demographic and SES characteristics of asthmatic children who live in poverty can be narrowed by adding new findings and future recommendations to the current literature.

Finally, this study is aiming to outline the following objectives:

1- To examine the impact of clinically relevant variables on the prevalence of asthma for minority race/ethnic children with asthma aged 0-17 and living in US.
2- To examine the differences in health insurance status among minority racial/ethnic children aged 0-17, who live in low-income neighborhoods in US.
3- To examine the impact of SES (parental level of education and family income) and Demographic status (gender, age, race/ethnicity) on the prevalence of childhood asthma.

1.3 SIGNIFICANCE OF THE PROBLEM

Due to the increasing rates of prevalence of chronic illnesses among minority children in the United States,[12] childhood asthma among this population has been considered as an area of rising concern for US healthcare agencies, care providers as well as policy and decision makers. Presently, between 15% to 18% of U.S. population are children who live with chronic illness.[12,13] Within this mounting experience of chronic illness among children, asthma remains the most common chronic childhood illness, affecting more than 6.8 million U.S. children.[12,14] According to the American Lung Association, there is an increasing trend in the prevalence rate of childhood asthma from 8.9% in 2006 to 9.3% in 2007.[14] Further to this, the ALA states that Within the children population, the

highest prevalence rate is among the age groups from 5-17-year-olds, at 106.3/ 100,000 children.[14] The increase in the incidence of childhood asthma and prevalence has occurred regardless the numerous national, state, and local initiatives intended to reduce the asthma among US children.[14]

It is of great importance to understand the differences between gender and race within the prevalence of childhood asthma. Interestingly, when examining asthma through the lens of health differences and equality, there are unequal numbers of minority children with asthma when compared to the US majority population. About an overall asthma rate of 8% among Non-Hispanic Black children, compared to a 5% rate among Non-Hispanic White children.[14] Specifically, the prevalence of childhood asthma rate for Non-Hispanic Black boys is frightening 16% and is considerably higher than the 8% rate for girls.[12] In addition to this, the prevalence of childhood asthma rate for Non-Hispanic Black boys increased from 12% in 2001 to 16% in 2004.[12] Similarly, Non-Hispanic Black girls have the next highest childhood asthma rate, but the rates for girls are decreasing from a peak rate of 12% in 2003 to 9% in 2004.[12] Large body of literature demonstrates how childhood asthma is unequally distributed among US population.

According to the Centers for Disease Control and Prevention, minority children living in urban zones have higher morbidity and mortality rates due to asthma compared to their Non-Hispanic White peers.[12] Although the overall rates of asthma mortality are declining, asthma was responsible for 3,816 deaths in 2004.[12] Luckily asthma infrequently leads to death in children, and only 141 (<5%) of the asthma deaths were young children of age less than 15 years.[12] According to the Third National Health and

Nutrition Examination Survey (NHANES), Non-Hispanic Black children of ages 10 years and younger had the highest risk for increased asthma prevalence, morbidity and mortality,[12] which is captivating indication that this is a serious health discrepancy in the United States. Policy and decision makers' needs to understand the significant costs related to asthma care. The ALA states that the annual health care cost of asthma in the United States is more than $19.7 billion, with $14.7 billion direct and another $5 billion indirect costs.[14] On a singular level, the average cost for a patient with asthma is $4,912 per year, and in case of a more severe illness the associated cost with asthma management will be significantly higher.[14] Families from economically disadvantaged minority groups are often suffering from a huge financial burden caring for a child with asthma. According to the US Census Bureau report in 2008, an average income of African American family is $30,858, as compared to the average income of $46, 326 for Non-Hispanic White family (The United States Census Bureau, 2008). Nearly 24.5% of African American families lived below the FPL, as compared to 10.5% of White families in 2007 (The United States Census Bureau, 2008). The cost of Hospital care for asthma is more expensive than outpatient care, and the hospitalization rate for African American children is 3 times higher than the rate for Whites.[15]

1.4 SIGNIFICANCE OF THE STUDY:

Despite the abundance of research relating children and asthma, the racial/ethnic differences in asthma threat have not been fully explained. In prior studies where black children still had a higher prevalence of asthma compared with white children. Income, education, and housing variables were studied in the previous literature; however these factors did not fully clarify the difference. More significantly, previous research has not

explained an approach to the wide difference in asthma and related problems. Therefore, conducting a consistent and new study on a large-scale nationally representative data and including Hispanic and Multi-racial/other Non-Hispanic children, a minority group that has been usually eliminated form racial/ethnic literature, and refined understanding of racial/ethnic differences in childhood asthma will sharpen research, clinical, and advocacy efforts to reduce disparities.

1.5 HYPOTHESIS

Following from the purpose and goals of the study, the research study seeks through a secondary data analysis to evaluate the underlying assumption that:

"Being from low SES and race/ethnicity group (Hispanic, Non-Hispanic Black, multi-racial/other Non-Hispanic) will increase the asthma and severe asthma rates among minority children living in the US."

To test the above presumptive statement, an analytical model will be used to determine whether acceptance or rejection of the assumption is also influenced by other predictors such as age, gender, race/ethnicity (Non-Hispanic Black, Non-Hispanic Whites, Hispanics and Others), SES (Parental Educational level and income level. If that is the case, what will be the degree of the associations between childhood asthma and its covariates among children aged 0-17 years old in the US? The proposed study will answer the following research questions:

1- What are the different major demographic factors associated with the prevalence and persistent racial/ethnic inequalities among all US children with asthma?

2- How is SES (parents' income and education) associated with the prevalence of asthma among all children living in US?

3- What is the impact of clinically relevant factors on the prevalence of asthma among all children living in US?

4- Among children living in US, what is the impact of adequate insurance status on the prevalence of asthma among all children living in US?

1.6 THEORITICAL FRAMEWORK

One of the challenges faced by asthmatic populations, especially minorities with low-income level, is to have proper access to healthcare services. Notwithstanding the wide range of children health insurance programs funded federally, proper health care services are usually not accomplished. Children who do not receive sufficient health care services usually use untimely services and suffer from extra severe medical complications.[16] This is even more significant because of its great impact on the future health status among children with unmet special healthcare needs (SHCN).[17] There are numerous different factors that impact any asthmatic child's access to appropriate health care services.

For better understanding to the impact of proper access to the needed healthcare services among the different racial\ethnic minority children groups with different SES and health insurance on the severity of asthma in US, Aday and Andersen framework Model have been chosen as a conceptual framework in this research (Appendixes Figure 1). This model was established to assist in better understanding why families use health services; to explain and assess equitable access to health care; and to help in developing policies to encourage equal access to care services.[18] Another measure of equal and

proper access to care is the utilization of special healthcare services (SHCS) by asthmatic children from all different race/ethnicities in US. Therefore, this framework was employed in this research to recognize predictors and determinants of the proper access to health care services, more specifically, the association between SES and insurance status and the current status and severity of asthma among the different racial/ethnic minority children in a US population. Based on Aday's and Andersen model, proper access to healthcare services is not only the utilization of the main healthcare services, but instead based on receiving the needed SHCS by a patient.

In the same manner, Aday & Anderson explained in their framework model that the use of healthcare services is a means of predisposing factors such as race/ethnicity, age, gender; enabling factors such as parental education, income level, insurance coverage); and need factors (asthma diagnosis or report by physician or parents/guardian) to examine the impact of access and utilization to healthcare services on outcome asthma and severe asthma. In this study, the predisposing factors include: age (0-5, 6-11 and 12-17), race/ethnicity (Hispanic, White Non-Hispanic, Black Non- Hispanic and Multi-racial/other Non-Hispanic) and gender (male & female). The enabling factors include: parental education, income level, insurance coverage, and region. The need factors include those factors related to the condition for which health care services is needed, which include: asthma diagnosis or report by physician and parents/guardian (Appendixes Table 43).

2.0 CHAPTER II LITERATURE REVIEW

2.1 LITERATURE REVIEW SOURCES AND SEARCH STRINGS

In order to build scientific and historical evaluation along with understanding to the current status of asthma prevalence among minority children aged 0-17 years in US, the researcher targeted publications that portrayed or assessed childhood asthma, socioeconomic impacts on childhood asthma in US, racial/ethnic differences among asthmatic children, the impact of proper access and adequate insurance coverage to the needed healthcare services on childhood asthma severity in US. The researcher also considered articles published in peer-reviewed journals, counting review articles and surveys, and conference proceedings that defined or evaluated such applications. Only articles in English with available online abstracts at the time of searching were evaluated. Abstracts, poster presentations, and editorial publications were excluded, as were studies that did not involve childhood asthma.

The researcher reviewed the electronic publication databases such as PUBMED, Science Direct, ProQuest and Google Scholars to build the literature in this research.

2.2 PREVALENCE OF CHILDHOOD ASTHMA IN UNITED STATES

Asthma is a lifetime illness that causes wheezing, breathlessness, chest tightness, and coughing. It can limit a person's quality of life. While we don't know why asthma rates are rising, we do know that most people with asthma can control their symptoms and prevent asthma attacks by avoiding asthma triggers and correctly using prescribed medicines, such as inhaled corticosteroids.

The number of people diagnosed with asthma grew by 4.3 million from 2001 to 2009.[19] From 2001 through 2009 asthma rates raised the most among black children, almost a 50% increase. Asthma was linked to 3,447 deaths (about 9 per day) in 2007. Asthma costs in the US grew from about $53 billion in 2002 to about $56 billion in 2007, about a 6% increase. Greater access to medical care is needed for the growing number of people with asthma.[19]

More than half (53%) of people with asthma had an asthma attack in 2008. More children (57%) than adults (51%) had attack. 185 children and 3,262 adults died from asthma in 2007. About 1 in 10 children (10%) had asthma and 1 in 12 adults (8%) had asthma in 2009. Women were more likely than men and boys more likely than girls to have asthma. About 1 in 9 (11%) non-Hispanic blacks of all ages and about 1 in 6 (17%) of non-Hispanic black children had asthma in 2009, the highest rate among minority racial/ethnic groups. The greatest rise in asthma rates was among black children (almost a 50% increase) from 2001 through 2009.[19]

Among children, current asthma prevalence is lowest among persons aged 0-4 years and highest among males, black and multi-race persons. Healthcare utilization among children and adults differed by race/ethnicity. Routine office visits, emergency department visits, and urgent care visits for asthma were higher among children compared with adults. Overall, children were more likely than adults to receive asthma self-management education. Older adults, women and blacks were more likely to die due to asthma.

Children were more likely to have one or more routine office visits, emergency department visits, and urgent care visits for asthma (75.7% vs. 55.2%, 22.2% vs. 13.8%,

and 39.8% vs. 24.1%, respectively). Current asthma prevalence was lower among children aged 0–4 years (6.3%) compared with children aged 5-9 years (10.0%), 10–14 years (9.4%), and 15–17 years (9.0%). Current asthma prevalence was higher among males (10.0%) compared with females (7.1%). Current asthma prevalence was higher among black (14.0%) and multi-race (13.2%) children compared with white children (7.4%).

Overall, children were more likely to receive asthma self- management education compared with adults. Children (83.3%) were more likely to be taught to recognize signs and symptoms of asthma compared with adults (66.6%). Children (90.3%) were more likely to be taught how to respond during an asthma attack compared with adults (77.5%). Children (48.6%) were more likely to report having an asthma action plan compared with adults (27.4%). Children (14.3%) were more likely to report learning how to manage their asthma by taking a course compared with adults (10.1%). Children (96.1%) were more likely to be taught how to use an inhaler compared with adults (91.4%).

Suboptimal asthma control can lead to loss of productivity resulting in missed school days and missed workdays. In 2007, the estimated cost of asthma to society from loss of productivity was $3.8 billion. More children missed one or more days of school (48.6%) compared with adults who missed one or more days of work (32.9%) due to asthma. The proportion of children (39.4%) and adults (35.1%) reporting no activity limitation due to asthma were similar. Children were more likely to report a little activity limitation (46.5%) compared with adults (40.1%). More adults compared with children reported a

moderate amount (15.7% compared with 10.9%) or a lot of activity limitation (9.0% compared with 3.2%) due to asthma.

2.3 SOCIOECONOMIC STATUS (SES) AND SEVERITY OF CHILDHOOD ASTHMA

In a recent study by Schechter and colleagues, disease severity inversely associated with three markers of SES (state insurance coverage, maternal education, and median household income by zip code).[20] Low SES was associated with increased use of IV antibiotics to treat pulmonary exacerbations, and in the Medicaid population more outpatient prescriptions for antibiotics were given. These findings imply that in the US, low SES patients have greater therapeutic intervention but remain to have worse outcomes. The cause of these poor outcomes is still unknown and can be related to an inherently more severe disease, differential maintenance management, or prospective a combination of factors. Understanding how care gets translated from clinic to home is an important target for future studies.

African American women are nearly twice as likely to deliver preterm, regardless of their education levels and SES status. African American women are at higher risk of preterm delivery and, consequently, demeanor children with lung diseases associated with prematurity. Moreover, African American infants are less likely to receive surfactant medication at delivery and less likely to get RSV prophylaxis after NICU discharge than other racial groups [21,22] Similarly, Racial differences were also noticed in the prescription of respiratory medications upon discharge from the NICU in patients with similar respiratory status as well as differences in recommended antibiotic prescriptions after discharge.[23] Mortality rates differed among infants discharged from the NICU with 3.3%

of African American infants dying prior to the 18 to 22 month follow-up visit compared to 1.7% of White infants, and none in the Hispanic infants.[21]

However, Studies examining the link between socioeconomic status and asthma confirm that the impacts of asthma are greatest in low-income populations.[24-27] For example, in the U.S. in 1996, pediatric hospitalizations for asthma were estimated to be five times higher for children in lower income families.[28] The National Cooperative Inner-City Asthma study demonstrated that over 50% of study participants, whom were poor children living in inner cities, found it difficult to get follow-up asthma care. Among those with severe asthma, less than half were using anti-inflammatory medication.[29, 30]

Race is considered a main social factor of health in US because of its long-standing association with poverty, discrimination, residential separation, and unequal access to health care.[31, 32] Several studies suggest that race/ethnicity is also a significant social classification that has robust associations with numerous health outcomes, even after adjusting for traditional measures of (SES) such as education and income. These findings are believed to underline the differences in a complex set of social, economic, and biological properties accessible to different race/ethnic groups [31-33] that are frequently incapable to be totally accounted for or poorly measured in practical work.[34-36]

Finally, wide body of the literature recommending conducting new studies on a large-scale nationally representative data and include Hispanic and Multi-racial/other Non-Hispanic children, a minority group that has been usually eliminated from the race/ ethnic differences literature in order to achieve better understanding to the nature of association between childhood asthma severity and the quality of access to healthcare services as

well as the healthcare insurance status in relation to reducing the racial/ethnic differences among minority groups in US population.[31-39]

2.4 PERSISTENT RACIAL/ETHNIC DIFFERENCES AMONG ASTHMATIC U.S. CHILDREN

The U.S. Census Bureau indicated in their 2010 annual report that the variety of race\ethnic profile of the U.S. population is more apparent among children. Although nearly 80% of U.S. population is non-Hispanic white adults over age 65, only 55% of the same population under age 18 falls into this category. A significant body of the literature forecasts indicates that the U.S. will become a "majority minority" population by 2050 where non- Hispanic whites will encompass less than half of the U.S. population).[40] These demographic preferences have carried improved policy and research consideration to racial/ ethnic minorities and their social and economic welfare, including significant understanding of racial/ethnic differences in population health.[41] While exact health data on racial/ethnic groups have only been obtainable since the mid-20th century, racial/ ethnic differences in health have become one of the most extensively studied topics in health inequalities research especially on differences between non- Hispanic blacks and whites in US, but there has been growing recommendations on giving more consideration to Hispanics and Asian-origin populations, groups that have both grown rapidly as a result of modifications in U.S. immigration policy in the mid-1960s.[42,43] According to the Agency for Healthcare Research and Quality report released in 2011 and the report of the US Department of Health and Human Services released in 2010, the major policy concern is whether the U.S. is making improvement regarding decreasing racial/ethnic inequalities in health.[44,45]

However, with the exception of outcomes among infants, the major volume of research in the literature studying the U.S. racial/ethnic health discrepancies has been dedicated to adults and variations over time in race/ethnic changes among adults have been well described. A number of recent studies such as [43-50] have also studied trends in adult inequities with regard to disability, major adult chronic diseases, and their risk factors.

Correspondingly, variations over time in infant mortality in US have similarly been well described in the literature, at least between blacks and whites populations. Through most of the 20th century, the black- white ratio of the infant mortality rate (IMR) has amplified (although the total black- white difference has dropped over considerable period of time).[51] According to Singh & Van Dyck, the IMR among black infants in 2000 was more than 2.5 times that of white infants, a comparative inequality that declined only marginally between 2000- 2007.[52] On the other hand, based on evidence from a body of literature, our knowledge is relatively less about changes over time in race/ethnic differences in child and adolescent health. With regard to race/ethnic changes, maybe the best-examined child health indicator is asthma.[52, 53] In addition, based on a nationwide representative study conducted by McDaniel, Paxson, & Waldfogel in 2006, they demonstrated that black - white differences in asthma prevalence raised between 1997-2003 where blacks showed higher asthma rates compared to whites.[54] More recent variations in the black- white inequality among US children with asthma have not been evaluated to our knowledge. Based on a recent technical report by the American Academy of Pediatrics (AAP), which revised the current evidence on race/ethnic differences, determined that "racial/ethnic differences in child health and health care are

wide-ranging, persistent, and prevalent, and appear across the field of health and health care".[55, 56] The AAP report also suggested that insufficient studies have assessed the trends in child health differences.

2.5 PARENTAL EDUCATIONAL LEVEL AND SEVERITY OF CHILDHOOD ASTHMA

The proper care of chronic lung disease of infancy begins with the prevention of premature delivery. According to findings from a Dutch cohort it was discovered that pregnant women with a low education level (primary school, lower vocational training, intermediate general school and 3 years' general secondary school) have an approximately two-fold increase in preterm births.[57]

Low parental education of children with asthma is associated with extra severe asthma in the children and consequently increased used of rescue medication.[58] In addition to this, poor awareness of asthma symptoms results in higher morbidity and mortality rates in asthma among children. According to a study of the association between ethnicity and awareness in asthma was conducted employing home peak flow and spirometry procedures, Fritz and colleagues concluded that Hispanic children were less accurate than non-Hispanic children in assessing their pulmonary function. The authors also conclude that Hispanic children over- estimated respiratory agreement compared to unbiased spirometry findings. Finally, Fritz and colleagues hypothesize that the greater the increase of symptom among Hispanics the higher likely their visits to ED, unscheduled office visits, hospitalizations, and sickness conduct, which probably leading to asthma inequalities among these minority groups. The authors also conclude that additional

education about symptom recognition, illness strategies and unbiased quantification of lung health such as peak flows meters or home spirometry) could be a technique to help foster decrease disparities.[59]

However, the level of communication between the patient and health care provider directly affects the capability of proper understanding of information. Poor provider to patient communication has been published to lead to health care gaps among minority populations with asthma.[60] In a study of primary care physicians, it was stated that asthma plans were distributed among 70% of the patients; yet only 27% of those patients indicated that they received one. Increasing asthma awareness and focusing on educational gaps are significant elements in decreasing health discrepancies among children with asthma. In a study of pediatricians related to Adjusting for management and severity of illness, indicated that when physicians joined interactive seminars on asthma guidelines and patient communication, the patients of the participants had notably less hospitalization, child's family satisfaction enhanced and care providers' notices that education was more efficient after the intervention.[61] Regular asthma education, consistent asthma action plans and medication technique reviews are key factors of respiratory visits that can help address self-management and improve inequalities.

2.6 THE BUREDEN OF PARENTAL INCOME LEVEL ON CHILDHOOD ASTHMA

One of the major public health goals initiated by the US Department of Health and Human Services is to remove racial/ethnic differences in children's health.[62, 63] There are several pediatricians and researchers who focused their attention on childhood asthma as

one of the most common chronic conditions and one that causes significant morbidity among children. However, racial/ethnic differences in the severity of childhood asthma are still not fully understood.[17, 64] According to group of authors; race/ethnicity could be associated to childhood asthma in numerous potential ways. Weitzman & Gortmaker 1990; Miller 2000; Akinbami and colleagues 2002; states that racial/ethnic differences could seemingly result from confounding; other issues associated with both race/ethnicity and asthma, like SES or social class, may justify why black children seem to suffer from higher rates of asthma compared to white children.[65-67] On the other hand, [65,68-70] argue that the relationship between race/ethnicity and prevalence of childhood asthma could be described by racism, which is associated with restricted employment opportunities, consequently lead to lower income levels. Further, lower income among Black families' compared to the US population could result in an accumulation of black children in poor quality housing with higher chances of exposure to asthma-related triggers, such as cockroach antigen, therefore strengthen the pivotal pathway between race/ethnicity and higher asthma prevalence. A third potential way to explain the association between race/ethnicity and the high prevalence of childhood asthma could be due to distinction of genetic predisposition to asthma, possibly interacting with adverse environmental exposures.

Nevertheless, aside from the described potential factors mentioned above, income level has been examined very well in the literature as a confounder to the association between race/ethnicity and asthma, and yielding contradictory results. For instance, group of studies have indicated that adjusting for income and other socioeconomic elements, such as urban residence and single-parent status, decreases or removes the higher risk of

asthma associated with black "race" in unadjusted analyses.[65, 68-70] Other body of the literature, however, have discovered an increased prevalence of childhood asthma among black children compared to white children at all levels of income, even after controlling for such factors.[66, 67, 71-74]

Moreover, some studies in the literature have described children just as poor or non-poor but did not assess the complexity and background of poverty which could strongly miss significant differences in the poverty incidents among black and white children, and as a result dimming potential causal triggers for racial/ethnic differences in asthma. For instance, poor black children are more likely to live in deep poverty than poor white children. Similarly, poor black children are 40% more likely than poor white children to live in families with annual incomes less than half of the federal poverty level (FPL).[76] In addition, poor black children are much more likely than poor white children to live in neighborhoods of condensed poverty, where more than 40% of individuals in the neighborhood are poor.[77]

2.7 ACCESS TO CARE AND UTILIZATION OF HEALTH CARE SERVICIES

Stratified race representations have indicated that disparities in preterm births arise as a result of insufficient prenatal care, due to lack of access or utilization.[78] Differential use of health care means are not only limited the neonatal period but also prolongs beyond it. In regard to childhood asthma, a medical home provides support, treatment, and teaching critical for sufficient control. The use of an emergency care setting for asthma adversely affects outcomes. The lack of an asthma care specialist or an asthma action plan has been associated with a higher rate of acute asthma exacerbations.[79] In the US, African

American and Hispanic children with asthma are hospitalized more often compared to white children, and they more frequently utilize emergency departments.[80] These emergency facilities often become the primary resources of acquiring medical attention for symptomatic asthmatic children for lower-income families. An Italian study indicated that children from underprivileged backgrounds were hospitalized more often and did not make regular use of spirometry.[81] Race/ethnicity associates with the overuse of emergency care, even after adjusting for insurance, symptom severity, and socioeconomic factors. Providing a medical home for pediatric patients with asthma is an important step to improving asthma care.

However, despite the fact that there are differences in access to effective outpatient healthcare management of asthma among US population, This deficiency of coordinated quality care for asthma often lead to the need for inpatient care and hospitalization. According to the Asthma and Allergy Foundation, of all the hospitalization rates for asthma in 2006, 44% of these hospitalizations are rated for children (Asthma and Allergy Foundation of America, 2008). The hospitalization rate for asthmatic African Americans is 1.4-4.0 times higher than that of Non-Hispanic Whites with asthma, [60] which is usually due to the lack of access to quality ambulatory care. Gupta and colleagues states in his article that asthmatic African American children are more likely to receive care in the emergency department as compared to their Whites peers.[15] According to their report in 2009, the ALA states that asthma ranks third in causes for children hospitalization and was responsible for more than 679,000 emergency department visits in 2005.[14] The improvement of dedicated pediatric care is a recognized as one technique to guarantee that these minority children receive quality care for asthma in the emergency situation but

such method does not replace the need for primary care, and these dedicated services are usually not reasonably viable for smaller hospitals. In addition, asthma is a major cause of disturbance to the child's ability to perform daily tasks and is the most common reason for school absenteeism. Consequently, Minority children often have other risk factors for low performance at school, and their achievement at school decreases with repeated school absenteeism.[82, 83] The short-term effects of poor achievement at school cause difficulties for children and their families. However, the long-term effects of such low achievements may include lower educational opportunities and earning power for a lifetime. The evidence shows that childhood asthma has a negative impact on school attendance, which is a crucial and influential aspect of a child's life.[82-84] Table 3 provides a focused summary of the factors that contribute to asthma as a health problem for this population.

It is of great importance to note that access to treatment comprises more than access to a medical professional. A patient or family's capability to attain prescribed treatment is an important factor of self- management. This financial burden may lead families from low-income groups to choose between medications and other basic living needs, leading to gaps in medical management. Access to healthcare facilities may also be limited in acquiring appropriate testing and diagnostics in a timely manner. Evaluation and testing for allergic triggers and the employment of environmental control measures occur less often in low and middle-income households and among minority children.[85] In diseases such as asthma, access to quality care is key component in health outcomes.

2.8 CHILDHOOD ASTHMA AND PAYING FOR CARE: IMPACT OF INSURANCE COVERAGE

In the United States, a vast majority of low-income families do not have any health-care coverage, and they lack access to important health services. Around 44.3 million in US lack health insurance.[86] Owing to their inability to pay, individuals without health insurance have fewer office visits for preventive and follow-up care,[87,88] less continuity of care (even when they receive primary care),[89] and decreased utilization of appropriate diagnostic and therapeutic ambulatory procedures.[90] In turn, the uninsured have a larger number of unmet HCN, a greater dependency on emergency departments and hospitals for rescue care [91] and increased overall morbidity and mortality from chronic illnesses. Therefore, Andrulis point in his article that this difference in insurance has led some to consider that access to health services is the primary factor of health outcomes in society, and that by providing comprehensive access to medical care, the health outcomes of SES may be decreased.[92] Finally, Sin and colleagues mentioned in their article that notwithstanding the rationality, there is a lack of data to indicate that enhanced access improves health indicators such as a decrease in the incidence of rescue care in EDs.[93]

2.9 PREDISPOSING FEATURES OF CHILDHOOD ASTHMA

2.9.1 PRENATAL FACTORS

According to recent studies, maternal exposure to second hand smoke appeared to be one of the associated factors with the increased risk of childhood asthma development, regardless if the mother does not actively smoke during pregnancy.[95] Likewise, medical reports revealed that maternal smoking during pregnancy was responsible for increasing

the risk for childhood asthma and wheeze, which persists into adolescence.[96] Alternative studies stated that maternal hostile life trials during the second half of pregnancy increased the risk for development of childhood asthma, and eczema.[97] These conclusions might provide highlights on prospective means, which describe the inconsistency in clinical appearance of allergic diseases.[98]

2.9.2 LIKELIHOOD TOOLS

Nowadays most great attention is focused on recognizing clinical markers, genetic markers, and biomarkers related to the progress of asthma in children that could be useful to better understand the likelihood of disease development. For instance, TH17 cell is one cell of interest in these clinical markers era. TH17 cells usually develop before the 3 months age and maintain a predisposition to convert monitoring T cells till 12 months of age. As a result, this could be an essential gate in the care of tolerance for allergens.[99] Similarly, it has been also stated in the literature that asthmatic children during school age demonstrate an unusual resistant response to pathogenic bacteria in infancy and therefore may lead to chronic airway inflammation and childhood asthma. 20 There has been great work done to classify the genetic connections that could be related to asthma. According to Kerkhof et al, [100] there is association between at least 3 chronic disruptive pulmonary disease genes during the growth and development of lung, and this association is indicating decreased airway capabilities in early childhood. Chronic pulmonary disease genes could also be participants in the newborn's lung reaction to smoke exposure while in the womb and in early life.[100] This similar gene set also known IL33–IL-1

receptor–like 1 (IL1RL1) path for polymorphisms related to asthma and specific wheezing phenotypes during early childhood.[101]

On the other hand, there is a growing attention been focused on the role of BCG vaccination in treating childhood asthma. Based on the Manchester Community Asthma Study report, it has been concluded that any defensive influence of BCG vaccination on childhood asthma is more likely to be temporary.[102] Nevertheless, additional researchers discovered pregnancy at younger age at birth as well as higher infant weight gains were associated with childhood asthma outcomes.[103] Alternative research team stated that premature delivery is strongly associated with atopic asthma in Puerto Rican children and that possibly premature delivery could partially explain the increased asthma prevalence among this ethnic group.[104] One more point of interest, which has been described in the literature, was the association of inner-city winter birth children with asthma and the increased risk of food allergen sensitization.[105] Hence there are many interacting factors between the progress of asthma and allergy that can be a participating role in age-linked disease growth.

Environment, stress and diet have been widely demonstrated as strong risk factors in childhood asthma development. According Chiu and colleagues, [106] community violence before birth and air pollution can lead to respiratory health problems in urban children. Additionally, psychosocial stressors could also affect host resistance, for instance, physical pollution can lead to adverse outcomes. Other researchers stated that reduced variety of food during the first year of life might elevate the risk of childhood asthma.[107] Further team of researchers discovered that a greater amount of eicosapentaenoic acid

was associated with a reduced risk of asthma when cow's milk allergy was adjusted for as a known confounder.[108] However, all the above-mentioned studies focused on the relationship between childhood asthma and the role of dietary and environmental modifiers.

Further to the wide attention on diet stress and environment, an ongoing research and focus is being directed to another group of risk factors, such as, the impact of the micro biome in adjusting asthma development. In a recent study conducted by Lapin and colleagues, [109] they concluded that there is a significant association between the use of antibiotics and consequent asthma stands only in case of respiratory tract infections, and explain the nature of association by opposite causation or confounding by indication. In a recent study of the inner city environment by Lynch and colleagues, [110] they determined that children with the maximum exposure to specific allergens and bacteria during first year of life were less likely to have persistent wheeze and allergic sensitization. Consequently, this surveillance could lead to new preventive approaches throughout the utilization of high levels of specific allergens and bacteria early in life.

One of the major resources for better understanding to the natural history of asthma, are birth cohorts. For instance, the Multicenter Allergy Study, which was conducted in 5 German cities, stated that the prevalence of childhood asthma increase due to parental asthma and nasal allergy. They moved further to this and proposed that avoiding tobacco smoke exposure during pregnancy, receiving early childhood vaccination, and starting day care between 1.5 and 3 years of age could prevent or delay the progress of asthma.[111] In conclusion, childhood asthma develops in early ages and looks to be drastically

increasing, with a decline in the age of asthma diagnosis. Asthma prevalence differ in children less than 3 years old.[112] As a result, there is an constant efforts been made to streamline likelihood tools for recognizing children with wheeze or cough and who are at high risk for asthma. For example, Pescatore and colleagues [113] offered a noninvasive low cost and easy technique to expect the risk of later asthma in symptomatic preschool children.

2.9.3 CHILDHOOD ASTHMA OUTCOMES

One of the crucial benefits of the primary and proper childhood asthma managements is to decreasing the association of morbidity and respiratory disease in adulthood. In a Melbourne Asthma Study, a natural history of asthma study stated that adulthood clinical and lung function outcomes are strongly determined by severity of asthma in childhood. Moreover, the decreased lung function noticed in adulthood is started in childhood and less likely to decline quickly during the adulthood regardless of remaining symptoms.[114] It also seems that the development of childhood asthma severity has improved over the past 20 years, as implied by the decreased use of chronic glucocorticoids, better asthma management, and less glucocorticoid-stimulated adverse effects.[115] This has been interpreted as a result of the expansion and presentation of highly efficient medications, particularly 2nd generation inhaled corticosteroids (ICSs) and grouping ICSs/long acting bagonists.[115] Enhanced global association of healthcare and ongoing improvement of asthma guidelines similarly participated a causal role. Current asthma statistics by CDC have improved brings more consideration to those with the maximum asthma burden. According to recent report by Akinbami and colleagues [116] through the use of at-risk

analysis, which describes the disparities in asthma prevalence, to evaluate racial inequalities in asthma outcomes indicates that amongst children with asthma, there is no discrepancy for asthma attack prevalence and that advancement has been made in reducing discrepancies in asthma-related ED visit and hospitalization rates. As stated by Thakur and colleagues, [117] socioeconomic status has an important function in predicting asthma but has different outcomes based on race/ethnicity. Additional stages are essential to better recognize the risk factors throughout which socioeconomic status could activate these populations to prevent asthma. It also seems that specialty and training of the physician apply significant role in the nature of diagnostic examinations performed, the association of comorbid diagnoses, and the use of ICSs for asthma treatment. This might have consequences on asthma management and in future practice guidelines to determine some consistency of management opinions.[118] There are wide range of information is produced on clinical burden and predictors of asthma exacerbations in children with severe asthma. Notwithstanding the numerous long-term management medications, children with severe or complicated asthma treatments getting high pace levels of therapy exist with substantial clinical burden and correspondingly high risk of future asthma exacerbations.[119] These actions can be combined with EMR infrastructures to clinicians in a national health program approach to classify children at high risk for asthma or asthma exacerbations and children who may develop severe asthma.

2.10 PREDESPOSING DESCRIPTIONS OF CHILDHOOD ASTHMA

2.10.1 GENETIC FACTORS – ALLELEIC AND EPIGENETIC

In this era of genetics discoveries and advancements, the science of genetics embraces great potential to deliver better understanding of the hereditary features of asthma as well as the triggered paths creating the disease. In a study conducted by Sharma et al,[120] they described the benefit of appearance measurable feature locus mapping in the association of novel asthma genes and specified evidence for the significance of the CD41+ lymphocyte variants fatty acid desaturase (FADS2), N-acetyl-α-D-galactosaminidas (NAGA), and Factor XIII, A1 (F13A1), in the pathogenesis of asthma. Schieck and colleagues on the other hand [121] elaborated in their study more on genetic variation in TH17 pathway genes, childhood asthma, and total serum IgE levels. They determined that this knowledge about adjustable expression could be beneficial in recognizing different approaches to target TH17 genes and TH17-related mechanisms for therapeutic purposes in diverse groups of diseases in the coming future.[121] In alternative innovative study, researchers used epithelial genes to highlight the significance of utilizing biology science to encourage more initiatives to distinguish the genetic susceptibility of many complicated diseases.[122] In addition, this same study advocates the impact of anti–thymic stromal lymphopoietin therapy on asthmatic children. On the other hand, Genetic variations have also been involved with the associated biomarkers with eosinophilic airway inflammation, more specifically, section of exhaled nitric oxide. This process involved the attachment of [123] three variations to exhaled nitric oxide regulation.[123] Based on the variety of literature studying genetics, it is of great important

for genetics studies to be conducted in a variety of populations which are described by race/ethnicity since genetic markers can differ from discoveries based on European populations.[124,125] Such type of studies may be essential in explaining the differences and as well as the risks for health disparities in a population. Similarly, the science of genetics (mainly epigenetics) could provide another useful pathway to understand asthma through the better understanding of the relationship between chronic psychosocial stress and the onset and course of asthma. Many studies in the literature have already started to emphasize detailed pathways in which stress controls epigenetic and the conversion of DNA to RNA activity in asthma-relevant cells as well as detecting the predisposing genes that may converse risk for stress-related asthma exacerbations.[126] Therefore, this new advancement in genetic science could possibly offer better understanding to childhood asthma management and prevention.

2.10.2 BIOMARKERS

Another potential branch of science is the utilization of biomarkers through the process of classifying the biomarkers of interest, which could be of valuable use in opening new avenues to understand the activation process of diseases and the responsible predictors for treatment response and observing disease activity. In relation to this, the investigator Poole and colleagues [127] have conducted such a study using the nasal transcriptome as a proxy for the lung airway transcriptome in asthmatic patients. As a result, the investigators determined that nasal airway gene expression outlines largely recapitulate expression outlines in lung airways. On a different study involving biomarkers, Romeo and colleagues [128] add a new perception on the existing and

developing treatments that target TH2-promoting receptor complexes. The proper interpretation of the molecular features of exact receptors might deliver better idea about different disease phenotypes and the adjustable effects from such targeted therapies. Correspondingly, another team of researchers such as Harvina and colleagues [129] suggested that by understanding the proper means to inhibit receptor activation on mast cells and basophils may result in developing novel therapeutics regarding the different molecules on mast cells and basophils. Hence it can be concluded that proper understanding of biomarkers and markers of cell activation is going to be a big jump toward customizing treatment strategies.

2.10.3 ENVIRONMENT EXPOSURES

There is accumulating evidence in the literature supporting the fact that outdoor air pollution can trigger exacerbations of pre-existing asthma through traffic and power generation, but inconstant evidence on its relation to new asthma onsets. It is believed that the method is associated with oxidative damage to the airways, resulting in inflammation, remodeling, and increased risk of sensitization.[130] The developing science proposes that environmental exposures during pregnancy and early childhood years elevate the risk for asthma.[131] For instance, Miller and Peden [131] has reviewed the these techniques which might be associated with transformed innate and adaptive immunity through gene-environment exchanges and epigenetic modulation. Besides to reducing the pollutant source, it might be of high importance to develop new therapeutic interventions to decrease asthma morbidity. In a recent study by Matsui and colleagues, [132] the impact of household airborne endotoxin exposure on asthma are adapted by co-exposure to air

nicotine and nitric oxide. These toxins have reverse effects on the relationships between endotoxin and asthma-related outcomes. Moreover, Fabian and colleagues obtained in their study [133] to assess the effect of developing interventions related to the quality of indoor air used by pediatric health care and consequently develop a childhood asthma model as an instrument to arrange individual building interventions. Contrary to this model is the model established by Brandt and colleagues [134] which separate the costs of local air pollution and surrounding highway air pollution exposure and finally summarized that the cost of air pollution is a significant burden on families and contribute to economic loss for a society.

2.10.4 IMMUNE DYSFUNCTION BASED-DISEASES AND CHILDHOOD ASTHMA

The major function of epithelial cells is to create a barrier to the outside surrounding at the first line of mucosal immunity. The mechanism of how damaged epithelial barrier function has been reviewed by Georas and Rezaee [135] and they concluded that these damaged cells may be linked to TH2 division in asthmatic patients and suggest a regulator model of barrier malfunction that links the extent of inhaled allergen particles as a major factor affecting adaptive immunity. It is also acknowledged in the literature that lung epithelial cells distinguish allergens through expression of form recognition receptors and mount an intrinsic immune response. Consequently epithelial cells might be essential in controlling the outcome of allergen inhalation.[136]

2.10.5 INFECTION

The role of viral infections and microbial organisms in the acute and chronic indicators of asthma has been under repeated interest for researchers and clinicians. For instance, Iwasaki and colleagues [137] have assessed the responses of antibody to every human rhinovirus species in asthmatic and non-asthmatic children with a known type of human rhinovirus infection. Interestingly, they found that low species-specific human rhino-virus C levels, regardless whether the virus was found in both asthmatic and non-asthmatic children, therefore, implying a less efficient immune response to these species. Furthermore, the detection of specific bacterial pathogens could also be enhanced through rhinovirus infection in children with and without asthma, recommending, that *Moraxella catarrhalis* and *Streptococcus pneumonia* participate in the severity of respiratory tract diseases, such as asthma exacerbations.[138] Likewise, bacterial migration of the airways in newborns is in the form of *S pneumoniae*, *Haemophilus influenzae*, or *M catarrhalis* has been linked with the high risk of *pneumonia* and *bronchiolitis* in early childhood independent of coexisting asthma.[139] Consequently, this will lead to understand the role of bacterial infections in the resulting increase of respiratory tract infections and asthma.[140] By better understanding to the nature of effects of asthma or other atopic conditions on the risk of microbial infections, this will lead to opening new perceptions in clinical practice, research, and public health concerning atopic conditions.[141] Additionally, extra research to study the cause/effects of asthma must be compared because they are self-guidance to one another.[141] Studies revealed that child with asthma and other atopic diseases shown to be at higher risk for bacterial infections. Such type of studies will be of valuable outcomes to figure out whether damaged immune responses to

bacterial infections in asthmatic patients do exist and whether increased exposure to bacteria significantly has greater severity of viral infections.[142]

2.10.6 PRETERM DELIVERY AND CHILDHOOD ASTHMA

Fetal development disorders influence individuals to cardiovascular diseases and diabetes in a later age.[143] In the literature it has been proposed that the fetal period and early childhood has essential role for asthma and other allergic diseases.[144] Shorter duration of pregnancy has been found an indicator of disorder in fetal development, and preterm delivery stops normal fetal development, therefore, might increase chances to abnormal health. Accordingly, the association between preterm delivery and the risk of asthma would be constant by hypothesizing that the fetal period has an important role in the development of asthma. Moreover, detection of prematurity as a cause of asthma might have clinical meaning because it would serve active treatment of any physiologic airflow obstruction in preterm babies.[145] Furthermore, preterm delivered children might get special consideration with regard to preventive procedures against known or suspected environmental and nutritional causes of asthma. Conversely, prior studies in the literature, which have assessed the association between preterm delivery and asthma, have provided confounding results.

3.0 CHAPTER III RESEARCH METHODS

3.1 RESEARCH DESING

This is a secondary analysis and cross sectional study. The outcome variables are asthma and asthma severity. The independent variables including age, gender, race/ethnicity, region, insurance status, parental education and income level. Additional clinically related variables were also considered in analysis as predictors. These variables included child's health premature delivery, birth weight, depression, developmental delay and uses of cigarettes, cigars, or pipe tobacco. It will be determined whether childhood asthma can be predicted by the predicted clinically relevant factors. Furthermore, the nature of association between childhood asthma severity and the different SES and/or Racial/Ethnicity among minority children living in US will be evaluated as well. Eligible asthmatic children will be sampled and stratified by age, gender, race/ethnicity, insurance status, parental education, and income level.

3.2 DATA SOURCES AND DATA ELEMENTS

The source of data used in the study is the National Survey of Children's Health (NSCH) (94). The NSCH provides a broad range of information about children's health and well-being collected in a manner that allows comparisons among states as well as nationally. The NSCH survey is designed to:

(A) Estimate national and state-level prevalence for a variety of child health indicators,

(B) Generate information about children, families, and neighborhoods to help guide policymakers, advocates, and researchers,

(C) Provide baseline estimates for federal and state performance measures, Healthy People 2020 objectives and state-level needs assessments, and

(D) Complement the National Survey of Children with Special Health Care Needs (NS-CSHCN).

For this research, data from the 2011-2012 survey will be employed to conduct the required statistical test using SAS SOFTWARE to answer the addressed research questions stated at the body of this research proposal. A total of 95,677 NSCH interviews were completed nationally by parents/caregivers of children and youth approximately 1,876 in each state and the District of Columbia, ranging from 1,811 in South Dakota to 2,200 in Texas. Survey results are adjusted and weighted to reflect the demographic composition of non-institutionalized children and youth age 0–17 in each state. Questionnaire topics include demographics, health and functional status, health insurance coverage, health care access and utilization, medical home, early childhood (0–5 years) issues, issues specific to middle childhood and adolescence (6–17 years), family functioning, parental health status and neighborhood and community characteristics. The dataset file single SAS DATASET file and was released with data for 95,677 children (one record for each age-eligible child that was randomly selected to be the subject of the interview). Each record contains all interview data for the child and the household in which the child resides, including the child's health and health care, family functioning,

parental health, neighborhood and community characteristics, health insurance coverage, and demographics.

3.3 STUDY VARIABLES

The outcome variables used in this study were a sample child's diagnosis of asthma, defined by the answer to the survey question: Has a doctor or other health care provider ever told you that the selected child [S.C.] had asthma? According to an adult parent/guardian, a sample child's currently have asthma, defined by the answer to the survey question: Does the [S.C.] currently have asthma? And the third outcome was a sample child's severity of asthma, defined by the answer to the survey question: Would you describe [His\Her] asthma as mild, moderate, or severe? The primary explanatory variables were the child's race/ethnicity as reported by an adult parent/guardian, and the family socioeconomic status (SES): parental or guardian education and income based on the FPL threshold in 2011. The following race/ethnicity categories were used in the present study: Hispanic, non-Hispanic white, non-Hispanic black, multi-racial/ other non-Hispanic; the educational level of the child's parent or guardian in the household (less than a high school; high school graduate and more than high school). In the NSCH dataset, the income to FPL ratio was considered by dividing family income by the poverty threshold (FPL; $22,350 for a family of 4 in 2011). We obtained four family income levels based on the NSCH categorization of family income as 0-99% FPL, 100-199 FPL, 200-399 FPL and 400 FPL or more.

Other covariates were selected based on previous studies and theoretical concerns, included the following: child's gender (male and female); child's age (0-5 years, 6–11

years, 12–17 years). HRSA Region as divided by the Health Resources and Service Administration (HRSA) into 10 regions: Region I: (CT, ME, MA, NH, RI, VT), Region II: (NJ, NY, PR, VI), Region III: (DE, DC, MD, PA, VA, WV), Region IV: (AL, GA, FL, KY, MS, NC, SC, TN), Region V: (IL, IN, MI, MN, OH, WI), Region VI: (AR, LA, NM, OK, TX), Region VII: (IA, MO, NE, KS), Region VIII: (CO, MT, ND, SD, UT, WY), Region IX: (AZ, CA, HI, NV, Guam, American Samoa, the Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Republic of the Marshall Islands, Republic of Palau)and Region X: (AK, ID, OR, WA). Additional clinically related variables were also considered in analysis as predictors. These variables included child's health premature delivery, birth weight, depression, developmental delay and uses of cigarettes, cigars, or pipe tobacco. The only sub-state geographic information included in the NSCH public use data set is a variable for Metropolitan Statistical Area (MSA) status. The MSA status information is available for the 35 states in which the population is at least 500,000 in both categories (MSA and non-MSA). Zip code data is collected with the NSCH; however, this information is not released in the public use data set due to confidentiality restrictions. Therefore, this variable was dropped from the study and replaced it with earlier mentioned HRSA Region variable (TABLE 1).

TABLE 1: Study Variables

Study Var	Var Name in NSCH Dataset	Var Describtion
Age	age3_11	1=0-5,2=6-11 and 3=12-17 (Categorical)
Gender	sex_11	1=Male and 2=Female (Categorical)
Race/Ethnicity	Race4_11	1=Hispanic, 2=White non-Hispanic, 3=Black non-Hispanic and 4=Multi-racial/other non-Hispanic (Categorical)
Parental Education Level	EDUC_PARR	1=Less than high school, 2=High school graduate and 3=More than high school
Household Income Level	povlev4_11	1=<100% FPL, 2=100-199% FPL,3=200-399% FPL and 4=400 or more FPL (Categorical)
Insurance Status	K3Q01	0=No and 1=Yes (Categorical)
Premature Delivery	ind1_7_11	0=No and 1=Yes (Categorical)
Birth Weight	ind1_8_11	1=Low Birth Weight (<2500g) and 2=Normal (Categorical)
Depression	depress_11	1=Yes and 2=No (Categorical)
Developmental Delay	devdelay_11	1=Yes and 2=No (Categorical)
Someone using Tobbaco	ind6_4_11	0=No and 1=Yes (Categorical)
Asthma Status	Asthma_11	1=No,2=Ever told but not now and 3=Yes (Categorical)
Asthma Severity	K2Q40C	1=Mild,2=Moderate and 3=Severe (Categorical)

3.4 NSCH SAMPLING DESING

The NSCH is a:

A- Cross-sectional telephone survey of US households with at least one resident child aged 0 to 17 years at the time of the interview

B- List-assisted random-digit-dial (RDD) sample of landline telephone numbers, supplemented with an independent RDD sample of cell-phone numbers

C- Complex survey design, with stratification by state and sample type (landline or cell-phone)

D- The NSCH used the same sampling frame as the CDC's National Immunization Survey (NIS) and immediately followed the NIS interview in selected households, using the NIS sample for efficiency and economy.

3.5 STATISTICAL ANALYSIS

The statistical analysis will begin with an estimation of the national prevalence of asthma among children aged 0-17 years and living in the United States. Characteristics of the sample population will then be compared with the general population to assess if there are differences or validate how the sample population represents the general population. The characteristic table will show how asthma and severe asthma levels are distributed among children by their age, gender, race/ethnicity, SES, and insurance status. A set of clinically relevant variables were added. The categorical variables included in this table will be displayed as frequencies and expressed by percentages. Following, the group of children with asthma and severe asthma conditions will be assessed for the sample and then stratified by SES, race/ethnicity and age category. Third, differences in the prevalence of each asthma and severe asthma conditions among children will be compared and tested using likelihood ratios and 2 X 2 tables based on clinically relevant variables. Finally, magnitude of the correlations between asthma risk factors and the sample characteristics will be assessed.

3.6 DATA ORGANIZATION AND CLEANING

The SAS Files will be sorted using the unique ID number for each household known as the identification number (IDNUMBER). Sorting the data is useful to guarantee that all observations are ordered in the exact order in which they are contained in the file. The

population will then be sub-stetted with a sample that has only the variables of interest using the KEEP command at DATA step. The sorted file will be copied into a single file using the unique identification numbers. The PROC CONTENTS procedure will be used to list contents of the copied file with the VARNUM option to order the listed variables according to their positions in the dataset. Categorical variables will then be created to help regulate cross tabulations of the outcome variable, asthma and severe asthma (Y), and its associated risks factors or covariates (X_{ys}), mainly; Age, Gender, Race/Ethnicity, SES (education and income level). Three age groups were identified as age group 0-5, 6-11 and 12-17 via data step programming using SAS procedures such as "If then, else" statements. Other selected variables will be re-coded for the purpose of grouping them by class labels. The classification will be useful in standardizing calculations. The outcome variables, asthma and severe asthma and the independent variables; age, gender, race/ethnicity and SES were all classified to facilitate generation of cross tabulations. The demographic variables, age, gender and race/ethnicity will be classified by the US Population Census Estimates stratification.

Responses of “Don’t know” or “Refused” and those with no response value will not be included in the analysis. In nearly all cases, the unknown values make up less than one percent of the responses to any single question. However, a few questions have higher proportions of unknown values, most notably household income (9.7% nationwide), and race/ethnicity (2.7% nationwide). The 2011-2012 NSCH public data file provided by the National Center for Health Statistics includes the household income variable POVERTY_LEVELR, which is missing values for 8,856 cases. This imputed income data files will be used to estimate household income for children with missing values.

The SAS codes for missing data will be coded as (.N, .L, .P, .M, .A for several legitimate skips in the original survey) and (6, 7 for don't know and refused to answer) based on the description of messiness by the National Center for Health Statistics (ABENDIXES Table 42).

In order not to bias analysis of the selected sample, weights will be applied to the selected sample variables using the final sampling weight variable (NSCHWT) provided in the SAS DATASET to account for sampling inequalities emanating from the sampling procedure. In the absence of these adjustments, it would be statistically invalid to infer results of the analysis on the entire US population. Imputed household income variable will be used to overcome the messiness in data in this variable. Regardless of the previous statistical adjustments, it is still not clear whether respondents' geographical location can be used to explain variations in the distribution of asthma among children aged 0-17 years living in the United States.

4.0 CHAPTER IV RESULTS

4.1 PREVALENCE OF CHILDHOOD ASTHMA IN US

The frequency analysis of the data showed that the majority of US children were older children within the age group 12-17 years old (34,601; 34.1%), followed by 6-11 years (31,079; 33.2%) compared with younger ages 0-5 years (29,997; 32.7%) (TABLE 3).

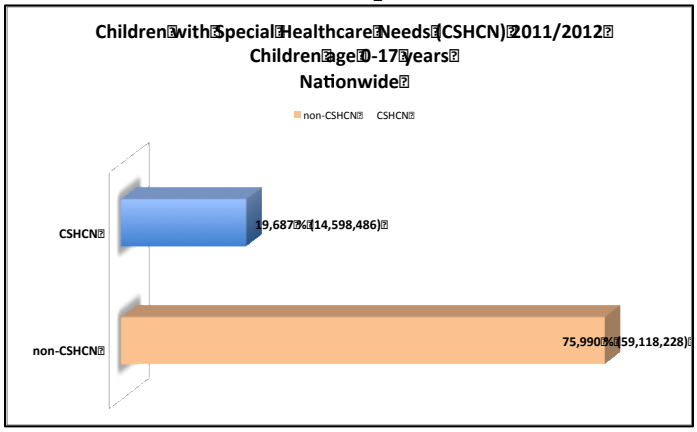
TABLE 2: Children by 3 age groups.

	0-5 years	6-11 years	12-17 years	Total %
%	32.7	33.2	34.1	100
C.I.	(32.1 – 33.4)	(32.5 – 33.9)	(33.4 – 34.7)	
n	29,997	31,079	34,601	
Pop. Est.	24,130,580	24,475,924	25,110,210	

The majority of children are in the age group 12-17 (34.1%) compared to the ages of 0-5 and 6-11, (32.7% and 33.2%) respectively.

Between 2011-2012, there are an estimated 14.6 million children with special healthcare needs in United States (Figure 1).

FIGURE 1: Children with Special Healthcare Needs.

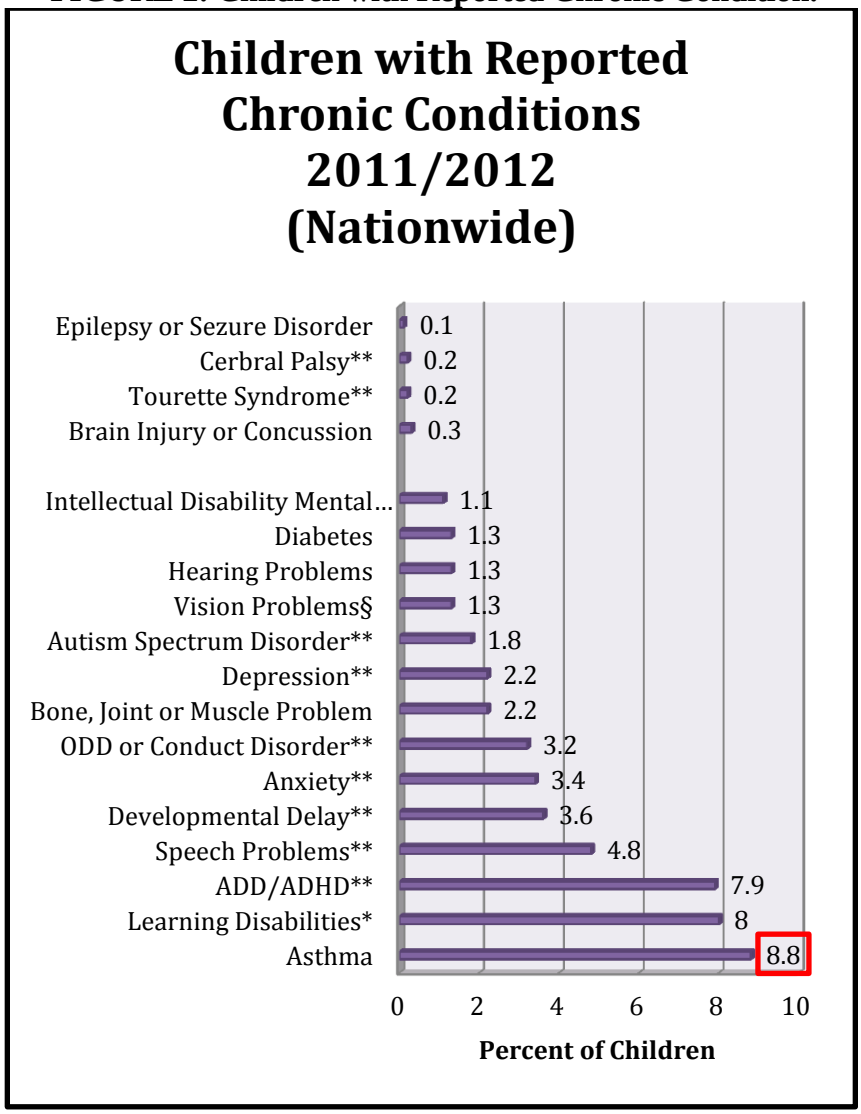


This includes an estimated 7 million child with 2 or more current chronic condition. Of the children who currently have at least one chronic condition, approximately half (49.8 percent) were reported to have at least one chronic condition that was moderate or

severe in its impact on the child health. However, it is important to note that these are parents' reports of their children's conditions and was not confirmed with medical records.

Asthma was the most often reported condition between 2011/2012, occurring among 1 out of every 11.4 children, followed closely by learning disabilities (1 in 12.5 children aged 3-17 years) (Figure 2).

FIGURE 2: Children with Reported Chronic Condition.



Children aged 3-17 years only*, children aged 2-17 years only**, includes only problems that cannot be corrected with glasses or contact lenses**§.**

Similarly, asthmatic children are estimated to be around 10.5 million (14.5 %) children between 2011-2012 (Table 3) and the incidence of asthma have been noticed at higher rates among older children aged 6-11 and 12-17 years (16.1 and 18.5% respectively) compared with younger age groups aged 0-5 years (8.6%) (Table 5) (FIGURE 5).

TABLE 3: Prevalence of childhood asthma

	Do not have condition	Had condition at some point, but not currently	Currently have condition	Total %
%	**85.5**	**5.7**	**8.8**	**100**
n	82,046	5,193	8,229	
Pop. Est.	**62,864,564**	**4,211,382**	**6,469,621**	

FIGURE 3: Prevalence of current asthma.

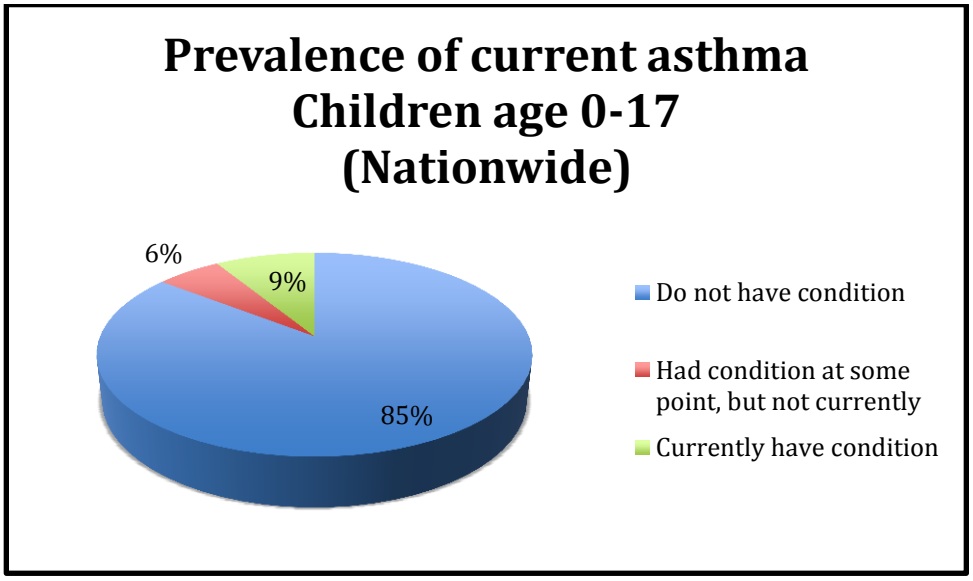


TABLE 4: Parent-rated severity of current asthma

	Do not currently have condition	Currently have condition, rated as mild	Currently have condition, rated as moderate or severe	Total %
%	**91.2**	**6.3**	**2.5**	**100**
C.I.	(90.8 - 91.6)	(5.9 - 6.6)	(2.3 - 2.7)	
n	**87,239**	**6,080**	**2,134**	
Pop. Est.	67,075,946	4,619,944	1,839,668	

Parent of children with asthma condition rated the severity of their child's asthma as mild, moderate or severe (6.3% and 2.5% respectively) and counting for a total sample size (N) = 8,214 child.

FIGURE 4: Parent-rated severity of current asthma.

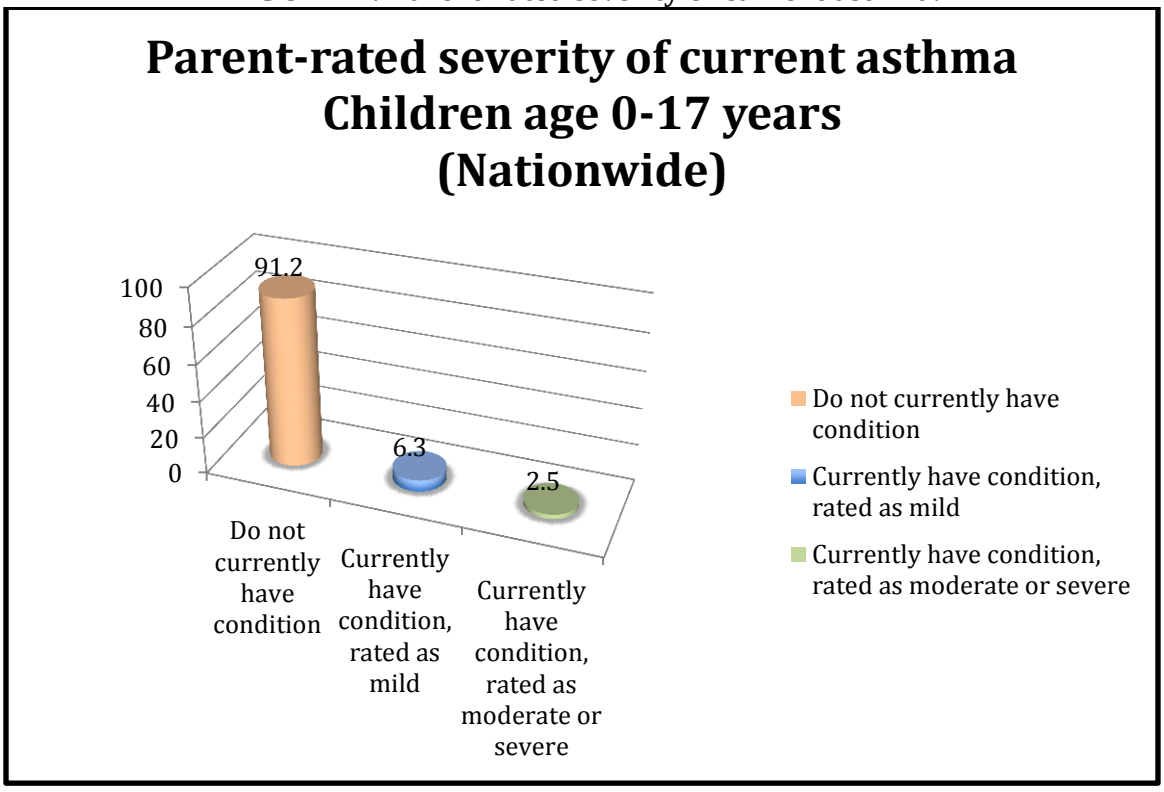
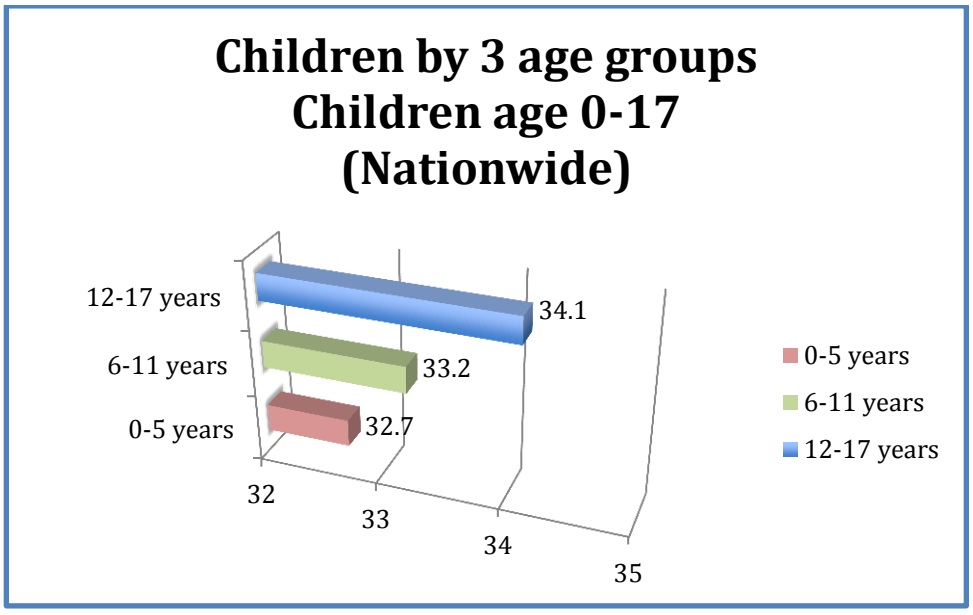


TABLE 5: Prevalence of childhood asthma by age group.

		Do not have condition	Had condition at some point, but not currently	Currently have condition	Total %
0-5 yrs old	%	**91.4**	**2.8**	**5.8**	**100**
	n	27,654	673	1,606	
	Pop. Est.	**21,992,560**	**676,497**	**1,403,273**	
6-11 yrs old	%	**83.8**	**6.1**	**10**	**100**
	n	26,100	1,812	3,117	
	Pop. Est.	**20,482,440**	**1,499,534**	**2,455,936**	
12-17 yrs	%	**81.4**	**8.1**	**10.4**	**100**
	n	28,292	2,708	3,506	
	Pop. Est.	**20,389,564**	**2,035,351**	**2,610,412**	

FIGURE 5: Children by 3 age groups.



The total sample size of children is (N) = 95,677, and it's divided among different age groups as following: 0-5 years old= 29,997, 6-11 years old= 31,079 and 12-17 years old counts for 34,601.

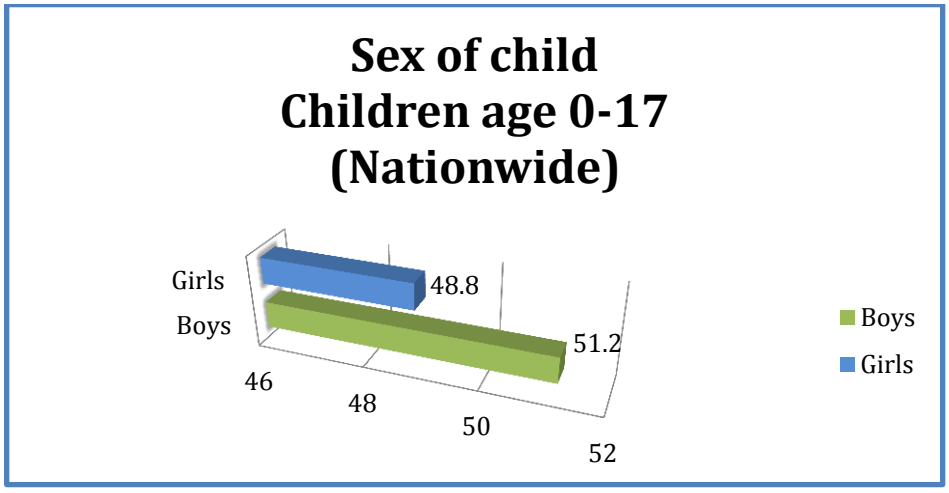
4.2 PREVALENCE OF CHILDHOOD ASTHMA AMONG DIFFERENT RACE/ETHNICITIES AND GENDERS

As can be seen in Table 6 and Figure 6, the US population was divided into 41,260 boys and 40,692 girls between 2011/2012. In terms of population gender of asthmatic children between 2011/2012, the incidence of asthma was more prevailed among boys (7,840; 15.9 %) higher compared to girls (2,058; 13.1 %) (Figure 6).

TALBE 6: Prevalence of childhood asthma among different genders

		Do not have condition	Had condition at some point, but not currently	Currently have condition	Total %
Boys	**%**	**84.1**	**6.3**	**9.6**	**100**
	C.I.	(83.4 84.8)	(5.9 6.8)	(9.0 10.1)	
	n	41,260	3,129	4,711	
	Pop. Est.	**31,583,039**	**2,376,495**	**3,601,146**	
Girls	**%**	**86.9**	**5.1**	**8**	**100**
	C.I.	(86.2 87.6)	(4.6 5.6)	(7.4 8.5)	
	n	40,692	2,058	3,509	
	Pop. Est.	**31,213,643**	**1,831,433**	**2,855,977**	

FIGURE 6: Sex of child.



As can be illustrated from the above Chart, the percentage of boys in the sample was slightly (2.4%) higher than the percentage of girls. The actual number of boys compared to girls was 49,219 boys and 46,349 girls

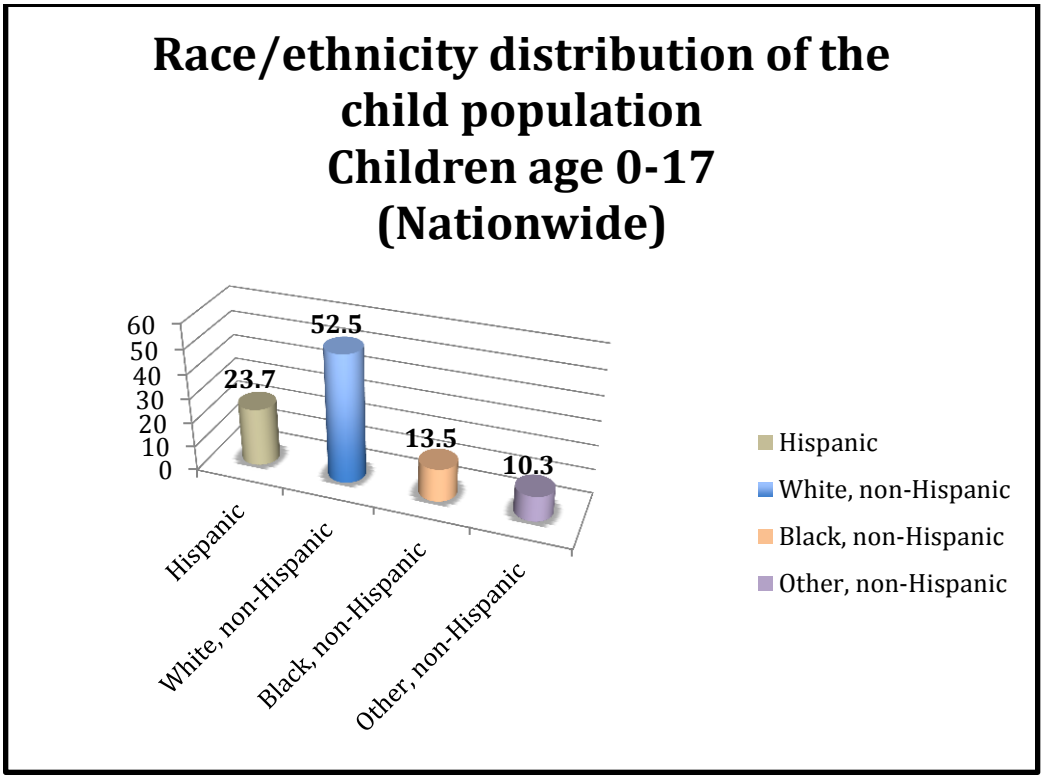
In regard to the race/ethnicity of the sample population, the majority were white Non-Hispanic children 53,597, Hispanic 10,817, Other Non-Hispanic 8,798 and Black Non-Hispanic 6,854 (TABLE 7).

TABLE 7: Race/ethnicity distribution of the child population.

	Hispanic	White, non-Hispanic	Black, non-Hispanic	Other, non-Hispanic	Total
%	23.7	52.5	13.5	10.3	100
C.I.	(22.9 – 24.4)	(51.8 – 53.2)	(13.1 – 14.0)	(9.8 – 10.7)	
n	12,682	61,381	8,875	10,446	
Pop. Est.	16,967,102	37,666,681	9,708,850	7,363,500	

White children accounts for more than half (52.5%) the US population compared to Hispanic, Black non-Hispanic and Multi-racial/other non-Hispanic.

FIGURE 7: Race/Ethnicity distribution of the child population in US.



However, even though Black non-Hispanic children were the smallest racial/ethnic group, they had the highest asthma prevalence compared to the rest of racial/ethnic groups Black Non-Hispanic 22.8%, Other Non-Hispanic 14.5%, Hispanic 13.1% and White Non-Hispanic 13%. Moreover, it can be illustrated from the table below that Black Non-Hispanic children were about 3 times more than white non-Hispanic children to have moderate or severe asthma (Table 8).

TABLE 8: Prevalence of childhood asthma among different races.

		Do not have condition	Had condition at some point, but not currently	Currently have condition	Total %
Hispanic	%	86.9	6.3	6.8	100
	n	10,817	814	1,029	
	Pop. Est.	14,713,047	1,061,098	1,153,190	
White, non-Hispanic	%	87	5.4	7.6	100
	n	53,597	3,062	4,599	
	Pop. Est.	32,695,075	2,040,731	2,852,928	
Black, non-Hispanic	%	77.2	6	16.8	100
	n	6,854	543	1,448	
	Pop. Est.	7,479,937	577,590	1,625,290	
Other, non-Hispanic	%	85.2	5.4	9.5	100
	n	8,798	657	971	
	Pop. Est.	6,264,123	394,374	694,916	

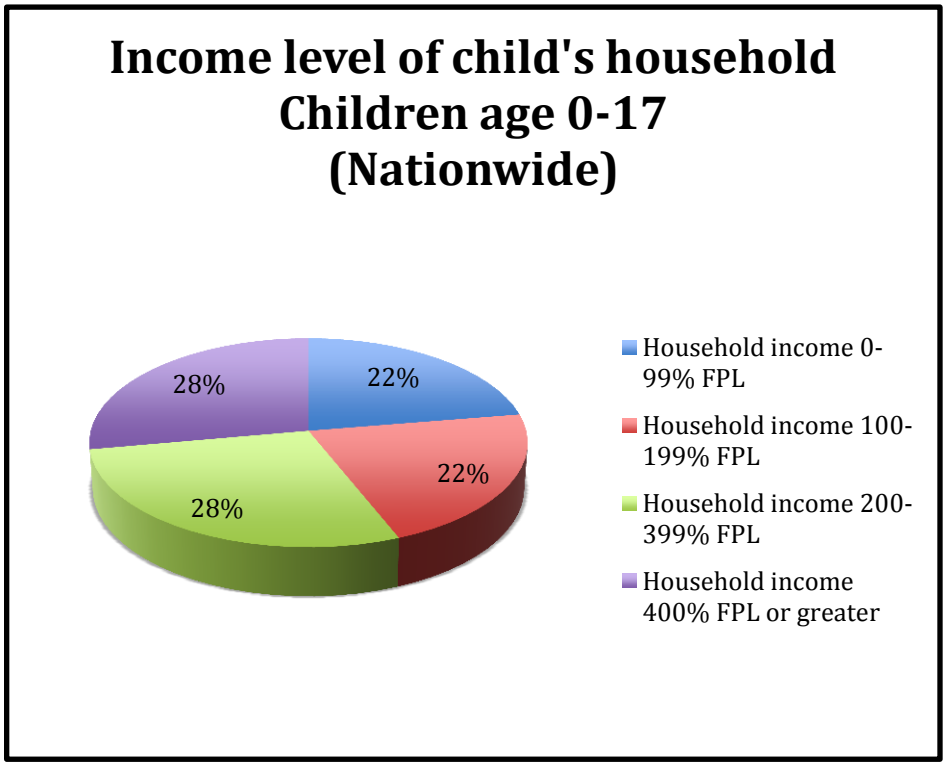
Furthermore, majority of children population were at the 400% FPL or more (34,642; 27.8%) followed by 200-399% FPL or more (28,942; 28.2%) then below 100% FPL (14,928; 22.4%) (Table 9; Figure 8).

TABLE 9: Income Level of Child's Household.

	Household income 0-99% FPL	Household income 100-199% FPL	Household income 200-399% FPL	Household income 400% FPL or greater	Total %
%	22.4	21.5	28.2	27.8	100
C.I.	(21.8 – 23.1)	(20.9 – 22.2)	(27.6 – 28.8)	(27.2 – 28.4)	
n	14,928	17,183	28,924	34,642	
Pop. Est.	16,549,336	15,878,815	20,778,480	20,510,083	

Based on the Federal Poverty Level (FPL) Guidelines the income level of Child's Household is distributed with small margins between the four income levels. Children from households income 0-199% FPL accounts for 43.9% compared to those with income 200- 400 FPL or greater with 56%.

FIGURE 8: Income level of child's household.



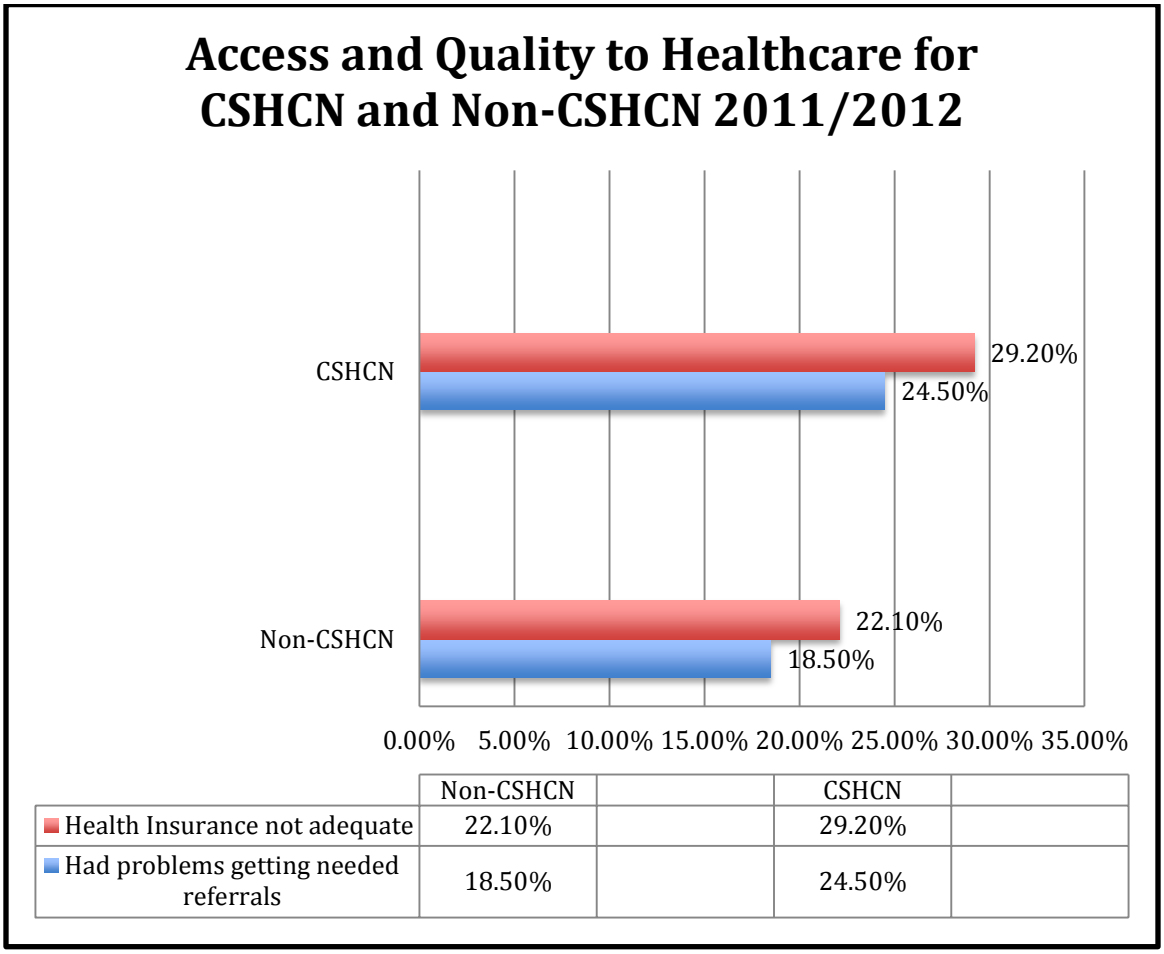
However, children who live in household below the federal poverty level (0-99 FPL) have higher incidence of moderate or serve asthma 659 (4.6%) compared to 463 (2.5%) (100-199 FPL), 551 (2.2%) (200-399 FPL) and 461 (2.2%) (400 FPL or higher) (Table 41). In regard to the prevalence of asthma, children who live in household <100% FPL reported higher rates of asthma (17.5%) followed by (15%) for those who live in households with 100-199% FPL, (13.6%) 200-399% FPL and the lowest are children live in households 400% FPL or higher (12.7%).

TABLE 10: Prevalence of childhood asthma by household income level.

		Do not have condition	Had condition at some point, but not currently	Currently have condition	Total %
0-99% FPL	%	**82.5**	**5.9**	**11.6**	**100**
	n	12,191	855	1,845	
	Pop. Est.	**13,628,928**	**967,437**	**1,913,743**	
100-199% FPL	%	**85.1**	**6.3**	**8.7**	**100**
	n	14,560	984	1,584	
	Pop. Est.	**13,461,021**	**991,670**	**1,370,513**	
200-399% FPL	%	**86.3**	**5.5**	**8.1**	**100**
	n	25,064	1,531	2,276	
	Pop. Est.	**17,899,247**	**1,150,622**	**1,684,983**	
400% FPL or higher	%	**87.3**	**5.4**	**7.3**	**100**
	n	30,231	1,823	2,524	
	Pop. Est.	**17,875,368**	**1,101,653**	**1,500,382**	

Moreover, children with special healthcare needs such as asthma control and management are 29.20% which is nearly 1.3% more than Non-CSHCN 24.50% whom health insurance is not adequate and are 1.3% more getting problems receiving the needed referrals to a specialist (Figure 9).

FIGURE 9: Access and Quality to Healthcare for CSHCN and Non-CSHCN 2011/2012.



	Non-CSHCN		CSHCN	
Health Insurance not adequate	22.10%		29.20%	
Had problems getting needed referrals	18.50%		24.50%	

4.3 SIGNIFICANCE OF THE ASSOCIATION BETWEEN CHILDHOOD ASTHMA/SEVERE ASTHMA, POPULATION CHARACTERSTICS AND RISK FACTORS

Table 11 illustrates the characteristics of the sample children and asthma/asthma severity prevalence by demographic class with all percentages weighted by using X^2 (Chisq) test. Overall, an estimated 14.6% and 8.8% of children had been reported with asthma and its level of severity respectively.

Table 11. Characteristics of children, as reported by parents/guardian in the household, and asthma/its severity level prevalence by demographic category

	Total Sample (N=95,677) Children aged 0-17	Asthma Prevalence (N=13,547) Children aged 0-17		Asthma Severity (N=8,214) Children aged 0-17 Weighted %		
	Weighted %	Weighted %	p-value	Mild	Moderate	Severe	p-value
AGE			<.0001[a]				0.0021[a]
0-5	32.7	8.7		63.9	29.1	7	
6-11 YEARS	33.2	16.3		72.2	23	4.8	
12-17 YEARS	34.1	18.7		74.9	20.1	4.9	
SEX (n=95,568)			<.0001	n=8,205			0.6056
Male	51.2	16		70.6	24.1	5.3	
Female	48.8	13.2		72.6	22.1	5.3	
RACE/ETHNICITY (n=93,384)			<.0001	n=8,033			0.0061
Hispanic	23.7	13.2[b]	0.8392	71.1	22.5	6.4	
White Non-Hispanic	52.5	13.1		75.9	20.3	3.8	
Black Non-Hispanic	13.5	22.9[c]	<.0001	65.7	27.4	6.9	
Multi-racial/other Non-Hispanic	10.3	14.9[d]	0.0035	67.8	26.2	6	
HOUSEHOLD INCOME LEVEL			<.0001				<.0001
<100% FPL	22.5	17.6[e]	<.0001	60.5	30.2	9.3	
100-199% FPL	21.5	15.1[f]	0.0008	71.5	23.1	5.4	
200-399% FPL	28.2	13.8[g]	0.018	73.1	23.1	3.7	
400% or more FPL	27.8	12.8		83.7	14.4	2	
PARENT/GUARDIAN EDUCATIONAL LEVEL (n=90,216)			0.0817	n=7,653			0.007
Less than High School	20.7	15.1[h]	0.2275	64.6	29.5	5.9	
High School Graduate	32.2	15.2[i]	0.5323	73.3	22.5	4.3	
More than High School	47.1	14		74.9	20.3	4.9	
HRSA REGION			<.0001	n=8,214			0.5273
Region I (CT, ME, MA, NH, RI, VT)	4.2	16.6		73.4	21.9	4.8	
Region II (NJ, NY, PR, VI)	8.6	15.8		70.6	22.8	6.6	
Region III (DE, DC, MD, PA, VA, WV)	9	16.3		72.7	21.7	5.6	
Region IV (AL, GA, FL, KY, MS, NC, SC, TN)	19.3	16.7		66.2	27.1	6.6	
Region V (IL, IN, MI, MN, OH, WI)	16.6	14.2		72.3	22.3	5.4	
Region VI (AR, LA, NM, OK, TX)	13.9	13.7		75.9	19.8	4.3	
Region VII (IA, MO, NE, KS)	4.5	13.1		73.6	21.7	4.7	
Region VIII (CO, MT, ND, SD, UT, WY)	3.8	10.7		67.7	25.8	6.5	
Region IX (AZ, CA, HI, NV, Guam, American Samoa, the Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Republic of the Marshall Islands, Republic of Palau)	16.1	13.5		73.6	23.6	2.8	
Region X (AK, ID, OR, WA)	4.1	11.8		75	19.8	5.2	
Premature Delivery (N=94,799)			<.0001				0.0814
No	88.5	13.8		72.5	22.5	5	
Yes	11.5	20.8		66.8	26.3	7	
Birth Weight (N=91,031)			<.0001				0.0253
Born with low Birth Weight (<2500g)	9.6	19.3		65.4	26	8.6	
Normal Birth Weight	90.4	14.1		71.9	23.3	4.8	
Depression (N=85,519)			<.0001				<.0001
Doesn't have condition	96.3	15.4		73.3	22.2	4.4	
Ever told but doesn't currently have condition	1.6	29.1		71.2	24.6	4.2	
Currently have condition	2.1	32.3		51.2	32.1	16.7	
Developmental Delay (N=85,496)			<.0001				<.0001
Doesn't have condition	94.3	15.4		74.2	21.1	4.7	
Ever told but doesn't currently have condition	2.1	24		59.6	34.5	5.8	
Currently have condition	3.6	24.5		54.1	38.3	7.6	
Tobacco use In house (N=94,754)			<.0001				0.0012
No	75.9	13.6		74	21.6	4.6	
Yes	24.1	18		66.3	26.5	7.2	
TOTAL	100	14.63					

[a] Chi Square test for association
[b] Hispanic vs. White non-Hispanic
[c] Black non-Hispanic vs. White non-Hispanic
[d] Multi-racial/other non-Hispanic vs. White non-Hispanic
[e] <100 FPL vs. 400 FPL or More
[f] 100-199 FPL vs. 400 FPL or More
[g] 200-399 FPL vs. 400 FPL or More
[h] Less than High School vs. More than High School
[i] High School Graduate vs. More than High School

The overall X^2 test for age and sex. For instance, the initial test revealed a strong association between age with childhood asthma (*p=<.0001) (*TABLE 12) but an association at the border line with level of asthma severity (*p= .0021*) (TABLE 13).

TABLE 12: X^2 test of association between childhood asthma and age.

Rao-Scott Chi-Square Test

Pearson Chi-Square	1369.4214
Design Correction	4.8813
Rao-Scott Chi-Square	280.5441
DF	2
Pr > ChiSq	<.0001
F Value	140.2720
Num DF	2
Den DF	190982
Pr > F	<.0001

Sample Size = 95593

TABLE 13: X^2 test of the association between severity of asthma and age

The SAS System

The SURVEYFREQ Procedure

Rao-Scott Chi-Square Test

Pearson Chi-Square	72.1688
Design Correction	4.2968
Rao-Scott Chi-Square	16.7960
DF	4
Pr > ChiSq	0.0021
F Value	4.1990
Num DF	4
Den DF	32448
Pr > F	0.0021

Sample Size = 8214

Next, the association between sex and childhood asthma has been evaluated as well to decide if this variable will fit for the next regression model to assess the nature and linearity of association between the study variables and the outcome variables. Consequently, sex of the child was also significantly associated with child hood asthma (*p= <.0001*) (TABLE 14) but the level of asthma severity was not associated with sex (*p= 0.6056*) (TABLE 15).

TABLE 14: X^2 test of the association between childhood asthma and sex.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	158.6176
Design Correction	4.9700
Rao-Scott Chi-Square	31.9148
DF	1
Pr > ChiSq	<.0001
F Value	31.9148
Num DF	1
Den DF	95382
Pr > F	<.0001
Sample Size = 95484	

TABLE 15: X^2 test of the association between severity of asthma and sex.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	4.3222
Design Correction	4.3093
Rao-Scott Chi-Square	1.0030
DF	2
Pr > ChiSq	0.6056
F Value	0.5015
Num DF	2
Den DF	16206
Pr > F	0.6056
Sample Size = 8205	

Further to this, we also assessed the overall significance of association between racial/ethnicity and the prevalence of asthma and its level of severity by using X^2 (Chisq) test. The majority of US population between 2011/2012 was white children Non-Hispanic (52.2 %), Hispanic (23.7 %), Black Non-Hispanic (13.5 %) and Multi-Racial/Other Non-Hispanic (10.3 %). Furthermore, the multiple logistic regression model in our study after we developed a model to assess the level of association between each and every racial/ethnic group compared to the reference group (White Non-Hispanic), has revealed that between 2011/2012 the Asthma was more prevailing among non-Hispanic black children (22.9%) than among non-Hispanic white children (reference group) 13.1% with strong statistical probability ($p=<.0001$) between Black Non-Hispanic and incidence of asthma. On the contrary, the prevalence of asthma did not differ significantly between Hispanic children (13.2%) and non-Hispanic white children (13.1%), and did not differ

significantly between children of multi-racial/other non-Hispanic (14.9%) and non-Hispanic white children. With regard to the severity prevalence of asthma, the overall association between race/ethnicity was less significant compared to prevalence of asthma in general (*p= 0.0061 vs. 0.0001*) (TABLE 16, 17).

TABLE 16: X^2 test of the association between asthma and race/ethnicity.

The SURVEYFREQ Procedure

Rao-Scott Chi-Square Test

Pearson Chi-Square	823.4211
Design Correction	5.1895
Rao-Scott Chi-Square	158.6710
DF	3
Pr > ChiSq	<.0001
F Value	52.8903
Num DF	3
Den DF	279627
Pr > F	<.0001

Sample Size = 93311

TABLE 17: X^2 test of association between asthma severity level and race/ethnicity.

The SURVEYFREQ Procedure

Rao-Scott Chi-Square Test

Pearson Chi-Square	83.1063
Design Correction	4.6077
Rao-Scott Chi-Square	18.0365
DF	6
Pr > ChiSq	0.0061
F Value	3.0061
Num DF	6
Den DF	47586
Pr > F	0.0061

Sample Size = 8033

In addition, the significance of association between asthma/asthma severity prevalence and income to FPL was strong at both levels with statistical probability of (*p*= <.*0001*) (TABLE 18).

TABLE 18: X^2 test of association between asthma and income to FPL.

The SURVEYFREQ Procedure

Rao-Scott Chi-Square Test

Pearson Chi-Square	244.5789
Design Correction	4.9131
Rao-Scott Chi-Square	49.7813
DF	3
Pr > ChiSq	<.0001
F Value	16.5938
Num DF	3
Den DF	286473
Pr > F	<.0001

Sample Size = 95593

TABLE 19: X^2 test of association between asthma severity and income to FPL.

Rao-Scott Chi-Square Test

Pearson Chi-Square	318.8849
Design Correction	4.1089
Rao-Scott Chi-Square	77.6075
DF	6
Pr > ChiSq	<.0001
F Value	12.9346
Num DF	6
Den DF	48672
Pr > F	<.0001

Sample Size = 8214

In contrast, when logistic regression model was developed to evaluate the magnitude and linearity of association asthma prevalence vary significantly based on the income-to-FPL, for instance, asthma was highly prevailed among poor children who live in households below 100-199 FPL (*p=<.0001; .0008 respectively*) compared with higher income levels (200-399 FPL; *p=.018)* and >400 FPL (reference group). The prevalence of asthma severity was strongly associated as the general prevalence of childhood asthma with income level.

In regard to parental/guardian level of education, the majority were households with more than high school education (46,688; 47.09%), high school graduate (30,691; 32.24%) and less than high school (12,837; 20.66%) respectively (TABLE 20). X^2 test illustrated no significance association between the prevalence of asthma and parental level of education (*p= 0.018*) (TABLE 21).

TABLE 20: Parental educational level.

	Less than high school	High school graduate	More than high school	Total %
%	**20.66**	**32.24**	**47.09**	**100**
C.I.	(20.2 21.2)	(31.7 32.7)	(46.6 47.6)	
n	**12,837**	**30,691**	**46,688**	
Pop. Est.	14,056,744	21,933,078	32,033,801	

Even-though the majority of children's parents are high or more than high school graduate, children with parent of less than high school education are estimated to around 14 million.

TABLE 21: X^2 test of association between asthma prevalence and parental/guardian education level.

Rao-Scott Chi-Square Test

Pearson Chi-Square	26.2466
Design Correction	5.2386
Rao-Scott Chi-Square	5.0102
DF	2
Pr > ChiSq	0.0817
F Value	2.5051
Num DF	2
Den DF	180092
Pr > F	0.0817

Sample Size = 90148

Contrary to the asthma prevalence, X^2 test determined a statistical significance, at boarder line, with asthma level of severity (*p= 0.007*) (TABLE 22).

TABLE 22: X^2 test of association between asthma severity level and parental/guardian level of education.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	60.3245
Design Correction	4.2792
Rao-Scott Chi-Square	14.0973
DF	4
Pr > ChiSq	0.0070
F Value	3.5243
Num DF	4
Den DF	30204
Pr > F	0.0070
Sample Size = 7653	

Similarly, the logistic regression model revealed that neither category of the educational level nor the overall educational level of parents and/or guardian is significantly associated with childhood asthma. For instance, the probability of parent with less than high school is (*p=0.2275)* and (*p=0.5323)* for parent with high school graduate level of education compared more than high school level of education level (reference group).

Region in which the survey has been completed has an overall significant association with the prevalence of childhood asthma only but no significance has been detected when compared to asthma severity level (*p= <.0001; 0.5273 respectively*) (TABLE 23, 24).

TABLE 23: X^2 test of association between asthma prevalence and region.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	211.3046
Design Correction	3.7313
Rao-Scott Chi-Square	56.6298
DF	9
Pr > ChiSq	<.0001
F Value	6.2922
Num DF	9
Den DF	859419
Pr > F	<.0001
Sample Size = 95593	

TABLE 24: X^2 test of association between asthma severity and region.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	58.7791
Design Correction	3.4702
Rao-Scott Chi-Square	16.9385
DF	18
Pr > ChiSq	0.5273
F Value	0.9410
Num DF	18
Den DF	146016
Pr > F	0.5273
Sample Size = 8214	

The clinically relevant variables in this study (premature delivery, birth weight, depression and developmental delay) were selected as predictors based on prior recommendations in the literature as strongly impacting the prevalence of childhood asthma and its level of severity. However, the literature didn't confirm that these predictors cannot be interpreted as cause and effect interaction with childhood asthma due to lack of enough evidence as of this date. Therefore, we decided to utilize them in our study to fill this gab in the literature by adding new findings to validate the nature of association between these clinically relevant predictors and childhood asthma.

The initial X^2 test of association between premature delivery and childhood asthma prevalence revealed strong association with probability of $p = <.0001$. On the other hand, the association was not significant with asthma level of severity ($p = 0.0814$) (TABLE 25).

TABLE 25: X^2 test of association between childhood asthma and premature delivery.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	382.4501
Design Correction	4.7163
Rao-Scott Chi-Square	81.0915
DF	1
Pr > ChiSq	<.0001
F Value	81.0915
Num DF	1
Den DF	94616
Pr > F	<.0001
Sample Size = 94718	

TABLE 26: X^2 test of association between asthma severity level and premature delivery.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	20.9828
Design Correction	4.1829
Rao-Scott Chi-Square	5.0163
DF	2
Pr > ChiSq	0.0814
F Value	2.5081
Num DF	2
Den DF	16042
Pr > F	0.0815
Sample Size = 8123	

Similarly, birth weight was also significantly associated with childhood asthma as can be seen in TABLE 27 (p= <.*0001*) (TABLE 27).

TABLE 27: X^2 test of association between childhood asthma and birth weight.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	170.1555
Design Correction	4.6205
Rao-Scott Chi-Square	36.8260
DF	1
Pr > ChiSq	<.0001
F Value	36.8260
Num DF	1
Den DF	90860
Pr > F	<.0001
Sample Size = 90962	

Same as for premature delivery, the association with asthma level of severity was not significant as can be implied from TABLE (*p= 0.0253*) (TABLE 28).

TABLE 28: X^2 test of association between asthma severity level and birth weight.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	33.8284
Design Correction	4.6001
Rao-Scott Chi-Square	7.3538
DF	2
Pr > ChiSq	0.0253
F Value	3.6769
Num DF	2
Den DF	15438
Pr > F	0.0253
Sample Size = 7821	

Depression surprisingly showed strong association with childhood asthma and its severity level prevalence with probability of p= <.0001 as TABLE 29, 30 confirms.

TABLE 29: X^2 test of association between childhood asthma and depression.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	564.7076
Design Correction	5.1970
Rao-Scott Chi-Square	108.6608
DF	2
Pr > ChiSq	<.0001
F Value	54.3304
Num DF	2
Den DF	170710
Pr > F	<.0001

Sample Size = 85457

TABLE 30: X^2 test of association between asthma severity level and depression.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	163.7891
Design Correction	3.2540
Rao-Scott Chi-Square	50.3353
DF	4
Pr > ChiSq	<.0001
F Value	12.5838
Num DF	4
Den DF	31612
Pr > F	<.0001

Sample Size = 8005

The developmental delay was also associated with strong significance with both childhood asthma prevalence as well as its level of severity with probability of p= <.0001 (TABLE 31, 32).

TABLE 31: X^2 test of association between childhood asthma prevalence and developmental delay.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	269.5614
Design Correction	3.8074
Rao-Scott Chi-Square	70.7996
DF	2
Pr > ChiSq	<.0001
F Value	35.3998
Num DF	2
Den DF	170660
Pr > F	<.0001
Sample Size = 85432	

TABLE 32: X^2 test of association between asthma level of severity and depression.

Rao-Scott Chi-Square Test

Pearson Chi-Square	129.3848
Design Correction	3.0170
Rao-Scott Chi-Square	42.8852
DF	4
Pr > ChiSq	<.0001
F Value	10.7213
Num DF	4
Den DF	31576
Pr > F	<.0001

Sample Size = 7996

The significance of association was strong enough between childhood asthma and household using tobacco in the house with probability *p=<.0001* (TABLE 33). However, the severity level of childhood asthma was not strongly associated with the use of tobacco by a household (*p=0.0012*) (TABLE 34).

TABLE 33: X^2 test of association between childhood asthma and someone using tobacco in the house.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	271.1453
Design Correction	4.7426
Rao-Scott Chi-Square	57.1718
DF	1
Pr > ChiSq	<.0001
F Value	57.1718
Num DF	1
Den DF	94572
Pr > F	<.0001
Sample Size = 94674	

TABLE 34: X^2 test of association between asthma severity and someone using tobacco in the house.

Rao-Scott Chi-Square Test

Pearson Chi-Square	57.1817
Design Correction	4.2302
Rao-Scott Chi-Square	13.5176
DF	2
Pr > ChiSq	0.0012
F Value	6.7588
Num DF	2
Den DF	16098
Pr > F	0.0012

Sample Size = 8151

Surprisingly, the insurance status at the time of survey was not associated with neither the prevalence of childhood asthma nor the level of asthma severity (*p=0.3144*; *p=0.1678* respectively) (TABLE 35, 36).

TABLE 35: X^2 test of association between prevalence of childhood asthma and insurance status.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	6.9945
Design Correction	6.9107
Rao-Scott Chi-Square	1.0121
DF	1
Pr > ChiSq	0.3144
F Value	1.0121
Num DF	1
Den DF	95327
Pr > F	0.3144
Sample Size = 95429	

TABLE 36: X^2 test of association between asthma severity and insurance status.

Rao-Scott Chi-Square Test	
Pearson Chi-Square	17.9421
Design Correction	5.0260
Rao-Scott Chi-Square	3.5699
DF	2
Pr > ChiSq	0.1678
F Value	1.7849
Num DF	2
Den DF	16196
Pr > F	0.1678
Sample Size = 8200	

4.4 THE ODD RATIOS OF HAVING ASTHMA AMONG DIFFEERNT CLINICALLY RELEVANT VARIABLES (PRIDCTORS):

We used logistic and multiple logistic regression models to assess associations between clinically relevant routes such as depression, premature delivery, low birth weight, developmental delay and someone uses tobacco and current asthma status. Analyses were adjusted for potential confounders, including maternal education and income level, sex, age, and region (TABLE 37).

TABLE 37: Survey logistic procedure for clinical predictors

The SURVEYLOGISTIC Procedure

Analysis of Maximum Likelihood Estimates

Parameter		DF	Estimate	Standard Error	Wald Chi-Square	Pr > ChiSq
sex_11	2	1	0.2289	0.0451	25.7078	<.0001
EDUC_PARR	1	1	0.0891	0.0719	1.5360	0.2152
EDUC_PARR	2	1	0.0422	0.0486	0.7516	0.3860
hrsareg	2	1	0.1534	0.0867	3.1288	0.0769
hrsareg	3	1	0.1339	0.0774	2.9959	0.0835
hrsareg	4	1	0.1947	0.0662	8.6593	0.0033
hrsareg	5	1	0.2926	0.0675	18.8043	<.0001
hrsareg	6	1	0.2932	0.0946	9.6109	0.0019
hrsareg	7	1	0.3060	0.0757	16.3308	<.0001
hrsareg	8	1	0.4396	0.0833	27.8389	<.0001
hrsareg	9	1	0.2210	0.0990	4.9775	0.0257
hrsareg	10	1	0.4010	0.0846	22.4663	<.0001
ind1_7_11	1	1	-0.4188	0.0720	33.8504	<.0001
ind1_8_11	1	1	-0.0760	0.0811	0.8786	0.3486
depress_11	2	1	-0.5688	0.1563	13.2364	0.0003
depress_11	3	1	-0.6843	0.1330	26.4531	<.0001
devdelay_11	2	1	-0.4047	0.1125	12.9526	0.0003
devdelay_11	3	1	-0.3479	0.0987	12.4141	0.0004
ind6_4_11	1	1	-0.2057	0.0509	16.3278	<.0001

Overall, after adjusting for relevant clinical variables in the multivariate analyses model, analysis revealed that the higher risk of asthma is among Black non-Hispanic children (OR 0.984 (95%CI 0.843-1.149); *p= <.0001*) relatively compared to non-

Hispanic white children (reference group). Similarly, Multi-racial/other non-Hispanic children were statistically significant at the border line (OR 0.809 (95%CI 0.704-0.9109); *p= 0.0035*) compared to white Non-Hispanic (reference group). On the other hand, our logistic model detected no statistical significance between Hispanic children and prevalence of asthma (OR 0.984 (95% CI 0.843-1.149); *p= 0.8392*) at the level of sub-groups after setting white Non-Hispanic as a reference group (Table 38).

TABLE 38: Association of race/ethnicity with asthma prevalence

	Adjusted OR (95% CI)		
Race/ethnicity		*p-value*	
Hispanic	0.984 (0.843-1.149)	0.8392	Reference
non-Hispanic White	Reference	Reference	Reference
Non-Hispanic Black	0.557 (0.493-0.630)	<.0001	Reference
Multi-racial/other non-Hispanic	0.809 (0.704-0.9109)	0.0035	Reference

NOTE: OR are calculated from multivriate model after adjusting for age, sex, parental/guardian education, HRSA Region.
p-value: propability of x.05
non-Hispanic White is the refrence group of asscociation

Therefore, we focused on racial difference and adding some clinically relevant variables that could justify the observed black vs. white difference in asthma rates among U.S population. Premature delivery, birth weight, depression, developmental delay and presence of someone use cigarettes, cigars, or pipe tobacco were selected based on their relevance to the prevalence of childhood asthma as being implied from the literature review. The clinically relevant variables were added to a multiple logistics model with adjusting for potential confounders. The reason for applying such adjustment is to avoid bias in result. For instance, the likelihood association between premature delivery (37 weeks or less) and the prevalence of childhood asthma was statistically significant with OR 0.654 and 95%CI (0.568-0.752) with adjusting the full-term delivery as reference. Similarly, the odds of low birth weight revealed strong likelihood of increasing the prevalence of childhood asthma if the baby born with less than 2500g (OR 0.928; 95%CI

0.792-1.087) (TABLE 40). In the same logistic model depression showed strong likelihood of impacting the prevalence of childhood asthma with odds ratios (OR 0.490; 95%CI 0.378-0.635). Similarly, the odds of developmental delay reflected high likelihood of increasing the chance of developing asthma (OR 0.704; 95%CI 0.580-0.855). Finally, the presence of someone using tobacco in the house has strong likelihood of developing asthma among US children aged 0-17 years between 2011/2012 (TABLE 38).

TABLE 39: Odds Ratio Estimates

The SURVEYLOGISTIC Procedure

Odds Ratio Estimates

Effect		Point Estimate	95% Wald Confidence Limits	
ind1_8_11	1 vs 2	0.927	0.791	1.086
depress_11	2 vs 1	0.566	0.417	0.769
depress_11	3 vs 1	0.504	0.389	0.655
devdelay_11	2 vs 1	0.667	0.535	0.832
devdelay_11	3 vs 1	0.706	0.582	0.857
ind6_4_11	1 vs 0	0.814	0.737	0.899

Association of Predicted Probabilities and Observed Responses

Percent Concordant	63.4	Somers' D	0.279
Percent Discordant	35.5	Gamma	0.282
Percent Tied	1.0	Tau-a	0.073
Pairs	745466085	c	0.639

Table 40: Differences in asthma prevalence among children, adjusted for clinically relevant variables.

Variable	Adjusted OR (95% CI)
Race/ethnicity	
Hispanic	0.958 (0.819-1.119)
Non-Hispanic White	Reference
Non-Hispanic Black	0.550 (0.484-0.624)
Multi-racial/Other Non-Hisapnic	0.812 (0.704-0.936)
Premature Delivery	
No	Reference
Yes	0.654 (0.568-0.752)
Birth Weight	
Born with low birth weight (<2500g)	Reference
Born with normal birth weight	0.928 (0.792-1.087)
Depression	
Doesn't have condition	Reference
Ever told but currently doesn't have condition	0.557 (0.410-0.756)
Currently have condition	0.490 (0.378-0.635)
Developmental Delay	
Doesn't have condition	Reference
Ever told but currently doesn't have condition	0.663 (0.533-0.824)
Currently have condition	0.704 (0.580-0.855)
Tobacco Use in house	
No	Reference
Yes	0.814 (0.737-0.899)
SES Variables	
PARENT/GUARDIAN EDUCATIONAL LEVEL	
Less than High School	1.090 (0.946-1.256)
High School Graduate	1.031 (0.938-1.13)
More than High School	Reference
HRSA REGION	
Region I (CT, ME, MA, NH, RI, VT)	Reference
Region II (NJ, NY, PR, VI)	1.166 (0.984-1.382)
Region III (DE, DC, MD, PA, VA, WV)	1.143 (0.982-1.331)
Region IV (AL, GA, FL, KY, MS, NC, SC, TN)	1.215 (1.067-1.383)
Region V (IL, IN, MI, MN, OH, WI)	1.340 (1.174-1.529)
Region VI (AR, LA, NM, OK, TX)	1.341 (1.114-1.614)
Region VII (IA, MO, NE, KS)	1.358 (1.171-1.575)
Region VIII (CO, MT, ND, SD, UT, WY)	1.552 (1.318-1.827)
Region IX (AZ, CA, HI, NV, Guam, American Samoa, the Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Republic of the Marshall Islands, Republic of Palau)	1.247 (1.027-1.514)
Region X (AK, ID, OR, WA)	1.493 (1.265-1.763)
AGE	
0-5 years	Reference
6-11 years	0.632 (0.560-0.713)
12-17 years	0.519 (0.460-0.586)
SEX	
Male	0.795 (0.728-0.869)
Female	Reference

NOTE: ORs are adjusted for all variables included in the table.
OR = odds ratio
CI = confidence interval
FPL = federal poverty level

TABLE 41: General Characteristics of US Children with Asthma/Severe Asthma.

	Asthma Status				Severity of Asthma			
	Do not have condition	Had condition at some point, but not currently	Currently have condition		Do not currently have condition	Currently have condition, rated as mild	Currently have condition, rated as moderate or severe	
	N (%)	N (%)	N (%)	TOTAL N (%)	N (%)	N (%)	N (%)	TOTAL N (%)
Age								
0-5 years	27,654 (91.4)	673 (2.8)	1,606 (5.8)	**29,933 (100)**	28,327 (94.2)	1,121 (3.7)	477 (2.1)	**29,925 (100)**
6-11 years	26,100 (83.8)	1,812 (6.1)	3,117 (10)	**31,029 (100)**	2,7912 (90)	2,287 (7.2)	829 (2.6)	**31,028 (100)**
12-17 years	28,292 (81.4)	2,708 (8.1)	3,506 (10.4)	**34,506 (100)**	31,000 (89.6)	2,672 (7.8)	829 (2.6)	**34,501 (100)**
Total (N) and Est. Pop	**66,794,564**	**3,535,562**	**1,409,896**	**95,468 (100)**	**67,075,946**	**3,727,139**	**1,839,668**	**95,453 (100)**
Gender								
Male	41,260 (84.1)	3,129 (6.3)	4,711 (9.6)	**49,100 (100)**	44,389 (90.4)	3,470 (6.8)	1,232 (2.8)	**49,091 (100)**
Female	40,692 (86.9)	2,058 (5.1)	3,509 (8)	**46,259 (100)**	42,750 (92.1)	3,470 (5.6)	901 (2.2)	**47,121 (100)**
Total (N) and Est. Pop	**62,796,682**	**4,207,928**	**6,457,123**	**95359 (100)**	**67,004,610**	**4,607,639**	**1,839,475**	
Race\Ethnicity								
White, Non-Hespanic	53,597 (87)	3,062 (5.4)	4,599 (7.6)	**61,258 (100)**	56,659 (92.4)	3,552 (5.8)	1,044 (1.8)	**61,255 (100)**
Black, Non-Hespanic	6,854 (77.2)	543 (6)	1,448 (16.8)	**8,845 (100)**	7,397 (83.3)	967 (11)	477 (5.8)	**8,841 (100)**
Other, Non-Hespanic	8,798 (85.2)	652 (5.4)	971 (9.5)	**10,421 (100)**	9,455 (90.6)	706 (6.4)	263 (3)	**10,424 (100)**
Hespanic	10,817 (86.9)	814 (6.3)	1,029 (6.8)	**12,660 (100)**	11,631 (93.2)	712 (4.8)	312 (2)	**12,655 (100)**
Total (N) and Est. Pop	**61,152,182**	**4,073,793**	**7,479,909**	**93.184 (100)**	**65,225,976**	**4,519,697**	**1,800,329**	
Income								
0 99% FPL	12191 (82.5)	855 (5.9)	1,845 (11.6)	**14,891 (100)**	13,046 (88.4)	1,179 (7)	659 (4.6)	**14,884 (100)**
100 199% FPL	14560 (85.1)	984 (6.3)	1,584 (8.7)	**17,128 (100)**	15,544 (91.3)	1,117 (6.2)	463 (2.5)	**17,124 (100)**
200 399% FPL	25064 (86.3)	1,531 (5.5)	2276 (8.1)	**28,871 (100)**	26,595 (91.9)	1,723 (5.9)	551 (2.2)	**28,869 (100)**
400% FPL or higher	30,231 (87.3)	1,823 (5.4)	2,524 (7.3)	**34,578 (100)**	32,054 (92.7)	2,061 (6.1)	461 (1.2)	**34,576 (100)**
Total (N) and Est. Pop	**62,864,564**	**4,211,382**	**6,469,621**	**95,468 (100)**	**67,075,945**	**3,464,785**	**1,839,669**	
Education								
Less than high school education	4,655 (84.4)	334 (6.7)	612 (8.9)	**5,601 (100)**	4,989 (91.1)	373 (5.6)	236 (3.2)	**5,598 (100)**
High school graduate	12,106 (83.4)	884 (6)	1,532 (10.7)	**14,522 (100)**	12,990 (89.4)	1,070 (6.8)	460 (3.8)	**14,520 (100)**
More than high school education	63,723 (86.3)	3.883 (5.4)	5,944 (8.3)	**73,550 (100)**	67,606 (91.7)	4,529 (6.3)	1,405 (2)	**73,540 (100)**
Total (N) and Est. Pop	**61,393,399**	**4,077,327**	**6,360,841**	**93,673 (100)**	**65,470,725**	**4,553,163**	**1,797,669**	
Current Insurance Status								
Insured at time of survey	78,310 (85.4)	4967 (5.7)	7,997 (9)	**91,274 (100)**	83,277 (91.1)	5,917 (6.4)	2,066 (2.5)	**91,260 (100)**
Not insured at time of survey	3,589 (86.7)	223 (7.1)	218 (6.2)	**4,030 (100)**	3,812 (93.8)	151 (3.8)	2,066 (2.3)	**6,029 (100)**
Total (N) and Est. Pop	**62,720,881**	**4,210,240**	**6,460,752**	**95,304 (100)**	**66,931,120**	**4,611,380**	**1,839,364**	

Figure10: CDSS Flowchart.

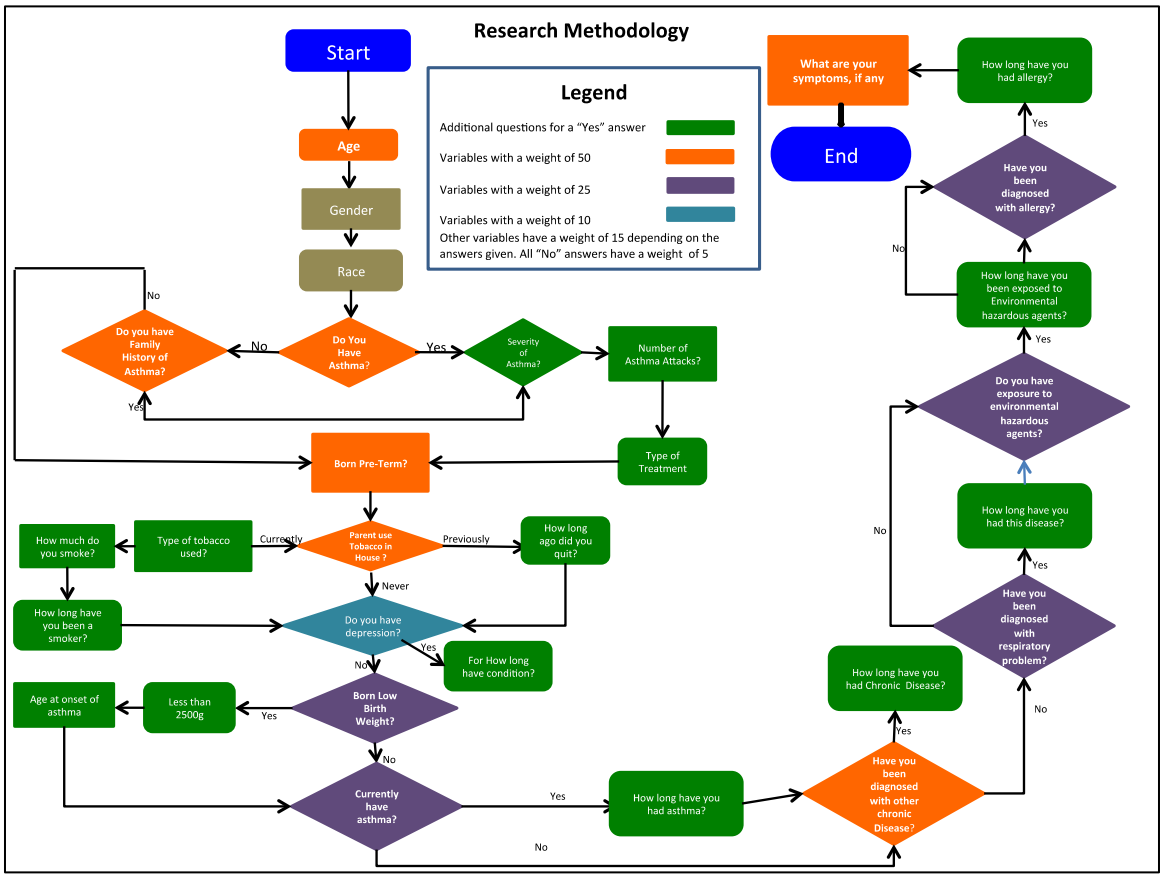


Figure 11: Childhood Asthma Risk Assessment CDSS Implementation using Exsys CORVID Software.

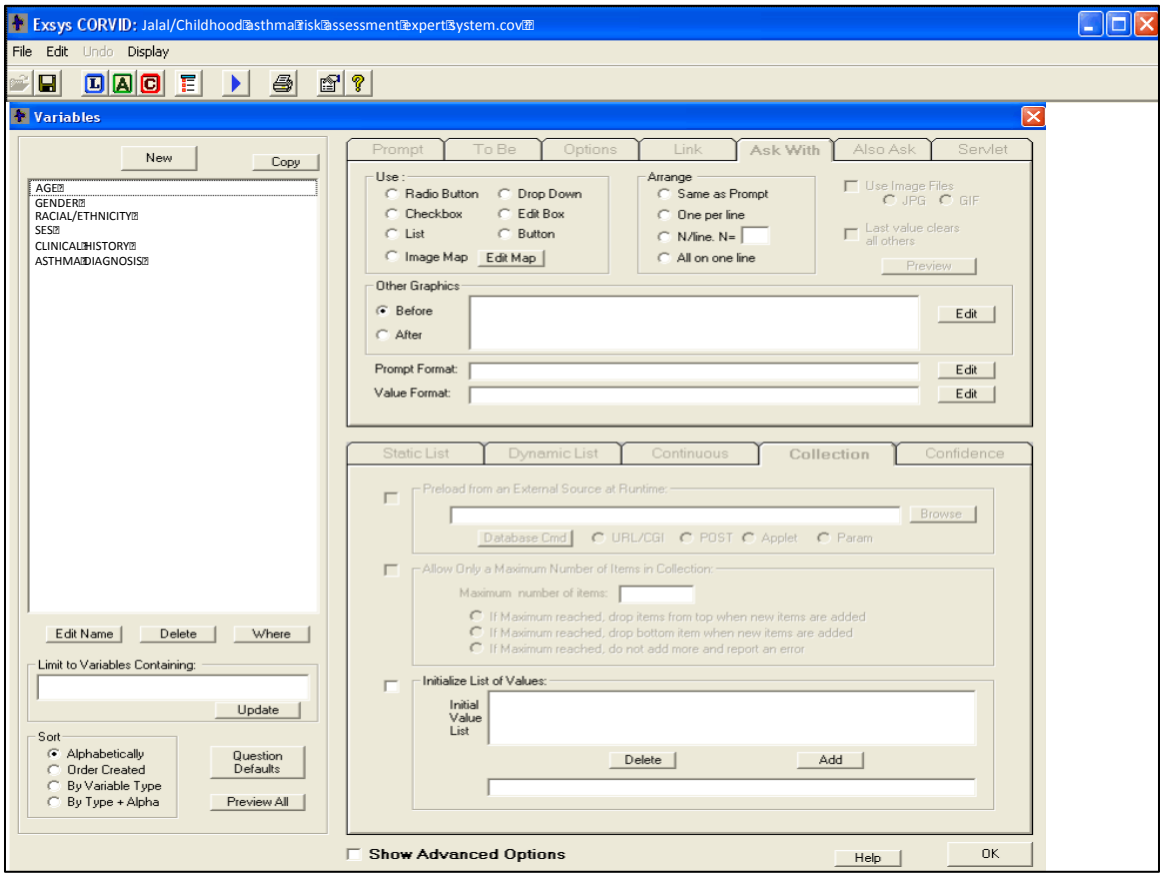


Figure 12: CDSS Variable List.

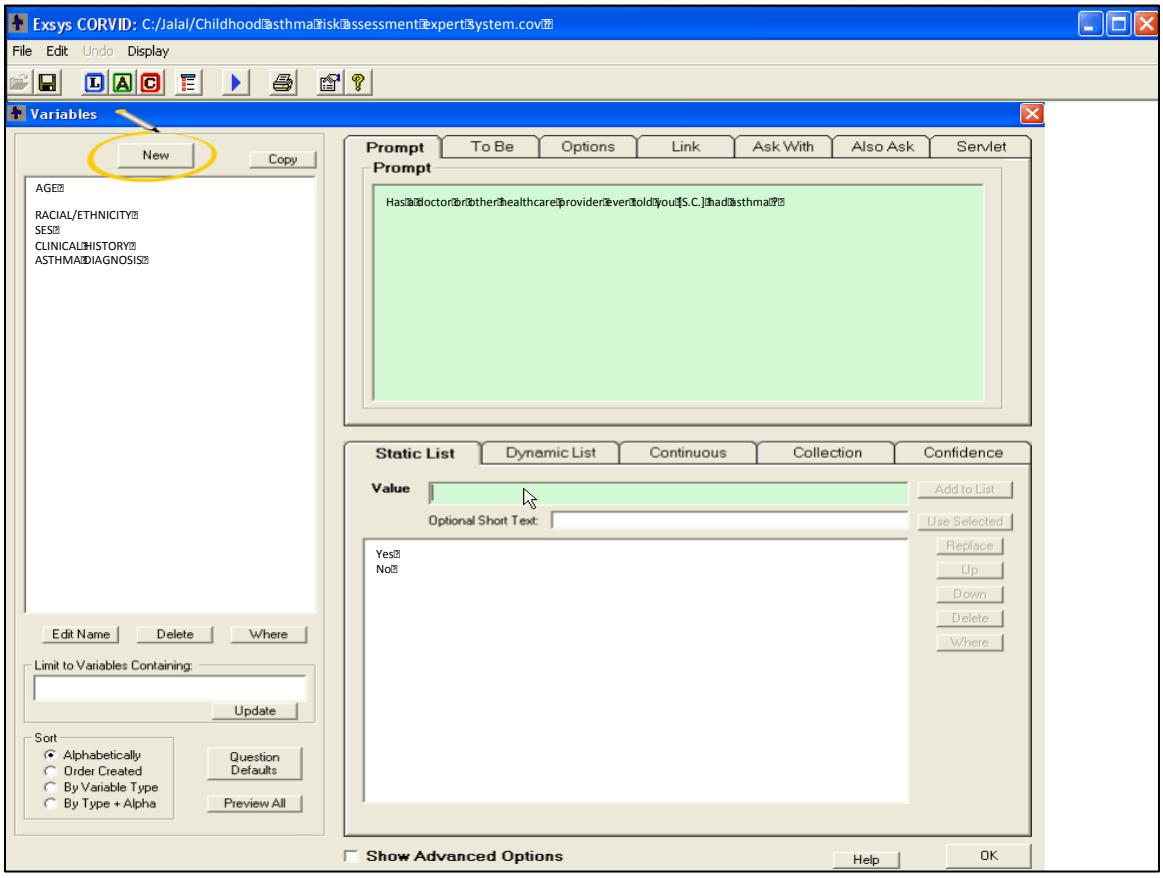


Figure 13: CDSS Logic Block for Childhood Asthma Risk Assessment.

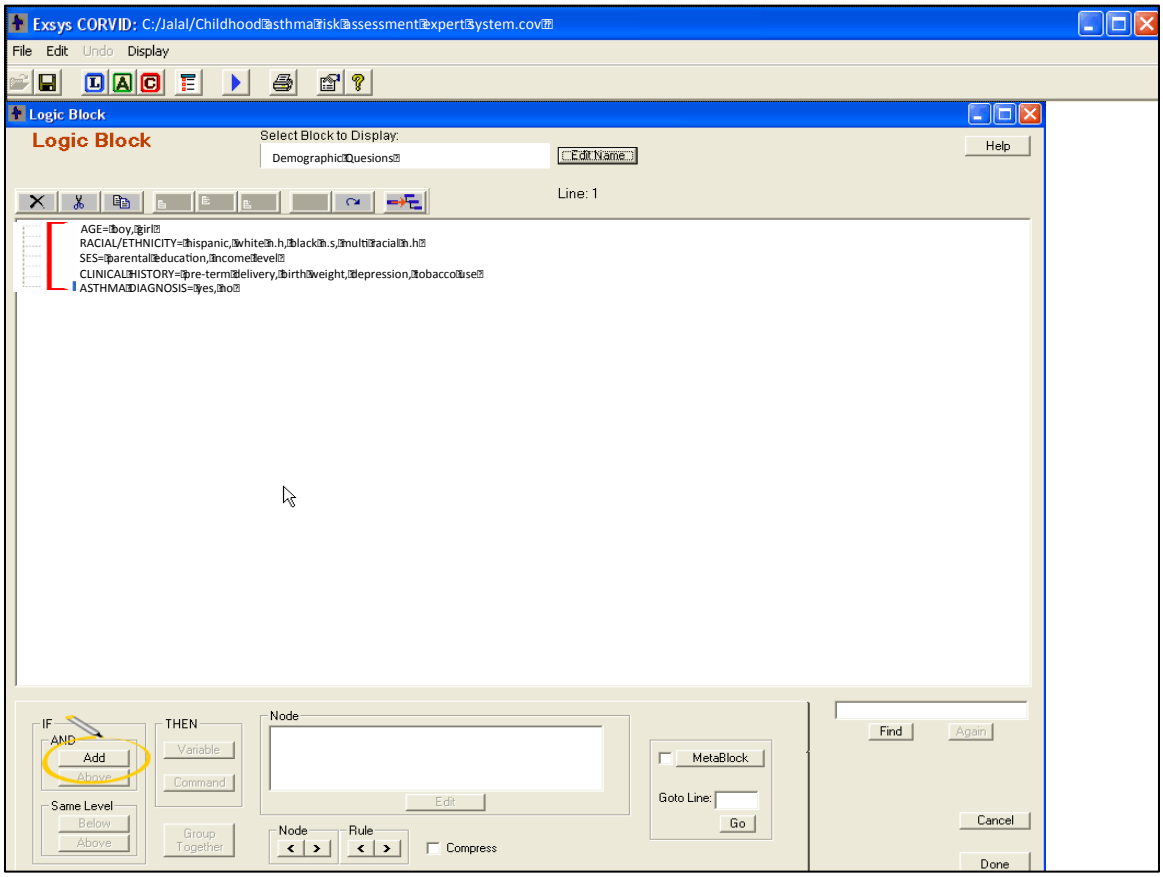


Figure 14: CDSS Logic Tree for Childhood Asthma Risk Assessment.

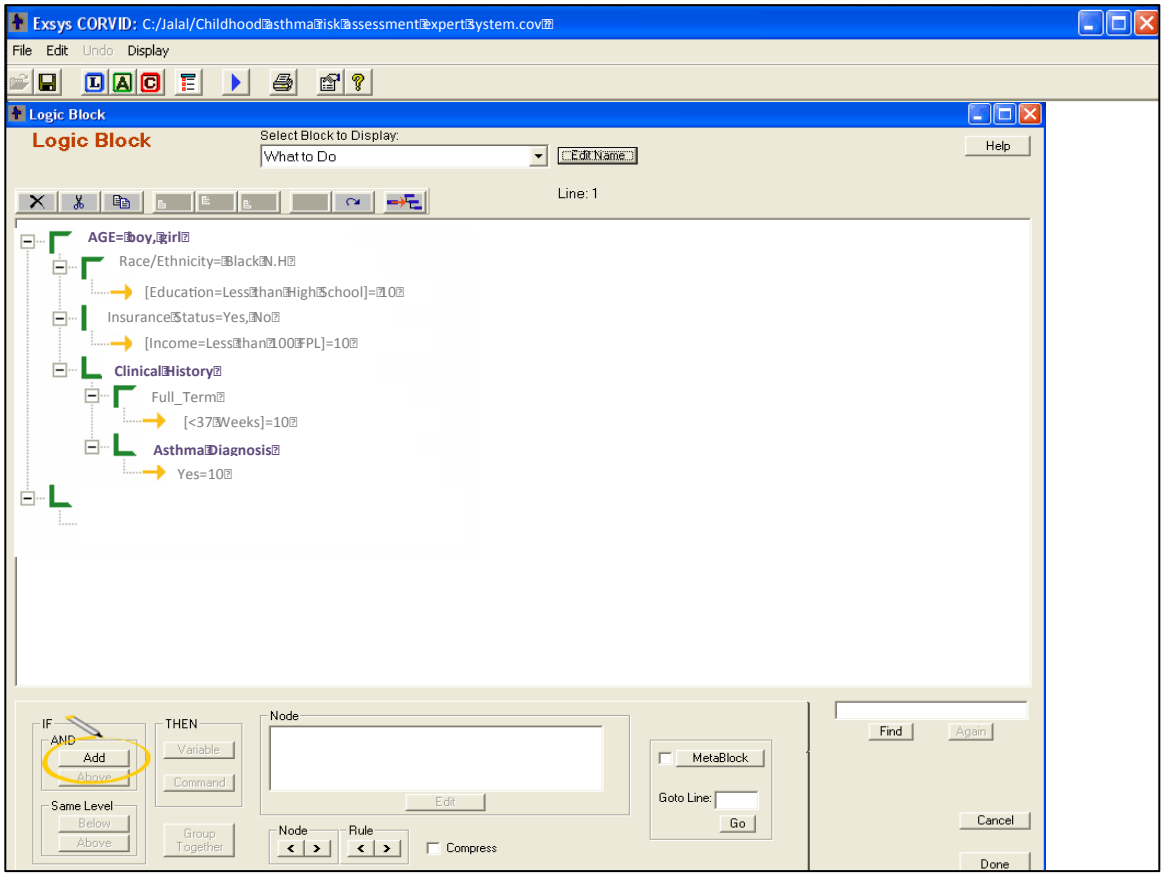
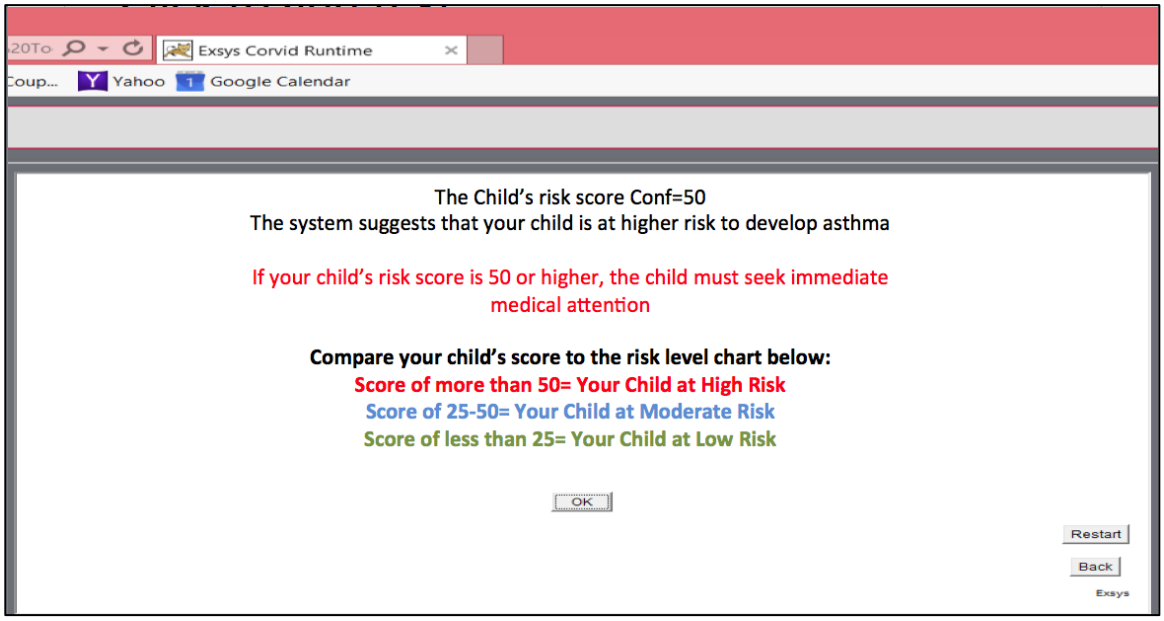


Figure 15: CDSS Result Output Page.



5.0 CHAPTER V DISCUSSIONS

During the initial phase of this dissertation, several essential questions were upraised to frame precise conceptual foundations for the secondary data analysis. In this chapter, an effort will be made to address these essential questions based on the study results. Results from this study will be compared with prior findings in the literature to assess efficiency of methods, principles, concepts, predictors, and limitations used in our secondary data analysis and to examine how possibly they could have impacted our results.

The benchmarks for determining what major factors are associated with childhood asthma are controversial to some extent in that the past. Currently, the literature proposed that several attempts for identifying key factors and determinants, which might impact the development of childhood asthma, were derived from either physical diagnosis by a physician or verbal response from a parent and/or guardian proxy knows the child's health status properly. For instance, Stephen and colleagues have examined the validity of documentation made during pediatric emergency care to decide if a patient-driven health information technology called 'ParentLink' produced better-quality data than documentation concluded by nurses and physicians. The findings of Stephen and colleagues revealed that of a pool of 1410 enrolled parents, 1111/1410 (79%) finalized the criterion standard interview. The interview results revealed that parents' valid reports of allergies to medications were higher than those of nurses (parents 94%, nurses 88%, $p<0.0001$ respectively). Similarly, parents' valid reports of allergies to medications were superior to those of physicians (parent 94%, physicians 83%, $p<0.0001$ respectively). Therefore, wide body of literature accepted parent's documentation as a valid source

about children's health condition.

A variety of hypothesis has been drawn and many others have been evaluated by researchers in regard to the relationship between racial/ethnic differences and the risk of developing childhood asthma. Enormous evidence is findings on access to healthcare, SES, impact of insurance coverage and their associated risk factors useful for better preparation, development and implementation of interventions. As of now, many underlying questions about childhood asthma have cleared. However, the principal evidence of the impact of clinical predictors on childhood asthma still remains controversial. While some researchers have tried to fill this gab by recommending cut-off points to define most associated clinical factors which have a direct impact on childhood asthma and its severity levels, others have considered it from a genetic perspective and responses to environmental factors. Notwithstanding the evident importance, there is still no best way of evaluating children at the highest risk of developing asthma. Although premature delivery and low birth weight risk factors in children are well familiar, inconsistency exist in the between these clinical factors and childhood asthma. The problem gets more complicated when it comes to selecting methods for analyzing population-based public health data. The use of cross-sectional methods to generate parameter estimates for an outcome of interest indirectly proposes that characteristic measures of the general population are the same universally and persist constantly during the selected period of analysis. In practice this is not the case since population-based metrics vary across subgroups. Likewise, setting parameter estimation models based on this hypothesis compromises statistically valid conclusions. To assume parameter

stability across subgroups of a population with no formal test of this assumption is problematic.

As mentioned earlier in the study design section in this dissertation; responses of "Don't know" or "Refused" and those with no response value will not be included in the analysis. In nearly all cases, the unknown values make up less than one percent of the responses to any single question. However, a few questions have higher proportions of unknown values, most notably household income (9.7% nationwide), and race/ethnicity (2.7% nationwide). The 2011-2012 NSCH public data file provided by the National Center for Health Statistics includes the household income variable POVERTY_LEVELR, which is missing values for 8,856 cases. This imputed income data files will be used to estimate household income for children with missing values.

The outcome variables used in this study were a sample child's diagnosis of asthma, defined by the answer to the survey question: Has a doctor or other health care provider ever told you that the selected child [S.C.] had asthma? According to an adult parent/guardian, a sample child's currently have asthma, defined by the answer to the survey question: Does the [S.C.] currently have asthma? And the third outcome was a sample child's severity of asthma, defined by the answer to the survey question: Would you describe [His\Her] asthma as mild, moderate, or severe? The primary explanatory variables were the child's race/ethnicity as reported by an adult parent/guardian, and the family socioeconomic status (SES): parental or guardian education and income based on the FPL threshold in 2011. The following race/ethnicity categories were used in the present study: Hispanic, non-Hispanic white, non-Hispanic black, multi-racial/ other non-

Hispanic; the educational level of the child's parent or guardian in the household (less than a high school; high school graduate and more than high school). In the NSCH dataset, the income to FPL ratio was considered by dividing family income by the poverty threshold (FPL; $22,350 for a family of 4 in 2011). We obtained four family income levels based on the NSCH categorization of family income as 0-99% FPL, 100-199 FPL, 200-399 FPL and 400 FPL or more.

Other covariates were selected based on previous studies and theoretical concerns, included the following: child's gender (male and female); child's age (0-5 years, 6–11 years, 12–17 years). HRSA Region as divided by the Health Resources and Service Administration (HRSA) into 10 regions: Region I: (CT, ME, MA, NH, RI, VT), Region II: (NJ, NY, PR, VI), Region III: (DE, DC, MD, PA, VA, WV), Region IV: (AL, GA, FL, KY, MS, NC, SC, TN), Region V: (IL, IN, MI, MN, OH, WI), Region VI: (AR, LA, NM, OK, TX), Region VII: (IA, MO, NE, KS), Region VIII: (CO, MT, ND, SD, UT, WY), Region IX: (AZ, CA, HI, NV, Guam, American Samoa, the Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Republic of the Marshall Islands, Republic of Palau)and Region X: (AK, ID, OR, WA).

Additional clinically related variables were also considered in analysis as predictors. These variables included child's health premature delivery, birth weight, depression, developmental delay and uses of cigarettes, cigars, or pipe tobacco. The only sub-state geographic information included in the NSCH public use data set is a variable for Metropolitan Statistical Area (MSA) status. The MSA status information is available for the 35 states in which the population is at least 500,000 in both categories (MSA and non-MSA). Zip code data is collected with the NSCH; however, this information is not

released in the public use data set due to confidentiality restrictions. Therefore, this variable was dropped from the study and replaced it with earlier mentioned HRSA Region variable.

5.1 DESCRIPTIVE ANALYSIS

Following the objectives mentioned through the body of this dissertation, this study is aiming to examine whether racial/ethnic differences in childhood asthma have been increasing or decreasing over a specific period of time. Therefore, we focused on the recent 2011-2012 period to evaluate the association of race/ethnicity; household education and income level with the prevalence of childhood asthma.

Between 2011-2012, there are an estimated 14.6 million children with special healthcare needs in United States. This includes an estimated 7 million child with 2 or more current chronic condition. Of the children who currently have at least one chronic condition, approximately half (49.8 percent) were reported to have at least one chronic condition that was moderate or severe in its impact on the child health. However, it is important to note that these are parents' reports of their children's conditions and was not confirmed with medical records.

Asthma was the most often reported condition between 2011/2012, occurring among 1 out of every 11.4 children, followed closely by learning disabilities (1 in 12.5 children aged 3-17 years) (Figure 2). Similarly, asthmatic children are estimated to be around 10.5 million children between 2011-2012 (Table 41) and the incidence of asthma have been noticed at higher rates among older children aged 6-11 and 12-17 years (16.1 and 18.5% respectively) compared with younger age groups aged 0-5 years (8.6%) (Table 41).

Similarly, the incidence of asthma was more prevailed among boys 2% higher compared to girls (Table 41). Black non-Hispanic children are about 3 times more than white non-Hispanic children to have moderate or severe asthma (Table 41). Similarly, children who live in household below the federal poverty level (0-99 FPL) have higher incidence of moderate or serve asthma 4.6% compared to 2.5% (100-199 FPL), 2.2% (200-399 FPL) and 2.2% (400 FPL or higher) (Table 41).

In regard to parental/guardian level of education, the majority were households with more than high school education (46,688; 47.09%), high school graduate (30,691; 32.24%) and less than high school (12,837; 20.66%) respectively.

Furthermore, children with special healthcare needs such as asthma control and management are 1.3% more than Non-CSHCN whom health insurance is not adequate and are 1.3% more getting problems receiving the needed referrals to a specialist (Figure 9).

5.2 ANSWERS OF RESEARCH QUESTIONS:

Q1: What are the different major demographic factors associated with the prevalence and persistent racial/ethnic inequalities among all US children with asthma?

At the body of this dissertation we hypothesized a presumptive statement as ***"Being from low SES and race/ethnicity group (Hispanic, Non-Hispanic Black, multi-racial/other Non-Hispanic) will increase the asthma and severe asthma rates among minority children living in the US."***

Our findings were consistent with previous studies as the present study indicates that racial/ethnic inequalities in asthma were determined with higher rates among groups who live below 100 FPL. That is, non-Hispanic black children living at <100 FPL were at significantly higher risk for asthma compared with non-Hispanic white children (reference group) who lives in households with similar incomes which is consistent with prior studies. At all other levels of income, no racial/ ethnic differences in asthma prevalence were witnessed. For instance, Hispanic children did not experience any statistically significant association with asthma relative to non-Hispanic white children at any income level >100 FPL. Handling income as a confounder without considering the racial/ethnic differences could possibly lead to a confounding results. However, whenever race/ethnicity differences become meaningless after adjustment for household income, this finding must also be interpreted carefully. For instance, according to group of authors, race/ethnicity could be associated to childhood asthma in numerous potential ways.[65-67] states that racial/ethnic differences could seemingly result from confounding;

other issues associated with both race/ethnicity and asthma, like SES or social class, may justify why black children seem to suffer from higher rates of asthma compared to white children.[65,68-70] on the other hand, argue that the relationship between race/ethnicity and prevalence of childhood asthma could be described by racism, which is associated with restricted employment opportunities, consequently lead to lower income levels. Further, lower income among Black families' compared to the US population could result in an accumulation of black children in poor quality housing with higher chances of exposure to asthma-related triggers, such as cockroach antigen, therefore strengthen the pivotal pathway between race/ethnicity and higher asthma prevalence. A third potential way to explain the association between race/ethnicity and the high prevalence of childhood asthma could be due to distinction of genetic predisposition to asthma, possibly interacting with adverse environmental exposures. While there is developing consideration to the genetics features of asthma, our finding of racial differences only among children who live in poor neighbors is inconstant with disparity in genetic susceptibility as the primary cause of black vs. white inequalities in asthma prevalence.

Q2: How is SES (parent's income and education) associated with the prevalence of asthma among all children living in US?

The initial X^2 test of association illustrated no significance association between the prevalence of asthma and parental level of ($p= 0.018$). Contrary to the asthma prevalence, X^2 test determined a statistical significance, at boarder line, with asthma level of severity ($p= 0.007$). These findings are consistent with prior study by [58]; they concluded that low parental education of children with asthma is associated with extra severe asthma in children and consequently increased used of rescue medication.

However, this association didn't stand valid after including the parental education factor in a logistic regression model. The logistic regression analysis revealed that neither category of the educational level nor the overall educational level of parents and/or guardian is significantly associated with childhood asthma. For instance, the probability of parent with less than high school is (*p=0.2275)* and (*p=0.5323)* for parent with high school graduate level of education compared more than high school level of education level (reference group). This inconsistency in our findings with prior studies might be explained because previous studies didn't consider the association at each category of parental education which have been seen in our study which revealed that parental education has strong association with asthma level of severity in general but when the association has been compared with different levels of parental education (less than high school, high school graduate or more than high school the association disappeared. Therefore, findings in the present study confirms that studying the overall relationship between the prevalence of childhood asthma with categorical factors and/or predictors has to be one category compared to the next category within the same factor to avoid any confounding results due to missing the significant impact of certain categories more than other ones within the same factor.

In addition, the significance of association between asthma/asthma severity prevalence and income to FPL was strong at both levels with statistical probability of (*p= <.0001*). In contrast, when logistic regression model was developed to evaluate the magnitude and linearity of association with asthma prevalence vary significantly based on the income-to-FPL, for instance, asthma was highly prevailed among poor children who live in

households below 100-199 FPL (*p=<.0001; .0008 respectively*) compared with higher income levels (200-399 FPL; *p=.018)* and >400 FPL (reference group). The prevalence of asthma severity was strongly associated as the general prevalence of childhood asthma with income level.

Aside from the described potential factors mentioned to answer the 1st question, income level has been examined very well in the literature as a confounder to the association between race/ethnicity and asthma, and yielding contradictory results. For instance, group of studies have indicated that adjusting for income and other socioeconomic elements, such as urban residence and single-parent status, decreases or removes the higher risk of asthma associated with black "race" in unadjusted analyses. [65,68-70] Other body of the literature, however, have discovered an increased prevalence of childhood asthma among black children compared to white children at all levels of income, even after controlling for such factors. [66, 67, 71-73]

Moreover, some studies in the literature have described children just as poor or non-poor but did not assess the complexity and background of poverty which could strongly miss significant differences in the poverty incidents among black and white children, and as a result dimming potential causal triggers for racial/ethnic differences in asthma. For instance, poor black children are more likely to live in deep poverty than poor white children. Similarly, poor black children are 40% more likely than poor white children to live in families with annual incomes less than half of the federal poverty level (FPL) .[76] In addition, poor black children are much more likely than poor white children to live in

neighborhoods of condensed poverty, where more than 40% of individuals in the neighborhood are poor.[77]

Q3: What is the impact of clinically relevant factors (predictors) on the prevalence of asthma among all children living in US?

Group of researchers have considered that racial differences in airway hyper-responsiveness and allergen hypersensitivity may be attributable to differences in clinical and genetic predisposition.[120] Nevertheless, such clinical differences might be recognized to non-genetic factors such as disparity exposures to allergens.[150] However, gene/environment interaction might justify our findings if exposures to asthma triggers were raised only among very poor black children and not among black children living in higher-income families. The collaboration of black group and income discovered in this study recommends that efforts to establish genetic influences on racial/ethnic differences in asthma must focus on the environmental risks that disadvantaged minority children face. This study also proposes that genetic cause/effect relationship with racial/ethnic differences must not be related until there thorough genetic data supporting such claim are available in hand. [147,148]

The clinically relevant variables in this study (premature delivery, birth weight, depression and developmental delay) were selected as predictors based on prior recommendations in the literature as strongly impacting the prevalence of childhood asthma and its level of severity. However, the literature didn't confirm that these

predictors cannot be interpreted as cause and effect interaction with childhood asthma due to lack of enough evidence as of this date. Therefore, we decided to utilize them in our study to fill this gab in the literature by adding new findings to validate the nature of association between these clinically relevant predictors and childhood asthma.

Our findings indicate that prematurity and birth weight are associated with the risk of persistent asthma later in childhood and that these birth characteristics mediate racial differences in asthma prevalence among U.S children. Consistent with previous reports, [95-99] our findings indicate that children born with low birth weight (less than 2500g) is independently associated with the increased prevalence of childhood asthma.

For instance, the initial X^2 test of association between premature delivery and childhood asthma prevalence revealed strong association with probability of *p= <.0001 (*TABLE 25*)*. On the other hand, the association was not significant with asthma level of severity (*p= 0.0814*) (TABLE 26). Similarly, birth weight was also significantly associated with childhood asthma as can be seen in TABLE 27 (*p= <.0001*). Therefore, we focused on racial difference and adding some clinically relevant variables that could justify the observed black vs. white difference in asthma rates among U.S population. Premature delivery, birth weight, depression, developmental delay and presence of someone uses cigarettes, cigars, or pipe tobacco were selected based on their relevance to the prevalence of childhood asthma as being implied from the literature review. The clinically relevant variables were added to a multiple logistics model with adjusting for potential confounders. The reason for applying such adjustment is to avoid bias in result.

For instance, the likelihood association between premature delivery (37 weeks or less) and the prevalence of childhood asthma was statistically significant with OR 0.654 and 95%CI (0.568-0.752) with adjusting the full-term delivery as reference. Similarly, the odds of low birth weight revealed strong likelihood of increasing the prevalence of childhood asthma if the baby born with less than 2500g (OR 0.928; 95%CI 0.792-1.087). In the same logistic model depression showed strong likelihood of impacting the prevalence of childhood asthma with odds ratios (OR 0.490; 95%CI 0.378-0.635). Similarly, the odds of developmental delay reflected high likelihood of increasing the chance of developing asthma (OR 0.704; 95%CI 0.580-0.855). Finally, the presence of someone using tobacco in the house has strong likelihood of developing asthma among US children aged 0-17 years between 2011/2012. Although children born prematurely or very prematurely were found to have increased risk of persistent asthma later in childhood, these risks were comparable among white and black children within each group. Similar to our results, most prior studies found preterm birth to be a risk factor for childhood asthma [98-100,103] although not all reports have confirmed this association.[115] The evidence for low birth weight as a risk factor for developing childhood asthma is less consistent; although there is supporting evidence from several studies,[97-99,115] others have not found a positive association between low birth weight and childhood asthma.[101-103,116] The conflicting results in these studies may be due to the overlap between low birth weight and preterm birth and the wide range of birth weights observed within gestational age classes.

Depression surprisingly showed strong association with childhood asthma and its severity level prevalence with probability of p= <.0001 as TABLE 30 confirms. The developmental delay was also associated with strong significance with both childhood asthma prevalence as well as its level of severity with probability of p= <.0001. The significance of association was strong enough between childhood asthma and household using tobacco in the house with probability *p=<.0001*. However, the severity level of childhood asthma was not strongly associated with the use of tobacco by a household (*p=0.0012*).

Q4: Among children living in US, what is the impact of insurance status on the prevalence of asthma among all children living in US?

Contrary to prior studies, [86-89] our findings confirm that the insurance status at the time of survey was not associated with neither the prevalence of childhood asthma nor the level of asthma severity (*p=0.3144*; *p=0.1678* respectively). The explanation for this inconsistency between our findings and previous studies might be due to the missing reported asthma exacerbations for those who doesn't have consistent insurance coverage and mainly count on the ED rooms in case of any asthma attack.

5.3 LIMITATIONS OF THE STUDY

This study has several potential limitations. First, Complex design of the NSCH must be accounted for in order to infer valid conclusions. Therefore, for the purpose of generating a sample for determining national estimates, the NSCH recommends the use of the weighted variable to draw proper estimations. Second, the weighted estimates used in the NSCH do not generalize to the population of parents, mothers, or pediatric health care providers. Analysts are advised to avoid statement about the percent of parents.

Third, the condition-specific information in this NSCH is based on parent report, not clinical diagnosis and therefore results might be underestimated. As mentioned previously, frequencies are underestimates due to item non-responses and unknowns. Although non-responses were coded as missing values, these were excluded from the tables. The recommended weighted factors for the survey sample and examination were applied to the analytical models in this analysis. However, their application did not yield significant accuracies to the generated estimates. Therefore, interpretation of estimates provided by this study must be made only after a careful consideration of the methods used to make the estimations. Lastly, this study is a U.S. population based; therefore, generalizing its results to other countries must be interpreted carefully.

6.0 CHAPTER VI SUMMERY AND CONCLUSIONS

6.1 SUMMARY

The purpose of this dissertation was to estimate current national prevalence of childhood asthma among minority groups (Black Non-Hispanic, Hispanic and Multi-racial/Other Non-Hispanic) and adolescents in the United States using the most recent data from a nationally representative population-based survey to inform decisions around strategizing approaches to improve population health. Specific objectives were to: examine whether racial/ethnic differences in childhood asthma have been increasing or decreasing over a specific period of time; determine the impact of clinical predictors on childhood asthma, and the role race/ethnicity, parental/guardian education and income level through secondary analysis study on children aged 0-17 years in the United States between 2011/2012.

To accomplish the study goals and objectives, we employed the recent National Survey of Child's Health; selecting the variables of interest; how variables were to be measured; and what type of statistical relationships must exist between the variables. The NSCH survey is representative of all non-institutionalized children aged 0 to 17 years in the US and in each state. The latest round of the survey, conducted in 2011-2012, provides a snapshot of children's physical, mental, and developmental health status; access to health care; activities at school, outside of school, and at home; and their safety and security in their neighborhoods and at school. The NSCH, supported and developed by the U.S. Department of Health and Human Services, Health Resources and Services Administration's Maternal and Child Health Bureau and conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention, is the only

nationally-representative survey that considers children's health and well-being within the contexts of the family and community. The 2011 NSCH surveyed the parents of a total of 95,677 children, or approximately 2,000 per State. The study sample of our data analyses consisted of a total of 95,677 for the sample child's diagnosis of asthma, 13,422 for sample children currently have asthma and 8,214 for sample child's severity of asthma. The present analysis provides the most current estimates of childhood asthma prevalence in US children aged 0-17 years. Similarly, asthmatic children are estimated to be around 10.5 million children between 2011-2012.

6.2 KEY FINDINGS

- Preliminary analysis: more than 10.5 million children had asthma between 2011/2012.
- Compared to 2003, 2007, the prevalence of asthma in 2011/2012 has increased from 12.4% and 13.5% to 14.5% among children living in U.S.
- Results showed that asthma was less prevailed among young children aged 0-5 years compared to 6-11 and 12-17 years (8.6%, 16.1% and 18.5% respectively.
- Similarly, results also revealed that boys reported asthma more than girls (15.9% and 13.1 respectively), which means 1.2 times more likely to have asthma.
- It was found that premature delivered children (<37 weeks) at higher risk of asthma compared to normal term children (20.8% vs. 13.8%).
- Results also founds that severe and moderate asthma are more prevailed among children with household below 100% FPL.
- The preliminary results indicated that there is a significant association between premature, low birth weight, depression and developmental delay factors and asthma of children with statistically significant probability of less than .05 ($p<.0001$).

- These data confirm findings of other studies that identify a strong association between low birth weight and asthma. For this 2011/2012 NSCH, an estimated 1,287,858 (19.3% vs. 14.1%) excess asthma cases were attributable to birth weight less than 2500g.

6.3 CONCLUSIONS

Racial/ethnic findings in this study illustrated that non-Hispanic black children were at higher significant risk of asthma compared non-Hispanic white peers in unadjusted analyses, but after stratifying the analyses by parental/guardian income level, this risk was existent only among children living in families with incomes <100 FPL. This result recommends that researchers examining racial/ethnic disparities in asthma should be careful about simply adjusting for income and should focus on the different arrays of social and environmental exposures that poor black and white children may face. Though genetic predisposition to asthma may eventually be discovered to differ across racial/ethnic groups, the result that racial/ethnic differences are concentrated among the poor recommends that the illustration of any assumed genetic risk would still vary greatly on forms of social and environmental exposures. Persistent stress on race/ethnicity as a social paradigm—with social implications that have inferences for health, relatively than as a demonstration for genetic differences—will focus consideration on the role of individual and appropriate risks for asthma and will benefit to target efficient clinical and community interventions to reduce racial/ethnic disparities.[67, 70, 42,148,149]

6.4 RECOMMENDATIONS

1- The current results confirm that clinically relevant predictors in children with associated prevalence of childhood asthma notably among older ages 11-17 years. A comprehensive asthma intervention programs can positively improve population health.

Future studies should focus on assessing the impact of intervention programs.

2- Majority of the studies that evaluated childhood asthma is cross-sectional and case studies, therefore, future studies may want to consider longitudinal designs.

3- Appropriate research designs and methods for evaluating behavioral and community interventions, and to examine how differential access to care contributes to morbidity is needed to better understanding of childhood asthma differences in U.S.

4- Research aimed at understanding risk factors for disparities in asthma health outcomes, to describe key barriers to improving asthma outcomes, and to establish priorities for future research is needed.

5- Future research to understand risk factors for asthma disparities and their mechanisms (e.g., gene-by-environment interactions and the role of clinical factors (e.g. premature, birth weight, depression and developmental delay) is needed to fill the gap in asthma prevention.

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8.0 APPENDIXES

TABLE 42 Description of Messiness by the National Center for Health Statistics.

Code	Category	Description
.N	Not in universe	Variable is missing because the child was not eligible for these questions (for example, children ages 0–5 were not eligible for section 7 and children ages 6–17 were not eligible for section 6).
.L	Legitimate skip	Variable is missing due to valid questionnaire paths based on a previous answer to a root question.
.P	Partially completed interview	Variable is missing because the respondent ended the interview after completing Sections 6 or 7 (depending on the age of the child) but before completing the full interview.
.M	Missing in error	May indicate that the variable is missing due to interviewer or system errors. In addition, all missing values for derived variables (i.e., variables whose response was not directly provided by the respondent) receive a ".M" code regardless of the reason for the missing data.
.A	Added question	Variable is missing because this question was added after the start of data collection and the interview was conducted before the variable was added.

TABLE 43 VARIABLES CATEGORY

Predisposing	Enabling	Need	Outcome
Age	Parental Education	Asthma Diagnosis or Report by Physician or Parents	Asthma
Gender	Income Level		Severe Asthma
Race/Ethnicity	Insurance Coverage		
	Region		

FIGURE 16: Aday & Anderson Framework for the Study of Access.

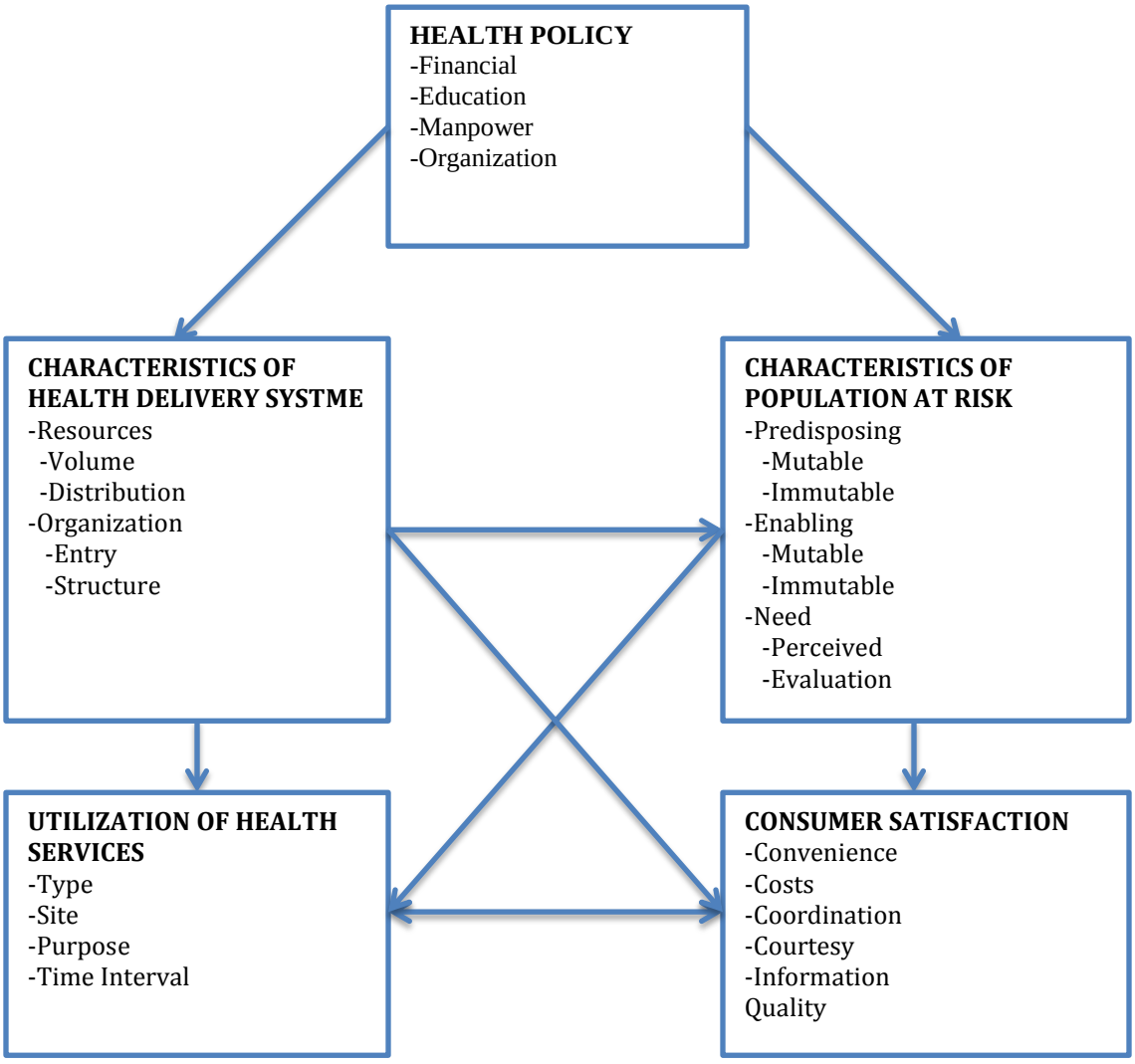

FIGURE 17 Prevalence of Asthma among Minority Population Children Aged 0-17 Years in the United States between 2011/2012