

**THE RELATIONSHIP BETWEEN BMI AND ASTHMA IS A SIGNIFICANT  
HEALTH CONCERN AMONG PRE-ADOLESCENT AND ADOLESCENT  
CHILDREN**

A Thesis

by

JENNIFER LEAH FURMAN

Submitted to the Office of Graduate Studies of  
Texas A&M University  
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

August 2007

Major Subject: Nutrition

**THE RELATIONSHIP BETWEEN BMI AND ASTHMA IS A SIGNIFICANT  
HEALTH CONCERN AMONG PRE-ADOLESCENT AND ADOLESCENT  
CHILDREN**

A Thesis

by

JENNIFER LEAH FURMAN

Submitted to the Office of Graduate Studies of  
Texas A&M University  
in partial fulfillment of the requirements for the degree of  
MASTER OF SCIENCE

Approved by:

Chair of Committee,	W. Alex McIntosh
Committee Members,	Jenna Anding
	Joseph R. Sharkey
Chair, Intercollegiate Faculty of Nutrition,	Nancy D. Turner

August 2007

Major Subject: Nutrition

## ABSTRACT

The Relationship Between BMI and Asthma Is a Significant Health Concern Among  
Pre-Adolescent and Adolescent Children. (August 2007)

Jennifer Leah Furman, B.A., Texas A&M University

Chair of Advisory Committee: Dr. W. Alex McIntosh

In the U.S., the prevalence rates of obesity and asthma have increased over the past 20 years and led to speculation that a correlation exists between them. Most of the research that has been conducted on the association between obesity and asthma has been done so in adults. Little data are available on this association in children. Obesity and asthma are associated with significant morbidity and mortality risk. Concern that the prevalence of overweight and asthma in children will persist and/or worsen in adulthood has prompted the current study. This study analyzed the association between asthma and allergy prevalence and measures of overweight, dietary intake, supplement use, ethnicity, child's guardian, mother's education level, and whether the mother smokes among children aged 9 to 11 years and 13 to 15 years. This study also analyzed the association between the use of prescription asthma and allergy medication and asthma and allergy diagnosis, measures of overweight, dietary intake, supplement use, ethnicity, guardian, mother's education level, and whether the mother smokes.

The findings of this study revealed that non-White children had greater odds of asthma diagnosis and were more likely to use asthma medications. A positive

correlation was found between asthma diagnosis and allergy diagnosis and between asthma diagnosis and the use of allergy medication. Positive correlations were found between asthma diagnosis and BMI category, subscapular skinfold thickness, and waist circumference among children 9 to 11 years old. Age and weight were found to be inversely correlated with asthma diagnosis among children 13 to 15 years old. BMI category was found to be positively associated with asthma diagnosis among males, but not among females. Asthma diagnosis in females was not found to be significantly correlated with any of the variables. Dietary intake, multi-vitamin/mineral and omega-3 fatty acid supplementation, guardian, waist/hip ratio, mother's smoking habit, and mother's education level were not found to be significantly correlated with asthma or allergy diagnosis or with asthma or allergy medication use. A significance value of  $p < 0.05$  was used for all analyses.

## ACKNOWLEDGMENTS

I would like to begin by thanking my committee chair, Dr. W. Alex McIntosh, and my committee members, Dr. Jenna Anding and Dr. Joseph R. Sharkey, for their incredible support throughout this thesis project. They are each remarkable mentors and their continued guidance has been greatly appreciated.

I would like to thank my friends and family who have encouraged me along the way, especially my mom, Irene Gutierrez. She has been so supportive of me in everything, but I am most thankful for the self-confidence and perseverance which she has instilled in me.

Finally, I would like to thank my husband, Barry, who has been an amazing role model and touchstone for me. His incredible love, support, and encouragement have been tremendous even from across such great distances.

## TABLE OF CONTENTS

	Page
ABSTRACT.....	iii
ACKNOWLEDGMENTS.....	v
TABLE OF CONTENTS.....	vi
LIST OF TABLES.....	vii
CHAPTER	
I INTRODUCTION.....	1
II LITERATURE REVIEW.....	8
Overweight and Obesity.....	8
Asthma.....	13
Asthma Among the Overweight and Obese.....	31
Summary.....	41
III METHODS.....	43
Data.....	43
Variables.....	45
Analysis.....	52
IV RESULTS.....	53
Frequency Distributions.....	53
Binary Regression Analysis.....	64
V DISCUSSION.....	69
VI SUMMARY.....	78
Limitations.....	79
Future Research.....	80
LITERATURE CITED.....	81
VITA.....	90

## LIST OF TABLES

TABLE		Page
1	Frequency (%) distributions for demographic variables.....	54
2	Sample size, mean, and standard error of height, weight, and BMI.....	56
3	Frequency (%) distributions for BMI category and waist and hip circumference.....	57
4	Frequency (%) distributions for skinfold thickness measurements.....	59
5	Frequency (%) distributions of allergy and asthma diagnoses and medication use.....	60
6	Frequency (%) distributions of dietary intake and supplement use.....	62
7	Results of binary regression performed on the entire sample.....	64
8	Results of binary regression performed on pre-adolescents.....	66
9	Results of binary regression performed on adolescents.....	66
10	Results of binary regression performed on males.....	68
11	Results of binary regression performed on females.....	68

## CHAPTER I

### INTRODUCTION

The world is plagued with a plethora of chronic diseases. Over the past 20 years, the U.S. has seen particularly rapid and simultaneous rises in the prevalence rates of both asthma (1-3) and obesity (4-8). Given the similar increases in both of these chronic diseases over the recent years, there has been much speculation that there may be a relationship between the two. Most of the research that has been conducted on this subject has been done so in populations of adults, but children have also been affected by the increases in asthma and obesity. To date, limited research is available on the effects of obesity and comorbid asthma in children and adolescents.

The most recent estimates report that almost one-third of Americans are obese and more than half are either overweight or obese (4). There are many possible explanations for the recent rise in overweight and obesity. The rise may be attributed to a combination of internal factors, including genetic and physiological, and external factors, including environmental, behavioral, cultural, and social. While there is research to support that all of these factors may be involved in the etiology of overweight and obesity, a study by Fiore et al. points to external factors as the major contributors (9). Parental obesity may be the most important risk factor for child obesity, which likely reflects an interaction between genetic, lifestyle, and behavioral factors (9, 10). Obesity gives rise to many comorbidities in children and adults,

---

This thesis follows the style of The Journal of Nutrition.



including type 2 diabetes, hypertension, dyslipidemia, and respiratory problems among many more (4, 9, 11-13, 5). The increasing burden of obesity and its comorbidities results in increased health care utilization (12) and ultimately increased financial burden (14). Most recent reports estimate the cost of obesity in the U.S. to be \$99 billion per year, more than half of which is attributable to hospital visits, emergency department visits, and medication usage (12, 15, 16).

In addition to overweight and obesity, asthma prevalence has also been on the rise in both the adult and child populations. Between 1980 and 1994, the prevalence of asthma increased by over 100% among Americans (1). Asthma prevalence more than doubled among children less than 18 years of age during this time period (2). Current estimates report that 6.5 million children currently suffer from asthma and more than two-thirds of these children experience at least one asthma attack per year (2). It has been reported that asthma exacerbations in the previous 12 months have contributed to a significant number of school absences, limitations in physical activity, and impaired functioning due to sleep disturbances (17, 18), which may then contribute to physical and mental dysfunction.

Asthma is defined as a chronic inflammatory disease of the respiratory tract. It is characterized by coughing, wheezing, breathlessness, airway obstruction, chest tightness, and bronchial hyperresponsiveness (19, 20). Like obesity, there are many possible factors involved in the development of asthma, including a combination of genetic, lifestyle, environmental, and socioeconomic factors (3, 21-24). Family history of asthma or allergy as well as a personal history of allergy seems to be the most important

predictors of asthma (10, 11, 25, 26). As many as 85% of school-aged children have been reported to have asthma combined with allergies to airborne triggers (27). Immunoglobulin E (IgE) and histamine are involved in the pathogenesis of allergic-asthma (28) and are responsible for acute asthma exacerbations and inflammation (29). Children with a combination of asthma and rhinitis use more health care resources than children with asthma alone (30). Allergic asthma usually establishes in childhood, increases throughout childhood and adolescence, and peaks in the second decade of life (29).

Diet and obesity are gaining interest as risk factors for the development of asthma (3, 6, 10, 31). Many studies report that asthma severity, control, and prevalence may be influenced by increasing body mass index (BMI) (32, 33). Some researchers suggest that excess body fat may have different effects on asthma in boys versus girls, and children versus adults (7, 21, 31, 33, 34). This subject will be further explored in the following chapter. Dietary intake, particularly inadequate intake of antioxidants and essential fatty acids, may be involved in the pathophysiology of asthma. Decreased intakes of fresh foods such as fruits, vegetables, and fish may have contributed to the decline in the intake of these micronutrients, mainly vitamin C and omega-3 fatty acids. Survey results regarding nutrient intake can be misleading and may not account for variations among individuals in a group. For example, research conducted at the Beltsville (Maryland) Human Nutrition Research Center, United States Department of Agriculture (USDA) reported that the average intake of vitamin C among children under the age of 18 years exceeds the dietary reference intakes (DRI) for vitamin C; however,

this is actually only being achieved by 73% of the children (35). Vitamin C and omega-3 fatty acids suppress inflammation associated with asthma by targeting prostaglandins and leukotrienes, respectively (10, 36), both of which will be described in further detail in the following chapter.

Asthma is the leading cause of illness and disability in children (5). Asthma can adversely affect an individual's ability to perform daily functions as well as negatively impact psychosocial health. Children miss more days from school due to asthma than any other chronic childhood disease (17, 18). Reports have estimated that as many as 60% of children have experienced school absences due to asthma, with an average of 10 missed school days per year (18). Asthma is also the leading cause of hospitalizations among children (22). The risk of hospitalization is compounded by the presence of asthma comorbidities, especially rhinitis. Children with asthma and comorbid allergic rhinitis utilize more health care resources than children with asthma alone (30). Risk for hospitalization increases with increasing asthma severity and medication use, and contributes to increasing financial burden and asthma mortality risk. Most recent reports estimate the annual cost of asthma in the U.S. to be \$11 billion (36), with hospitalizations and medication use accounting for the majority of this (18).

Acute asthma poses a considerable risk for death, despite advancements in awareness and understanding of the disease. Disease severity, level of disease control, and the presence of psychosocial disturbances may increase the risk for asthma mortality (37). The risk for asthma death increases with age, with the greatest rates among adolescents (2). Chronic lower respiratory disease is one of the ten leading causes of

death among adults and children according to the most recent reports by the National Center for Health Statistics (5).

The National Heart, Lung, and Blood Institute (NHLBI), the Global Initiative for Asthma (GINA), and the National Asthma Education and Prevention Program (NAEPP) all provide guidelines that help to identify asthma, determine asthma severity, and establish appropriate treatment. The Asthma Control Test (ACT) and the Asthma Therapy Assessment Questionnaire (ATAQ) are valuable tools that may be used to determine the level of asthma control and are helpful in determining if therapeutic goals are being achieved.

Children with asthma that is complicated by excess body weight may require special care and treatment from health care providers. Obese asthmatics suffer from a greater number of symptoms, greater frequency of symptoms, greater medication use, and greater limitation in daily activity, which is often indicative of greater asthma severity (14). Individually, asthma and obesity contribute to a decline in the quality of life of those afflicted with the diseases. The decline in quality is much greater in obese individuals with asthma compared to those with asthma or obesity alone (38).

Like asthma, obesity is an inflammatory disease. Adipose tissue releases substances, such as leptin, that stimulate the production and release of inflammatory factors. Some of these inflammatory factors include interleukin (IL)-6, tumor necrosis factor (TNF)-alpha, C-reactive protein (CRP), and chemokines (6, 33, 39). Many researchers suspect that the increase in obesity prevalence has contributed to the increase in asthma prevalence by way of an inflammatory mechanism. The release of

inflammatory factors may adversely affect the respiratory system (19, 40), which may then lead to the development of asthma. More information regarding these factors and their mechanisms will follow. Other researchers believe that obesity gives rise to asthma via a mechanical mechanism rather than an inflammatory mechanism. Obesity, especially central obesity, exerts mechanical stress on the airways, which leads to a sequence of events that result in asthma. This is also explored in the following chapter.

Bronchial hyperresponsiveness (BHR), a common symptom of exercise-induced asthma is more pronounced in individuals with excess weight. BHR is also a common symptom of other respiratory conditions, so its presence alone may not indicate an asthma diagnosis (41, 42). If an individual is suspected of having asthma, he should undergo further analysis. Confirmation of asthma should include lung function tests in order to determine appropriate treatment (17, 39).

Many of the studies conducted on the relationship between asthma and obesity provide inconsistent results. Cross-sectional studies, by design, make it difficult to determine a causal relationship between the two because sequence of onset cannot be determined. A handful of studies have demonstrated that obesity precedes the onset of asthma, thus supporting the hypothesis that obesity is involved in the development of asthma. However, in view of the fact that there is limited information available, it is important that more research be conducted, especially among children.

The data used in this thesis were drawn from the Parental Time, Role Strains, Coping, and Children's Diet and Nutrition project conducted by Texas A&M University, College Station between July 2001 and June 2002, with permission by the authors (43).

Concern about the rise in asthma and overweight among children and concern that these health conditions may continue or perhaps even worsen in adulthood has led me to investigate the subject further. Given the limited availability of research regarding the relationship between asthma and overweight in children and the inconsistencies among this research, it is the objective of this thesis to investigate the relationship between asthma and measures of overweight, allergies, dietary intake of vitamin C and omega-3 fatty acids, the use of dietary supplements, ethnicity, with whom the child lives, parent education, and whether the parent smokes.. The relationship between these variables and allergies will also be investigated. This thesis will analyze the relationship between asthma and allergy medication use and the variables listed above in addition to the presence or absence of asthma and allergy diagnosis. Analyses performed in this thesis are controlled for age and gender. The current data set was chosen and is valuable because it contains randomly drawn subjects and includes variables regarding household structure, medication and dietary supplement use, as well as dietary intake. The availability of these factors allows for the ability to research the possible effects of genetic, cultural, behavioral, and external factors on chronic childhood conditions.

## **CHAPTER II**

### **LITERATURE REVIEW**

This chapter contains a literature review that explains in more detail about the rising prevalence of obesity and asthma in the U.S. It includes a detailed description of each disease as well as explanations on their adverse effects in those afflicted. A special section is devoted to research that explains the possible relationship between asthma and obesity.

#### **Overweight and Obesity**

##### *Prevalence*

The prevalence of obesity has been on the rise for the past two decades and has now become an epidemic. Most recent estimates suggest that the proportion of U.S. adults who are obese is equal to 30%, an increase from 23% in 1994 (4). This means that almost one-third of U.S. adults are obese. If we also consider those who are overweight, this proportion increases to 65% (4). In other words, more than half of U.S. adults are either overweight or obese. In terms of actual numbers, this is equal to 130 million Americans (1).

This trend is not exclusive to adults, though. American children and adolescents have also experienced a significant rise in the prevalence of overweight over the past two decades (7, 8). The prevalence of overweight among children increased more than 100% from 1980 to 1994 (6). From 1980 to 2002, the prevalence of overweight among 6 to 11 year-olds more than doubled from 7% to 16%, and the prevalence of overweight

among 12 to 19 year-olds more than tripled from 5% to 16% (4, 5). The most recent data from the 2003-2004 National Health and Nutrition Examination Survey (NHANES) report that 17.1% of children and adolescents between the ages of 2 and 19 years are overweight (8). Overweight prevalence in the state of Texas has been reported to be 21.3% for elementary-aged girls, 23.6% for elementary-aged boys, 16.7% for middle school-aged girls, and 21.4% for middle school-aged boys (44). Not only is it alarming that more children and adolescents are overweight, but they are now even more overweight than children and adolescents of the past (13). The severity of this disturbing trend is reflected by its inclusion in the Healthy People 2010 objectives. The goal is to decrease the prevalence of overweight among children to 5% by the year 2010 (16, 45). Overweight prevalence also appears to be higher among Hispanics, African Americans, those living in urban areas (3), and those living in poverty (9). This suggests an interaction of several possible factors, which will be discussed below.

What determines whether a person is of a healthful weight or is overweight? BMI, which is dependent on age and gender, is just one way to assess body fat (46). BMI examines weight for height and is more accurate for measuring body fat than by weighing alone (4). BMI correlates relatively well with body fat in children, but does differ with age (10). Other measures that may be used to assess body fat include bioelectrical impedance analysis (47), waist circumference, waist/hip ratio, relative weight, skin folds, dual energy x-ray weighing, and tomographic scans, all of which show a high correlation to body composition (48). Waist circumference and waist/hip ratio are becoming more important as measures of central adiposity (40), but BMI is the



measurement most commonly used in epidemiologic studies (34, 40) because it is easy and inexpensive to measure (47). However, BMI tends to overestimate body fat in those with large muscle mass, common among men (49), and underestimates body fat in those with a normal weight for height but with high adiposity (4, 47).

In adults, a BMI of 25 and above, but below 30 is considered overweight. A BMI of 30 and above is considered obese. Due to variations in growth and development among children, obesity is more difficult to define compared to adults (9). Because all children do not develop at the same rate, the Centers for Disease Control and Prevention (CDC) provides growth charts for children between the ages of 2 and 20 years, which are specific for age and gender (50). For children, BMI is calculated the same way as for adults. Then, it is plotted on the growth curves to obtain a percentile score. If the child's BMI is between the 85<sup>th</sup> and the 95<sup>th</sup> percentiles, he is considered at risk for overweight. If the child's BMI is in the 95<sup>th</sup> percentile or greater, he is considered overweight (50). Because the classification of overweight for children is different from that of adults, the terms overweight and obese are commonly used interchangeably by researchers when referring to the child's weight or BMI.

### *Risk Factors*

Obesity may result from an interaction between behavioral, cultural, environmental, genetic, physiological, and social factors (5, 16). According to one study, obesity that originates from internal effects, such as genetic or physiological factors, accounts for less than 10% of all cases, and obesity that originates from external effects, such as behavioral, cultural, environmental, or social factors, accounts for more

than 90% of all cases (9). A few studies report that the most important risk factor for childhood overweight is parental weight (9, 10), which may be caused by a combination of genetic, lifestyle, or behavioral factors (10). Environmental factors such as changes in entertainment, safety, transportation, and work habits have all contributed to decreased opportunities to participate in physical activity (9). Behavioral factors such as physical inactivity and increased consumption of convenience foods and increases in portion sizes have contributed to the energy imbalance that is believed to be a major contributor to the rise in overweight and obesity in the U.S. (4). Whatever the causes of overweight in children and adolescents may be, it is well known that compared to children with a healthful weight, overweight children have a much higher risk for adult obesity and the morbidities associated with it (9, 19).

### *Consequences*

Overweight and obesity are of concern among Americans of all ages because they are well known to be associated with a number of morbidities (5, 39). Obesity is believed to be an inflammatory state that may contribute to the onset of many chronic diseases (33). Some of these include type 2 diabetes, heart disease, cancer, hypertension, respiratory problems, dyslipidemia, stroke, gall bladder disease, gout, osteoarthritis (4, 5, 9, 11-13), and psychosocial problems (13). Likewise, compared to normal weight children, overweight children have a higher risk of developing psychosocial disorders, dyslipidemia, diabetes, asthma, hypertension (9, 13, 51, 52), and accelerated growth (9) in adulthood and in childhood. The recent increase in type 2 diabetes is believed to be the result of the recent rise in obesity (13). Obesity is also

thought to be responsible for half of adolescent hypertension cases (13). Psychosocial problems, such as depression, low self-esteem, negative self-image, and withdrawal from peers, may be the result of discrimination experienced by overweight individuals at school, at work, in health care, and in social settings (40). Obese children and adults also have a higher risk for reflux, sleep disturbances, and respiratory symptoms (19, 40, 53).

The distribution of body fat also seems to be an important indicator of morbidity risk. Abdominal obesity especially is associated with a high level of morbidity (39, 49). Waist circumference may be measured in addition to BMI to enhance assessment of morbidity risk (39). Increasing triceps skinfold measurement may be associated with an increase in LDL-cholesterol and triglycerides and a decrease in HDL-cholesterol (13). With increasing weight, mortality becomes an increasing risk as well (4, 5, 39, 40). For example, in the U.S. in 2000, obesity was the cause of 400,000 deaths, most of which were likely preventable (4, 39).

BMI and its comorbidities are significant predictors of greater risk of hospitalization (12). According to a study by Raebel et al., obese individuals had more than three times the risk of hospitalization than non-obese individuals (12). In general, obese individuals have higher rates of hospitalizations, outpatient visits, and prescription drug use (12). They consume more asthma, allergic rhinitis, cardiovascular, diabetes, and thyroid medications (12). The prevalence of chronic diseases associated with obesity, increases in medications available, and increases in the consumption of current medications may have contributed to increases health care utilization (5).

Considering all of the morbidities associated with overweight and obesity, along with the increased risk of health care utilization and prescription drug use, it is no surprise that the increasing prevalence of overweight and obesity in the U.S. is associated with great financial burden. The direct and indirect costs associated with overweight and obesity are considerably high (14). Because individuals who are overweight or obese have higher rates of health care and prescription drug use compared to individuals who are not overweight or obese, they have higher hospitalization, prescription, and overall healthcare costs (12). In 2000, the cost of obesity in the U.S. accounted for 7% of total healthcare costs. Most recent estimates report that obesity costs the U.S. \$99 billion per year, 52% of which is related to medical costs (12, 15, 16).

## **Asthma**

### *Prevalence*

Another increasing concern is asthma. Like overweight and obesity, the prevalence of asthma has increased considerably over the past two decades. From 1980 to 1994, the prevalence of asthma among Americans of all ages increased by over 100%. By 1995, asthma affected almost 15 million Americans. Currently those affected by asthma represent 7% of the American population (1). Though the trend is evident among all races, age groups, and genders, the most substantial increases were among 0 to 4 year-olds, with a 160% increase, and among 5 to 14 year-olds, with a 74% increase (3). Asthma is the most common chronic disease in childhood (6), and it is a major cause of disability (2) and medical emergencies (54) in children. Between 1980 and 1996, the prevalence of asthma among children under 18 years of age more than doubled

to historically high rates from 3.6% to 7.5%, with a peak in 1995 (2). In 2005, 12.7%, or 9 million children, had received a diagnosis of asthma, and 70% of these children, 6.5 million, still currently suffered from the disease. According to a study conducted by Petronella et al., asthma prevalence in Texas was reported to be 13.9% (55). Almost two-thirds of children currently suffering from asthma have experienced at least one asthma attack in the previous 12 month period (2). Large-scale studies have reported as many as five or more asthma attacks per year in up to one-third of children with asthma (18). Asthma prevalence among children remains historically high, although it tends to improve from childhood into adulthood (27).

It is possible that the current estimation of asthma does not represent the true asthma prevalence. Asthma may be undiagnosed in individuals who exhibit mild symptoms of asthma, or individuals who have asthma-like symptoms could be falsely diagnosed as having asthma when they do not (48). Depending on self-report to determine asthma prevalence may underestimate the actual prevalence of asthma (17). Many practitioners and researchers disagree on the definition of asthma; therefore, those who may have asthma may not have been diagnosed (17, 33). Individuals who have asthma, but who have not received a diagnosis, may not be receiving the treatment they may need to control their symptoms. Undiagnosed asthma was defined by one study based on the report of cough, wheeze with or without a cold, wheeze with dyspnea, cough or wheeze with exercise, and chest tightness on three occasions in the past 12 months (56).

The prevalence of undiagnosed asthma remains high despite increasing awareness of the disease (57). The possibility of undiagnosed asthma is high among all ethnic groups (58). A study by Clark et al. reported that 9% of children with asthma symptoms had not been diagnosed with the disease. They also found that only 25% of the individuals exhibiting mild symptoms, 35% exhibiting moderate persistent symptoms, and 26% exhibiting severe persistent symptoms had been prescribed medications to treat their asthma. In addition, of the individuals being treated with medication, 74% were still experiencing symptoms (42). Another study conducted by Nolte et al. reported undiagnosed asthma prevalence among individuals at baseline to be as high as 67% (59). On average, research suggests that, among American children, the prevalence rate of undiagnosed asthma is between 5% and 20% (56).

One study suggests that asthma improves with age from childhood (27). This, in fact, may not be due to actual improvement in symptoms, but perhaps may be due to an increased perception of asthma status. This could lead to an underestimation of asthma in adolescents because they have less inclination to seek care for their asthma (58). Another possible explanation for undiagnosed asthma is due to the symptoms themselves. Respiratory symptoms in general are better tolerated than other conditions, such as pain in the chest or the back. Someone experiencing pain may be more likely to report this problem to a health care provider rather than respiratory problems (57). Also, basing a diagnosis of asthma only on recent symptoms can underestimate the prevalence of asthma, which may subsequently lead to undertreatment of the disease (23).

Individuals with undiagnosed asthma may be experiencing symptoms so mild they may not be recognized to be asthma, or the symptoms may be mistaken for other respiratory diseases (57). For example, one study reported that symptoms in undiagnosed individuals did not impair functional status, suggesting a mild form of the disease (56). Undiagnosed individuals with moderate or severe persistent asthma have functional impairment similar to moderate or severe persistent asthmatics with a diagnosis, suggesting that undiagnosed individuals were more tolerant of their symptoms or may not have had access to appropriate care (56). Quality of life (QOL) seems to be impaired in both individuals with asthma diagnosis as well as in those without asthma diagnosis. QOL appears to be lower in those with a diagnosis compared to the undiagnosed group, suggesting that the undiagnosed group has a milder form of asthma (60).

Data regarding the presence or absence of atopy in undiagnosed cases of asthma are conflicting. While one study reports that children with undiagnosed asthma are more likely to have allergic disease (17), another reports the opposite. Joseph et al. reported that children with diagnosed asthma are more likely to report allergies (56). Research also suggests that children with undiagnosed asthma suffer from a greater extent of morbidity compared with diagnosed children (17).

Many studies report an underestimation of asthma prevalence and, subsequently, undertreatment of the disease (42, 59, 60). Clark et al. reported that more than half of subjects were not being treated appropriately according to NAEPP recommendations (42). Not only were individuals with asthma symptoms not being diagnosed, but also

were individuals with asthma symptoms and concomitant allergic rhinitis (59). As many as one-third of the individuals suffering from asthma and allergy symptoms in a study conducted by Nolte et al. had not been diagnosed with either conditions. Likewise, almost half of these individuals were not being treated but would have benefited from treatment according to international guidelines (59).

### *Definition*

Asthma, derived from the Greek word “asthmaino” meaning “panting or gasping” was first described by Hippocrates (460-377BC). Galen was the first to describe the relationship between the upper and lower airways and the etiological link of asthma to bronchospasm (61). In 1911, histamine was described in the pathophysiology of asthma (61). In 1966, IgE and its role in mast cell activation were discovered (61). These discoveries suggested a role of mast cells in airway hyperresponsiveness in asthma (61).

Today, asthma is defined as a chronic inflammatory disorder, which affects the airways. It is often characterized by recurrent episodes of wheezing, breathlessness, chest tightness, airway obstruction, and excessive bronchial responsiveness (19, 20). Researchers have shown elevated levels of inflammatory factors in individuals with persistent asthma, even when the individuals express no symptoms (62). When exposed to a trigger, T-helper lymphocytes stimulate the release of arachidonic acid-derived eicosanoids, called leukotrienes, from inflammatory cells via lipoxygenase action. These leukotrienes are believed to be major mediators in the development of asthma through an inflammatory mechanism (36).



Arachidonic acid can also be converted to thromboxane A<sub>2</sub> and prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) via the action of cyclooxygenase. The PGE<sub>2</sub> activates T-helper lymphocytes, which stimulate the production of IgE from inflammatory cells (36). IgE is responsible for reactions to allergens, and elevated levels of IgE have been reported in individuals with asthma (36). Asthma has been recognized as a disease involving inflammatory cytokine release, including IL-4 and IL-5. Another cytokine, IL-6, has been recognized in obese asthmatics and may suggest a relationship between obesity and asthma through an inflammatory mechanism (53).

According to NAEPP guidelines, the diagnosis of asthma is based on the presence of episodes of asthma symptoms and a demonstration of at least partial reversibility of airway obstruction (48). Symptoms include wheeze that lasts two or more consecutive days; chronic cough that responds to bronchodilation and that persists for 3 to 6 weeks in the absence of rhinitis or sinusitis; and sleep disturbance with dyspnea, cough, or wheeze (42). These symptoms must be exhibited on at least three occasions in the past 12 months for a clinical diagnosis of asthma (60). The diagnosis should be confirmed by lung function testing, including response to bronchodilation and exercise or chemical challenge (42). Other potential diagnoses should be excluded before the diagnosis of asthma is given (48). Frequent pneumonia or bronchitis in a non-smoker, a family history of atopy, or food sensitivity should suggest the possibility of asthma (22). Traditionally, physicians have diagnosed asthma based on the individual's self-report of asthma symptoms. Epidemiological questionnaires mirror this mode of diagnosis (60).

Asthma is characterized by inflammation and narrowing of the airways. It can occur in response to certain triggers, such as allergens, exercise, infections, or respiratory irritants (5). An asthma attack may be characterized by coughing, wheezing, pain or tightness in the chest, shortness of breath, and/or increased mucus production (2, 5, 19). In treatment or diagnosis of asthma, it is important to determine environmental history in order to identify potential triggers (22). Exacerbation of the aforementioned symptoms at night is indicative of asthma (22).

### *Risk Factors*

Asthma may be the result of a combination of environmental, genetic, lifestyle, and socioeconomic factors (3, 21-24). Environmental factors that have been noted as potential causes of asthma include allergens, air pollution, respiratory infections, and tobacco smoke (63, 64). Family history of asthma, family history of allergies, and gender all potentially increase asthma risk (64). Other factors such as crowding, drug use, poverty, poor access to health care, and psychosocial problems, among many more, increase the risk of asthma morbidity (22). Asthma risk is especially important in children, whose airways are anatomically different from those of adults. Children have a shorter neck, a larger tongue, and larger tonsils and adenoids, all of which may influence airway obstruction (19).

Family history of asthma or allergy, exposure to tobacco smoke, rhinitis, and obesity have been recognized by many as the most important risk factors for asthma (6, 11, 25), with allergy posing the biggest threat for asthma onset and asthma symptoms in children and adults (10, 11, 25, 26). As mentioned previously, childhood asthma is

usually allergy-related (26). Most allergic reactions are mediated by IgE and involve the action of histamine. IgE antibodies recognize and bind to an invading antigen then stimulate mast cells to release histamine. Histamine binds to H1 receptors on smooth muscle cells in the lungs, which causes bronchoconstriction (28). In one study, 81% of asthma patients had at least one positive skin prick test (65). Allergic asthma increases in prevalence from childhood into adolescence and peaks in the second decade of life (29). Allergic reactions mediated by IgE are generally responsible for acute asthma symptoms and chronic airway inflammation (29). It has been reported that as many as 85% of school-aged children have asthma combined with allergy to airborne triggers (27). Bloom and Freeman reported that 11% of children in the U.S. under the age of 18 years suffered from allergies in the previous 12 months (66). Non-Hispanic White children are more likely to have respiratory allergies than Hispanic or non-Hispanic Black children (66). The prevalence of respiratory allergies is also influenced by region and by parent education level. Children in the South (15%) are more likely to have respiratory allergies than are children in the Midwest (11%), in the Northeast (10%) or in the West (9%) (66). Children whose parents have at least a high school diploma are more likely to have respiratory allergies (66).

It has already been noted that many factors may be attributed to the development of asthma. More of these factors include income, education level, residence, and race. People with lower education levels and lower incomes, regardless of race, have been reported to experience more asthma symptoms (23). Residence in urban areas is an important risk factor for asthma morbidity and mortality, but this may be influenced

more by socioeconomic status or race rather than place of residence (24). Asthma prevalence is generally higher in minority children compared to White children (17); although, White children have shown a higher prevalence of wheeze specifically, when compared with children of other races (10). Considering race, non-Hispanic Black children have twice the prevalence of asthma compared to non-Hispanic White children and Hispanic children (58), especially among 3 to 10 years-olds (5). The high rates of asthma prevalence among Blacks and Hispanics may not be due solely to race; these rates may be influenced by socioeconomic status as well, since a greater proportion of these races compared to other races tend to live in poverty (3).

Dietary intake is also gaining interest as a risk factor for the development of asthma (6). Inadequate intakes of a number of nutrients are thought to influence asthma, possibly by augmenting inflammation and bronchial hyperresponsiveness (48, 67). In particular, low intakes of antioxidants and omega-3 fatty acids are thought to enhance wheezing (31). Low intakes of fresh, unprocessed foods, especially fruits and vegetables, may be involved in a decline of lung function in children, which may result in an increase in asthma symptoms (31). One study proposes that increasing the intake of fresh fruits and vegetables, especially those rich in vitamin C and other antioxidants, may protect against asthmatic inflammation (10).

A handful of studies have suggested that intake of certain foods, including fresh fruits and vegetables, protects against asthma (10, 31, 67). This is mainly due to the antioxidant effects of nutrients found in these foods, mainly vitamins A, C, and E (10, 67). Vitamin C is a major antioxidant that is present in the surface liquid of the lungs

(10, 67). It works by scavenging superoxide radicals and inflammatory factors, such as PGF<sub>2</sub>-alpha, which contribute to bronchoconstriction (10). Romieu et al. showed an association with vitamin C intake and a decrease in wheezing and an increase in lung function (10). A study by Baker and Ayres also showed an association between vitamin C and improved lung function but not with a decrease in asthma symptoms (67).

Research conducted at the Beltsville Human Nutrition Research Center, U.S.

Department of Agriculture reports that the mean intake of vitamin C for boys and girls 18 years and younger is above the DRI for vitamin C. However, this may disguise variations among individuals. For example, only 73% of the boys and girls in this age group were reported to be meeting the recommended dietary allowances (35). Dietary supplements may be recommended in some cases, but should not replace food intake.

However, research shows that the use of supplements, such as vitamins and minerals, in conjunction with prescribed therapeutic treatments is becoming more and more common, especially among patients with chronic illnesses (68). The safety and efficacy of supplements is not well known, nor is it known whether supplements interact with prescription medications (4, 67). It is recommended that nutrient needs be met through the consumption of healthful foods (4). Healthful food sources of vitamin C, for example, include citrus fruit, strawberries, bell peppers, and broccoli (4).

The prevalence of allergic diseases, such as rhinitis, may be influenced by fatty acid content of foods, mainly omega-6 and omega-3 fatty acids (49). It has been demonstrated that fat intake can change the composition of fatty acids in the body. Through this mechanism, dietary fat may influence eicosanoid production and therefore

may alter inflammatory response (36). Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are long-chain omega-3 fatty acids found in shellfish and fish such as salmon and trout (4, 69). They may also be synthesized in the body from dietary alpha-linolenic acid, an omega-3 fatty acid (36). Omega-3 fatty acids suppress inflammation by inhibiting the mechanisms of eicosanoids, mainly leukotrienes, which are products of arachidonic acid (ARA) (36). The effect of omega-3 fatty acids on ARA metabolism is similar to the mechanism of corticosteroids, which are most commonly used to treat asthma (36). EPA inhibits the production of ARA, an omega-6 fatty acid, and inhibits the action of 5-lipoxygenase, the major enzyme in leukotriene synthesis (36). Recommended intake of omega-3 fatty acids is 1.6 g/day for men, 1.1 g/day for women, and 0.5-1.2 g/day for children aged 0 to 13 years (36). No studies have reported any adverse side effects as a result of omega-3 fatty acid supplementation (36).

Obesity is becoming an increasingly important concern as a risk factor for asthma (3, 6, 10, 31). It has been reported in some studies that increasing weight is associated with worse asthma control (32), and now we are beginning to see correlations between excess body weight and asthma onset as well. Risk for asthma development has been reported to be higher in overweight children compared to children with a healthful body weight (33). Several studies have shown that obesity, especially abdominal obesity (49), contributes to asthma prevalence, even after controlling for physical activity (31); although, this association may be weak when compared to other risk factors, such as allergy (70). In children, overweight at birth or later in childhood contributes to the risk of future asthma (70).

Some studies have observed higher asthma prevalence rates, asthma hospitalization rates, and asthma mortality rates in boys compared with girls (64). Others have observed higher prevalence rates, hospitalization rates, and mortality rates in girls, particularly due to non-allergic asthma (33, 71). These disparities may be the result of differences in airway anatomy or mechanics, hormones, or environmental exposures (31, 33, 64). In general, rates of asthma are typically higher in boys during preadolescence, while in girls the rates are higher during adolescence (31, 64). In adults, women are more likely to have non-allergic asthma (26) and asthma emergency department visits compared with men (15). Factors possibly contributing to gender differences for asthma symptoms, asthma prevalence, and asthma severity include variations in hormones, exposure to allergens, airway caliber, and many lifestyle factors, including diet, activity level, or presence of obesity (7, 64, 72, 73).

The severity of asthma prevalence is indicated by its inclusion in the Healthy People 2010 objectives (45). The identification of risk factors for asthma and allergic diseases is important for the development of appropriate prevention and treatment plans (74). Identification of these risk factors will help to alleviate disease morbidity and mortality (17, 41) and will help to identify individuals with asthma who have not yet been diagnosed (56). Disease prevention and management is an important part of lowering the financial burden associated with chronic diseases (40, 59). Identification of risk factors will also help to control the asthma epidemic in children (33).

### *Consequences*

Asthma is a major cause of morbidity and mortality in the U.S. (21, 75) despite advances in disease management (75), which contributes to a decline in quality of life and a rise in health care costs (36). A survey based on the Behavioral Risk Factor Surveillance System reported that 30% of individuals had an unexpected visit to their physician for asthma, 30% experienced some level of activity limitation due to asthma, and more than 50% had sleep disturbances caused by asthma (76). Asthma and its comorbidities, such as allergic rhinitis, are associated with greater asthma burden and higher health care costs compared to asthma alone (30). Asthma is generally characterized by reversible airway obstruction; however, if asthma is persistent for extended periods of time, it may lead to irreversible airway obstruction (39).

Asthma is responsible for more illness and disability in children than any other childhood disease (5). Asthma may adversely impact playing, learning, and sleeping, which often requires specific interventions that result in increasing medical costs (2). Lenney reported that about one-third of asthmatic children experienced five or more asthma episodes in the previous 12 months, which contributed to a significant number of school absences, limitations in physical activity, and impaired functioning due to sleep disturbances (18). Most notably, children of families that have difficulty complying with asthma management therapy, such as medication use, symptom monitoring, and avoidance of allergens, are likely to experience more frequent exacerbations, activity limitations, school absences, missed sleep, and functional, learning, and behavioral disabilities (63).



Asthma contributes to more school absences than any other chronic medical condition in childhood (17, 18). Some asthma surveys report that as many as 40% of children miss school because of asthma (77), while others have reported a proportion as high as 60% (18). Children with asthma miss more school days than children who do not have asthma (63). Data suggest that students with asthma miss an average number of 10 days of school each year due to asthma symptoms (18). A study by Akinbami reported that approximately 4 million children had an asthma attack in the previous year, which subsequently led to school absences (2).

Having a chronic illness can be stressful, especially for children who may have trouble managing their disease at school (73). Individuals with asthma commonly experience sleep disturbances, which may then affect physical and mental health. Interrupted sleep can cause irritability, aggression, inability to concentrate, and possible memory loss (40). Children with a more severe form of asthma have more trouble adapting and incur more behavioral and academic problems (63).

Both children with diagnosed asthma as well as those with undiagnosed asthma experience decreased quality of life as a result of their asthma (2, 60). Children with diagnosed asthma tend to score lower on quality of life surveys than children with undiagnosed asthma, probably because children who are undiagnosed have a very mild form of asthma (60). A decline in quality of life is usually exacerbated by frequent asthma symptoms, such as dyspnea during exercise, and comorbidities, such as rhinitis (78). Studies show that up to two-thirds of individuals suffer from activity limitations

due to asthma (23). The degree of limitation should be considered when instituting asthma therapy goals (23).

### *Mortality*

Asthma has become an important cause of morbidity and mortality since 1980, and it continues to be a concern despite advances in understanding of the disease and its management (21, 75). There are many factors that could potentially lead to death from asthma. These include severe disease, poor disease control, and psychosocial problems (37). Life threatening asthma exacerbations are of particular concern among children, and may be caused by a number of factors. Parents may not recognize asthma symptoms in their children, or they may ignore them (22). Children may not recognize their own symptoms, may deny them, or may choose not to seek medical attention (22). Or, asthma control may be overestimated and the need for interventions may not be implemented, resulting in worsening asthma that could lead to death (76). Poor adherence to therapeutic treatment of asthma also contributes to asthma morbidity and mortality (79).

Asthma mortality increases with increasing severity, increasing medication use, and increasing age (37). Asthma death rates increased steadily from 1980 to 1998 (2). In 2004, the asthma death rate among children was 2.5 deaths per 1 million children, for a total of 186 asthma deaths (2). Asthma deaths among children under 15 years of age is relatively low, at 2% of all asthma deaths (18), while adolescents seem to be at greater risk for asthma death compared to younger children (2).

In 1980 chronic lower respiratory disease was the 5th leading cause of death among the American population, with 56,000 deaths reported. In 2003, chronic lower respiratory disease increased to the 4<sup>th</sup> leading cause of death, with more than 126,000 deaths reported. In 1980, chronic lower respiratory disease was not a significant cause of death among children and adolescents. In 2003, however, chronic lower respiratory disease was the 8<sup>th</sup> leading cause of death among 5 to 14 year-olds, and it was the 9<sup>th</sup> leading cause of death in 15 to 24 year-olds (5).

Black children seem to be at greater risk for asthma death compared with White children, a disparity which continues to widen (2). The higher prevalence of asthma morbidity seen among minority groups (3) and among those living in urban areas (24) is likely influenced by poverty or socioeconomic background. Children who have severe asthma, uncontrolled asthma, a history of hospitalizations for asthma, or life-threatening episodes for asthma are at highest risk for asthma mortality (2).

### *Health Care*

Health care utilization for asthma continues to rise. Resources commonly utilized for disease care and management include hospitals, emergency departments, physician offices, and outpatient clinics. The observed increase in health care use may be due to an increase in awareness and diagnosis of asthma, which may increase the likelihood that individuals will seek care for asthma symptoms. It may also be due to increasing disease severity, which may warrant a higher number of health care visits (2). As asthma control worsens the number of visits to physician offices, hospitals, and emergency department increases (23, 80) as does the use of rescue medications (23). The

degree of health care utilization for asthma is a good indication of the level asthma control and management (30). In addition to frequency of hospital and emergency department visits (24), medication use is also a good indicator of control, with the use more medication indicating worse asthma control (30).

Compared to children with no asthma, asthmatic children use more health care resources. Children with a combination of asthma and rhinitis use more health care resources than children with asthma alone (30). Although asthma prevalence increases with age, the youngest children use more health care resources for asthma than older children. This may be due to a decline in asthma severity, increased asthma control, or lack of access to health care resources in older children (2).

In 2002, asthma was one of the five leading causes of hospitalizations, along with other ambulatory care-sensitive medical conditions, including gastrointestinal disorders, pneumonia, and seizures (81). These conditions continue to account for 36% of all hospitalizations (81). Risks for asthma hospitalization include previous admission for asthma, previous intubation, and severe asthma symptoms (2). Those with more severe disease, such as moderate to severe persistent, had fourfold higher hospitalization rates, twofold greater urgent visits, and fourfold greater risk of missing more than five days of work due to asthma compared to individuals with a mild form of asthma (23).

Asthma is the leading cause of hospitalization in childhood (22). In 2003, there were 132,000 hospitalizations, 475,000 emergency department visits, and 4.6 million visits to physician offices or outpatient clinics for asthma among children 3 to 17 years of age (5). Race may play a role, as Black children, compared with White children, have

a 250% higher hospitalization rate, a 260% higher emergency department visit rate, and a 500% higher death rate due to asthma, and this disparity continues to widen (2). In 2004, the rate of hospitalization due to asthma was 27 hospitalizations per 10,000 children, for a total of 198,000 hospitalizations (2). The rise in hospitalizations may be influenced by diagnosis of asthma (2), asthma severity, access to health care resources, or compliance with disease management therapy (64).

In 2004, the rate of emergency department visits for asthma was 103 visits per 10,000 children, for a total of 750,000 visits. For children under 18 years of age, visits to the emergency department represent about 2.8% of all emergency department visits for this age group (2). Asthma exacerbations that require the emergency department may indicate severe asthma, uncontrolled asthma, inadequate access to a respiratory specialist, or inappropriate use of the emergency department (2). Recurrent visits to the emergency department may be a risk factor for adverse outcomes including death (2).

### *Cost*

In 1990, health care costs attributed to asthma in the U.S. exceeded \$6.2 billion. Most recent estimates report asthma-related health care costs in the U.S. to be approximately \$11 billion. The increasing morbidity and mortality associated with asthma has played a role in the rising costs (7). The main factors contributing to asthma-related health care costs are hospitalizations and medications (18).

As the number of symptoms increases, so does the cost associated with primary care and secondary care utilization. An increase in symptoms is also associated with an increase in the costs associated with prescription medications, both in individuals with

diagnosed and undiagnosed asthma (82). Although, children with an asthma diagnosis tend to incur higher health care costs than children without a diagnosis of asthma, failure to include these undiagnosed children in the process of estimating asthma-related health care costs, will underestimate the true cost associated with treating asthmatic children (82). As asthma control worsens, health care costs have a tendency to rise. It is reported that health care costs may be more than twofold greater for individuals with uncontrolled asthma compared with individuals whose asthma is under total control (80).

### **Asthma Among the Overweight and Obese**

#### *Prevalence*

Both asthma and obesity have increased at similar rates over the past two decades, suggesting a possible relationship between the two (10, 70). In many countries, there has been a greater increase of obesity in adults with asthma compared with those without asthma (48). Estimates report that a higher proportion of children in the 85<sup>th</sup> percentile or above for BMI developed asthma compared to children below the 85<sup>th</sup> percentile (13, 21). This suggests that excess weight increases the risk of asthma. Yet, others report no significant associations between BMI and asthma in children (33).

An association has been reported between high BMI and asthma prevalence independent of age, gender, birth weight, breast feeding, passive exposure to smoke, socioeconomic status, and exercise (31). In general, this rise is also independent of leisure time activity in men and women (46). In addition to asthma prevalence, research also shows an association between increasing BMI and the lifetime prevalence of

wheeze, previous 12-month wheeze, exercise-induced wheeze, asthma diagnosis, asthma treatment, and airway hyperresponsiveness (31).

Research shows that there may be increased asthma risk especially with increasing abdominal obesity (49). A threshold weight level for which the risk of asthma increases has not been indicated, though (70). It has been suggested that obesity has a greater effect on non-allergic asthma than allergic asthma (26, 33). Many studies report a higher prevalence of obesity among non-allergic asthmatics compared with allergic asthmatics, suggesting no association between obesity and atopy (26, 33, 49). This is often supported by measuring circulating levels of immune factors, such as IgE. Chen et al. showed no correlation between BMI and hay fever; nasal allergies; IgE levels for dust mite, grass, or cat dander; or total IgE levels, suggesting no involvement of atopy in the obesity-asthma association (26). Individuals with allergic asthma are more likely to be children, so evidence suggests that the association between BMI and asthma in these individuals is weak (26). While the absence of immune factors seems to suggest that BMI is not associated with asthma through an allergic pathway, it may be that BMI may have different effects on individuals with allergic asthma compared with those with non-allergic asthma (26).

Many studies report gender differences in the risk of asthma with overweight. In a study by Gold et al., both overweight girls and girls with the largest annual increases in BMI had a greater risk of asthma onset compared with girls with lower BMIs (7). This same study also reported that both boys with the highest annual increases and boys with the lowest annual increases in BMI were at greater risk of asthma onset (7). Other

studies report an increased risk of asthma in overweight boys but not in overweight girls (31, 33). The differences reported in these studies may suggest that there is a vulnerable age for boys and for girls during which overweight increases asthma risk (21).

Schachter et al. reported associations between high BMI and a higher prevalence of atopy, wheeze in the last 12 months, wheeze ever, cough, and medication use in girls but not in boys; however, no association was reported between high BMI and asthma prevalence for either gender, indicating possible changes in mechanical function that could lead to asthma-like symptoms (34). In contrast, another study did report an association between high BMI and asthma prevalence, but this was observed to higher degree in boys than in girls (21).

Similar to the findings in children, gender differences have also been reported in the association between obesity and asthma in adults. Chen et al. reported a 6% increase in asthma risk for every one unit increase in BMI in women. Conversely, men only experienced a 3% increase in asthma risk with every one unit increase in BMI (26). So, while both men and women experienced an increase in asthma risk with increasing BMI, the effect was more pronounced in women (26). Adiposity, as measured by body fat percentage, was related to current asthma in women. In men, adiposity, whether measured by body fat percentage or BMI, was not related to asthma (47). The risk of asthma is twice as great among overweight or obese women compared to women with a healthful body weight (46). In men, increasing BMI was clearly related to asthma, but not clearly so with wheezing, allergic rhinitis, conjunctivitis, or atopic dermatitis (46).



Conversely, in women, increasing BMI was related to wheezing, allergic rhinitis, and conjunctivitis (46).

Obesity is commonly associated with symptoms of asthma including bronchial hyperresponsiveness (41), lifetime wheeze, previous 12-month wheeze, and exercise-induced wheeze (31). BHR (31), low pulmonary volumes, and low thoracic wall expansion have been shown to be more common in obese children compared with children who are not obese (10). Obese children are also more likely to suffer from coughing, wheezing, dyspnea (10, 34), shortness of breath (33), and a greater number of symptom-days than non-obese children (48). Adult asthmatics who were obese reported more symptoms, a greater number of symptom-days, greater activity limitations, and greater medication use (1).

Some studies suggest that BMI is associated with symptoms of asthma and not with asthma itself (34, 83). Many obese individuals exhibit a higher frequency of respiratory symptoms, such as dyspnea, especially on exertion and tend to report more bronchodilator use, yet they often show less frequent airway obstruction compared with non-obese asthmatics. This suggests that these obese individual may just be suffering from asthma-like symptoms, and may have been falsely diagnosed with asthma and are being treated inappropriately (48). Another possible reason for a lack of association between BMI and asthma is that asthma may be underdiagnosed by physicians who inaccurately attribute asthma-like symptoms in overweight children to a lack of physical fitness when the child actually has asthma. Other reports, however, do show an

association between BMI and physician diagnosis of asthma, regardless of atopy, family history of asthma, and exposure to cigarette smoke (34).

Obesity seems to be more prevalent among asthmatics with poor control (53). One study shows an association between high BMI and poor asthma control independent of age, gender, and asthma severity (1). Children with BMI's in the 85<sup>th</sup> percentile or greater tend to have greater asthma severity (3) and worse asthma control (39). This is often measured by number of missed school days, lung function tests, and number of prescribed medications (3). Obesity also seems to hinder the transition from unacceptable asthma control to an acceptable state in both adults and children, even despite optimal pharmacological management (32).

While there may be an association between asthma control and excess weight, findings regarding an association between excess weight and asthma severity are inconsistent (1, 15). In a study by Thomson et al., asthma exacerbations were similar in non-obese asthmatics compared with those who were obese (15), suggesting no difference in asthma severity. While in a study by Lavoie et al., adult asthmatics who were obese reported more symptoms, a greater number of symptom-days, greater activity limitations, and greater medication use (1), indicating a greater asthma severity compared with those who were not obese (14). Overall, asthmatics with excess weight experience worse asthma control, more limitations in daily activities, and a greater number of symptoms. All of these factors contribute to a decline in quality of life in obese asthmatics, compared with non-obese asthmatics (1).

### *Relationship Between Asthma and Obesity*

The association between obesity and asthma may be due to many factors including diet, physical activity, hormones, immune factors, and mechanical factors (31). Body fat is believed to impart excess mechanical load on the airways resulting in hyperresponsiveness. It is also believed to release inflammatory factors which may affect breathing (33, 53). Also, because of anatomical differences in the airways of children and adults, it is believed that excess body weight may have different effects on breathing in children and adults (34).

Adipose tissue is known to release factors that have an effect on most parts of the body. Obesity is considered to be an inflammatory disorder because adipose tissue releases substances, such as leptin, that stimulate the production and release of inflammatory factors, including IL-6, TNF-alpha, C-reactive protein, and chemokines (6, 33, 39). Thus, it is believed that obesity may be associated with asthma through an inflammatory mechanism (6, 32, 33, 38-40, 47, 49, 53). The release of inflammatory factors adversely affects the respiratory system (19, 40), which leads to the development of asthma.

A study conducted by McLachlan et al. shows no association between body fat and airway inflammation (47). In contrast, research conducted by Dixon et al. does show an association between body fat and airway inflammation (53). Serum levels of inflammatory markers have been reported in obese asthmatics (53). An association between asthma and obesity has been reported when a measure of waist circumference was used to define obesity (49). If the nature of the association between obesity and

asthma is through a common inflammatory mechanism, Kronander et al. suggest that it may be better to measure waist circumference rather than BMI (49).

In general, obese individuals breathe at a higher frequency but at lower tidal volumes than individuals who are not obese (6). Central obesity has been shown to lead to a decrease in chest wall compliance and diaphragm movement (19, 47). Decreased movement of the diaphragm leads to less compression on the thorax, less stretching of the bronchial muscles, and a decrease in tidal volume (49). The increased load on the chest in combination with the decreased work of respiratory muscles results in increased work of breathing (19, 39, 47). Increased demand on respiration and a decline in lung function result in airway hyperresponsiveness in obese individuals (19, 39, 49, 53). These changes in lung function result in increased risk of wheeze, shortness of breath, and other asthma-like symptoms (26). A decline in lung function imposed by this mechanical load includes a decrease in forced expiratory volume (FEV), forced vital capacity (FVC), total lung capacity, functional residual capacity, and expiratory reserve volume (39). This leads to breathlessness and other asthma symptoms (26), which is particularly evident during exercise (40). Some studies report an association of adiposity with FEV/FVC ratio but not with airway inflammation. This supports the suggestion that excess weight leads to airway obstruction due to a mechanical mechanism rather than an inflammatory mechanism (47).

It has been reported that a larger proportion of women than men suffer from asthma, leading to the hypothesis that female sex hormones may play a role in this disparity (11). This is of particular concern in obesity, because there is an enhanced

conversion of testosterone to estrogen by adipose tissue, resulting in an increased risk of asthma (39). Estrogen may enhance cytokine profiles in obese individuals, especially women, which would enhance the inflammatory mechanism of obesity on asthma (46). Obesity is also believed to result in reduced progesterone levels. This would in turn lead to a reduction in beta-2 receptor function and therefore a reduction in bronchial smooth muscle relaxation (26). This is also supported by the observation that asthmatic women experience increased symptoms and decreased pulmonary function during the menstrual cycle when estrogen levels are at their peak (41). Post-menopausal women experience less bronchial hyperresponsiveness compared with pre-menopausal women; however, bronchial hyperresponsiveness increases in post-menopausal women who are being treated with estrogen replacement therapy (41).

There is some evidence that diet may play a role in the association between obesity and asthma (11, 33). Overweight and obese individuals tend to consume diets high in calories and low in important micronutrients (10). Some researchers have identified specific components of the diet that may play a direct role in this association, particularly vitamin C and omega-3 fatty acids, as were mentioned earlier in this chapter (49, 70). Research on the association between fruit and vegetable intake on overweight among children is limited (84); however, one study did find that overweight children and adults consumed fewer fruits than healthy weight children and adults. The correlation between vegetable consumption and weight status was found to be significant only among adults, though (85). Finally, obese individuals generally have high levels of comorbidities, which may play a role in the association between obesity and asthma.

For example, obese individuals have a higher occurrence of gastroesophageal reflux disease (GERD), which has been identified as a risk factor for asthma (19, 29, 53, 70). It has been reported that 45% to 65% of asthmatic children and adults also suffer from GERD (29).

Some have suggested that obesity is a consequence of asthma rather than a cause. Individuals with asthma that is poorly controlled may be prone to adopt a sedentary lifestyle that then leads to weight gain (9, 38, 46, 49). Another mechanism may be that individuals with asthma may be taking medications, mainly corticosteroids, which may lead to weight gain (48).

### *Consequences*

Obese asthmatics suffer from comorbidities commonly associated with asthma, such as rhinitis and sinusitis, to a similar extent as non-obese asthmatics (53). Additionally, they tend to suffer more from GERD and sleep disturbances compared to non-obese asthmatics (53). GERD, as mentioned previously, is believed to be a risk factor for asthma. Therefore, obese individuals are at greater risk for asthma and asthma-like symptoms (53). Obesity is also a risk factor for obstructive sleep apnea as well as other respiratory problems (19, 40). Obstructive sleep apnea is characterized by a partial or complete obstruction of the airways that leads to hypoxia, then to arousal and sleep disturbance. This often is the cause of daytime sleepiness, irritability, aggression, impaired concentration, and a lower quality of life (19). In children, it may result in impairment of school function, learning, and memory (13).

Adult asthmatics with obesity reported more symptoms, a greater number of symptom-days, greater activity limitations, and greater medication use (1), indicating a greater asthma severity compared with those who were not obese (14). Likewise, being overweight was associated with worse asthma severity and asthma control in children. Because overweight in children has been associated with poor asthma control, it is feared that this may lead to an increase in the number of young adults with severe asthma (39).

A study by Van Gent et al. suggests that asthma with excess body weight results in a lower quality of life than asthma alone or excess body weight alone. Based on the effect of asthma alone and excess body weight alone on quality of life, it was expected that asthma with excess body weight would result in a 15% lower score on quality of life. However, the combined effect was a 25% lower score, proving to be the lowest quality of life of all the groups of children (38). Measures of quality of life provide insight into functional impairment in the patient's daily life (38). Men typically experience a sharper decline in quality of life compared to women. This is likely because men tend to be more active than women, therefore, loss of the capacity to perform certain activities may be more meaningful in men (1).

Overall, obese asthmatics suffer more symptoms, use more medications, and visit the hospital and emergency department more frequently than non-obese asthmatics (1, 15, 48, 86). As much as 75% of asthmatics visiting the emergency department for asthma symptoms may be obese (15). Obese asthmatics tend to use more rescue medications than non-obese asthmatics, most likely indicating worse asthma control

(53). Compared with non-obese children, obese children who also have asthma are more likely to be treated with oral corticosteroids (38, 48) or inhaled corticosteroids (38). In general, obese asthmatics are reported to use high-dose corticosteroids (65) and long-acting bronchodilators more frequently than those who are not obese (1). They are also more likely to be taking a combination of two or more medications to treat their asthma (38, 48, 86).

### **Summary**

Many researchers agree that evidence leans toward excess body weight as a risk factor for the onset of asthma (14, 31, 38, 48, 49). Some argue that because obesity is associated with asthma severity independent of possible confounders, it is therefore a potentially modifiable risk factor for asthma (14). The observation that weight reduction in obese asthmatics results in improvement of respiratory symptoms and improves lung function also supports the hypothesis that obesity is a risk factor for asthma (14, 38, 40, 49). Multiple studies reported a positive association between BMI and asthma development. The development of asthma followed the development of obesity in these studies, which supports the hypothesis that excess body weight is a risk factor for asthma (48). It may be that preconditions for asthma exist in certain individuals before the age of 6 years, and increasing BMI after this age further enhances the risk of developing asthma symptoms (7).

Despite the research that has been conducted, there is still disagreement on the existence of a causal link between obesity and asthma (7, 14, 39, 40, 83, 86). There are those who suggest that there is no association between obesity and asthma or airway



hyperresponsiveness (34), and then there are others who hypothesize that asthma is a risk factor for obesity in children, adolescents, and adults (3, 14, 48). This may be supported by the observation that asthmatics tend to adopt a sedentary lifestyle in order to avoid exacerbations, therefore they are prone to gain weight (14). Others suggest that weight gain develops as a side effect of corticosteroid therapy (48), a regimen commonly used to treat asthma.

In many cases, it cannot be determined whether obesity or asthma occurred first (7), mostly due to the nature of the study design (39, 86). It is difficult to determine the sequence of onset of asthma and obesity in cross-sectional studies (9, 10, 31, 41). Data regarding the relationship between excess body weight and asthma in children are limited (38, 77), and the results of those studies that have been conducted on the relationship between asthma or asthma symptoms and obesity are often inconsistent (10, 11).

As mentioned in the previous chapter, it is the objective of this thesis to analyze the relationship between asthma and obesity in pre-adolescent and adolescent children. Asthma diagnosis will be compared with measures of obesity, allergy diagnosis, dietary vitamin C and omega-3 fatty acid intake, vitamin C and omega-3 fatty acid supplement use, ethnicity, with whom the child lives, mother's education, and whether the mother smokes. The use of prescription asthma medication and prescription allergy medication will be compared with the presence of asthma and allergies, as well as with measures of obesity.

## **CHAPTER III**

### **METHODS**

This chapter will describe the data and methods used to collect the data in more detail. The data used in this analysis were derived from the Parental Time, Role Strains, Coping, and Children's Diet and Nutrition project conducted by Texas A&M University between July 2001 and June 2002, with permission by the authors. The complete data set is quite extensive and was not used in its entirety. Instead, variables of interest for this analysis were selected from the data set. The resulting sample and its variables are described in more detail later in this chapter. Analysis was conducted on asthma prevalence, overweight prevalence, and the association between the two in males and females between the ages of 9 and 11 years and between 13 and 15 years. Statistical analysis was conducted using SPSS statistical software. Tests used in the analysis of the sample are discussed at the end of this chapter.

#### **Data**

A random sample of approximately 300 households in the Houston Metropolitan Statistical Area, an area with a high concentration of ethnic groups, was generated by random digit dialing. A randomly generated sample of more than 10,000 phone numbers was obtained from Survey Sampling, Inc. Unassigned, disconnected, and non-household phone numbers had been removed from the sample. A 50% contact rate, 50% cooperation rate, and 50% completion rate were assumed when determining sample size. The project was designed to include households with a child between the ages of 9 and

11 years or between 13 and 15 years. To avoid difficulties with self-reporting data, children younger than 9 years were excluded. Because children often undergo puberty at age 12 (43), children of this age were also excluded in order to create a sample of pre-pubertal and post-pubertal children. Data were collected in the form of a telephone interview of each parent, a self-administered questionnaire of each parent, a personal child interview, a 24-hour child diet recall, and a child two-day diet diary. The interview and questionnaire were identical for each parent and the child's interview mimicked the parent interview. The 24-hour diet recall for the day prior to the interview was obtained through a multiple-pass method. A two-dimensional booklet containing photographs of specific foods was used to help determine portion sizes (87). The booklet was retained by the children to aid in the two-day diary. Information regarding the two-day diet diary was obtained by the interviewer over the telephone. Dietary intakes for the three days were analyzed with the Food Processor program to obtain nutrient intakes for each day (88). A brief physical exam was administered to children to obtain body composition measurements. Anthropometric measurements, including height, weight, waist circumference, hip circumference, triceps skinfold thickness, and subscapular skinfold thickness, were obtained according to procedures established by Lohman et al (89). Interviewers were trained to take body composition measurements and were not allowed to examine the children until they could obtain three consecutive measures within one centimeter of each other. Children were asked to wear lightweight, loose-fitting clothes and were asked to remove their shoes to obtain height and weight. Height was measured to the nearest 1/8 inch using a non-stretch metal tape and a metal triangle. Weight was

measured to the nearest 0.5 pound using a 12 inch by 12 inch 500 pound parcel scale. Waist circumference was measured to the nearest 0.1 cm using a flexible nylon tape measure. The measurement was taken at the narrowest section below the rib cage and above the navel. Hip circumference was taken while the child was standing. Three triceps and three subscapular skinfold measurements were taken on the right side of the body to the nearest millimeter using a skinfold caliper. The average of the three measures was used for analysis.

The parent telephone interview and self-administered questionnaire were designed to gather information regarding employment, parenting style, concern about children's eating habits, parental health, children's health, and demographics and sociological information, economic information, and time use, respectively. The child interview was designed to gather information on child's relationships with parents, parenting style, child's health, self-esteem, family meal rituals, children's work, dietary behavior, activity level, and demographics. Data were collected from more than 300 families, including mothers, fathers (if present), and one child. The final data set consisted of over 2000 variables for each family.

### **Variables**

For this thesis project, the variables of interest for the child included dietary vitamin C intake, dietary omega-3 fatty acid intake, supplement usage, including the use of multi-vitamin/mineral supplements, omega-3 fatty acid supplements, and vitamin C supplements; vitamin C and omega-3 fatty acid dietary intake; ethnicity; gender; age; height; weight; waist circumference; hip circumference; triceps skinfold thickness;

subscapular skinfold thickness; composition of household; allergy diagnosis; allergy medication use; asthma diagnosis; and asthma medication use. The variables of interest for the parents included smoking habit, weight, height, and education completed. If the subject could not be categorized by age or gender, the case was thrown out. Only cases in which the subject was between the ages of 9 and 11 years or 13 and 15 years were retained for analysis. Because fathers were not always present, because many responses from fathers were missing, and because parents completed identical interviews and questionnaires, only responses from mothers were retained for analysis. The resulting sample contained 303 cases and 31 variables.

The parent variables of interest were obtained from the parent interview. MOMWT was a continuous variable that represented the weight of the mother in pounds. It was labeled as ‘mother weight (lbs)’ and was derived from the response to the question, “How much do you weight?” MOMHT was a continuous variable that represented the height of the mother in inches. It was labeled as ‘mother height (in)’ and was derived from the response to the question, “How tall are you?” MOMBMI was a continuous variable that represented the mother’s BMI. It was labeled as ‘mother BMI’ and was calculated from the variables MOMWT and MOMHT. MOMBMICAT was a categorical variable that represented the mother’s BMI category. It was determined by categorizing MOMBMI into the following categories according to the CDC: underweight, normal weight, overweight, or obese. MOMSMK was a categorical variable that represented whether the mother smokes. It was labeled as ‘mother smokes’ and was derived from the response to the question, “Do you smoke cigarettes?”

Responses included yes or no. MOMEDU was a categorical variable that represented the level of education completed by the mother. It was labeled as ‘mother level of education’ and was derived from the response to the question, “How much education have you completed?” Optional responses ranged from ‘some grammar school’ to ‘completed graduate school’.

Child variables regarding diagnosed medical conditions and prescription medication usage were also obtained from the parent interview. ALLERGIES was a categorical variable that represented the diagnosis of the child with allergies. It was labeled as ‘diagnosed with allergies’ and was derived from the response to the question, “Has your child ever been diagnosed with the following conditions?” Optional responses included asthma, diabetes, heart disease, high blood cholesterol, eating disorder, attention deficit hyperactivity disorder, and other. ASTHMA was a categorical variable that represented a diagnosis of the child with asthma. It was labeled as ‘diagnosed with asthma’ and was derived from the response to the same question asked for the diagnosis of allergies. ALLRX and ASTHRX were categorical variables derived from the question, “Why was it prescribed?” following a ‘yes’ response to the previous question. ALLRX represented the use a medication for allergies and was labeled as ‘taking allergy prescription’. ASTHRX represented the use of a medication for asthma and was labeled as ‘taking asthma prescription’. If the medication was reported to be taken for allergies, a ‘yes’ response was marked for ALLRX and the remaining responses for that variable were marked as ‘no’. If the medication was reported to be

taken for asthma, a 'yes' response was marked for ASTHRX and the remaining responses for that variable were marked as 'no'.

Child variables were also obtained from the child interview. GUARDIAN was a categorical variable that represents with whom the child lives. It was labeled as 'with whom child lives' and was derived from the response to the question, "Do you live with your real or biological mom and dad, other relatives, or with step-parents?" Optional responses included with real (biological) mom and dad, with biological mom only (no other parent), with biological mom and step-dad, with real dad and step-mom, with biological mom and other relatives, or with adoptive or foster parents. Given that a vast majority of responses that consisted of 'biological mom and dad', alternative responses were collapsed into a single category labeled as 'other'. AGE was a continuous variable that represented the age of the child in years. It was labeled as 'age (yrs)' and was derived from the response to the question, "How old are you?" ETHNICITY was a categorical variable that represented the ethnic background of the child. It was labeled as 'ethnic background' and was derived from the response to the question, "What is your ethnic background?" Optional responses included Hispanic, Black, White, or other. Given the numerous responses of White ethnicity, responses other than White were collapsed into a single category labeled as 'other'. GENDER was a categorical variable that represented the gender of the child. It was labeled as 'gender' and was determined by the interviewer as male or female.

OMEGA, VITC, and MULTI were categorical variables that were derived from the response to the question, "During the past month, have you taken supplements in pill,

liquid, or powder form?” Children were allowed to choose from an extensive list of supplements and were given the opportunity to report any supplements not on the list. OMEGA represented the use of an omega-3 fatty acid supplement by the child and was labeled as ‘omega-3 fatty acid supplement’. If a child reported using an omega-3 fatty acid supplement, then a ‘yes’ response was marked for this variable. If no use of this supplement was reported, a ‘no’ response was marked for this variable. VITC represented the use of a vitamin C supplement and was labeled as ‘vitamin C supplement’. If a child reported using a vitamin C supplement, then a ‘yes’ response was marked for this variable. If no use of this supplement was reported, a ‘no’ response was marked for this variable. MULTI represented the use of a multi vitamin/mineral supplement by the child and was labeled as ‘multi vitamin/mineral supplement’. Reported supplementation with a multiple vitamin and/or a multiple vitamin/multiple mineral by the child was combined into one variable, MULTI, and was marked as a ‘yes’ response for this variable. If no use of either type of supplement was reported, a ‘no’ response was marked for MULTI.

Child anthropometric variables were obtained by the interviewer during an anthropometric assessment. HEIGHT was a continuous variable that represented the height of the child in inches. It was labeled as ‘height (in)’ and was measured to the nearest 1/8 inch without shoes and without leaning against a wall. WEIGHT was a continuous variable that represented the weight of the child in pounds. It was labeled as ‘weight (lbs)’ and was measured to the nearest half pound without shoes. BMI was a continuous variable that represented the child’s BMI. It was labeled as ‘BMI’ and was



calculated from the variables WEIGHT and HEIGHT. BMIPER was a continuous variable that represented the child's BMI percentile. It was labeled as 'BMI percentile' and was determined using the variable BMI and the CDC growth curves. BMICAT was a categorical variable that represented the child's BMI percentile category. It was labeled as 'BMI percentile category' and was determined by categorizing BMIPER into the following categories according to the CDC: underweight, healthy weight, at risk for overweight, or overweight. WAIST was a categorical variable that represented the waist circumference of the child in centimeters. It was labeled 'waist circumference (cm)'. Measurements were then categorized into the following ranges for analysis: less than 50.0 cm, 50.0 cm to 59.9 cm, 60.0 cm to 69.9 cm, 70.0 cm to 79.9 cm, 80.0 cm to 89.9 cm, 90.0 cm to 99.9 cm, or 100.0 cm or more. HIP was a categorical variable that represented the hip circumference of the child in centimeters. It was labeled as 'hip circumference (cm)'. Measurements were then categorized into the following ranges for analysis: less than 50.0 cm, 50.0 cm to 59.9 cm, 60.0 cm to 69.9 cm, 70.0 cm to 79.9 cm, 80.0 cm to 89.9 cm, 90.0 cm to 99.9 cm, or 100.0 cm or more. WAISTHIP was a categorical variable that represented the waist/hip ratio of the child. It was labeled as 'waist/hip ratio' and was calculated from the variables WAIST and HIP then was categorized into the following ranges for analysis: less than 0.70, 0.70 to 0.74, 0.75 to 0.79, 0.80 to 0.84, 0.85 to 0.89, 0.90 to 0.94, 0.95 to 0.99, 1.00 to 1.99, or 2.00 to 2.99. TRICEPS was a categorical variable that represented the triceps skinfold thickness of the child in millimeters. Three measurements were taken and averaged. The result was then categorized into the following ranges for analysis: less than 5.00 mm, 5.00 mm to 9.99

mm, 10.00 mm to 14.99 mm, 15.00 mm to 19.99 mm, 20.00 mm to 24.99 mm, 25.00 mm to 29.99 mm, 30.00 mm to 34.99 mm, 35.00 mm to 39.99 mm, 40.00 mm or more. SUBSCAP was a categorical variable that represented the subscapular skinfold thickness of the child in millimeters. Three measurements were taken and averaged. The result was then categorized into the following ranges for analysis: less than 5.00 mm, 5.00 mm to 9.99 mm, 10.00 mm to 14.99 mm, 15.00 mm to 19.99 mm, 20.00 mm to 24.99 mm, 25.00 mm to 29.99 mm, 30.00 mm to 34.99 mm, 35.00 mm to 39.99 mm, 40.00 mm or more.

OMEGAINAKE was a continuous variable that represented dietary intake of omega-3 fatty acids in grams by the child. It was labeled as ‘omega-3 fatty acid intake (g)’ and was determined using the child’s 24-hour diet recall and 2-day food diary.

VITCINTAKE was a continuous variable that represented dietary intake of vitamin C in milligrams by the child. It was labeled as ‘vitamin C intake (mg)’ and was determined using the child’s 24-hour diet recall and 2-day food diary. MEETOMEGA was a categorical variable that represented whether the child’s dietary intake met the AI for omega-3 fatty acids. It was labeled as ‘meeting omega-3 fatty acid AI’ and was determined using the DRI tables provided by the USDA (90). Responses consisted of ‘yes’ if the child’s intake met the omega-3 fatty acid AI or ‘no’ if it did not.

MEETVITC was a categorical variable that represented whether the child’s dietary intake met the DRI for vitamin C. It was labeled as ‘meeting vitamin C DRI’ and was determined using the DRI tables provided by the USDA (90). Responses consisted of ‘yes’ if the child’s intake met the vitamin C DRI or ‘no’ if it did not.

**Analysis**

Dependent variables were dichotomous, and included allergy diagnosis, asthma diagnosis, and prescription medication use. Therefore binary regression analysis was used to analyze the relationship between these variables and measures of overweight, asthma/allergy diagnosis, ethnicity, guardian, household salary, mother's education, and mother's smoking habit. All analyses were conducted using SPSS statistical software and were controlled for age and gender. The Wald statistic was used to determine the presence of a relationship between dependent and independent variables. Odds ratios provided insight into whether an existing correlation between the dependent and independent variables was a positive or inverse correlation. All tests were analyzed using a significance level of  $p < 0.05$ .

## CHAPTER IV

### RESULTS

This chapter will discuss variable frequencies and percentages and binary regression analysis. All analyses were performed on the sample that was described in the previous chapter, the pre-adolescent sample, the adolescent sample, males, and females. Different sample sizes for each variable represent different response rates. Tests of significance were measured at  $p < 0.05$ . The results of analyses are represented by tables throughout the chapter, which contain significant findings only.

#### **Frequency Distributions**

The entire sample was comprised of 303 subjects, roughly half male and half female. The sample can also be divided by age group, containing approximately half pre-adolescent children and half adolescent children (Table 1). The male and female samples showed a similar age distribution with 52.9% pre-adolescents and 47.1% adolescents for males and 53.4% pre-adolescents and 46.6% adolescents for females. The mean age of the sample was  $11.94 \pm 2.168$  years (Table 1). The pre-adolescent sample, which was composed of children that have not yet entered into adolescence, is comprised of 9, 10, and 11 year-olds. The mean age of this sample was  $10.06 \pm 0.808$  years. The adolescent sample, composed of children who have already entered into adolescence, included 13, 14, and 15 year-olds. The mean age for this sample was  $14.08 \pm 0.803$  years. The mean ages of males and females were similar at  $12.00 \pm 2.144$  years and  $11.89 \pm 2.199$  years, respectively. The ethnic distribution of the entire sample was

**TABLE 1** Frequency (%) distributions for demographic variables.

<b>Gender</b>	Entire Sample	Pre-Adolescents (9-11 years)	Adolescents (13-15 years)	Male	Female
<b>N</b>	303	161	142	155	148
1 = Male	155 (51.2)	82 (50.9)	73 (51.4)	155 (100)	0
2 = Female	148 (48.8)	79 (49.1)	69 (48.6)	0	148 (100)
<b>Age</b>					
<b>N</b>	303	161	142	155	148
9	48 (15.8)	48 (29.8)	0	19 (12.3)	29 (19.6)
10	56 (18.5)	56 (34.8)	0	34 (21.9)	22 (14.9)
11	57 (18.8)	57 (35.4)	0	29 (18.7)	28 (18.9)
13	40 (13.2)	0	40 (28.2)	20 (12.9)	20 (13.5)
14	50 (16.5)	0	50 (35.2)	25 (16.1)	25 (16.9)
15	52 (17.2)	0	52 (36.6)	28 (18.1)	24 (16.2)
<b>Ethnicity</b>					
<b>N</b>	301	159	142	154	147
1 = White	217 (72.1)	117 (73.6)	100 (70.4)	108 (70.1)	109 (74.1)
2 = Other	84 (27.9)	42 (26.4)	42 (29.6)	46 (29.9)	38 (25.9)
<b>Guardian</b>					
<b>N</b>	294	157	137	151	143
1 = Biological mom and dad	236 (80.3)	131 (83.4)	105 (76.6)	122 (80.8)	114 (79.7)
2 = Other	58 (19.7)	26 (16.6)	32 (23.4)	29 (19.2)	29 (20.3)

represented by a White majority (Table 1). In fact, White ethnicity composed at least 70% of all of the samples, which may make it difficult to determine the effect of specific ethnic groups on certain factors. Results of analyses using this variable will only reveal the effect of being White or not being White. A vast majority (80%) of children reported living with their biological mom and dad (Table 1). The remaining 20% was comprised of children living with their biological mom only, with their biological mom and step dad, with their biological mom and other relatives, and with adoptive or foster parents, combined. Results of analyses using this variable will reveal the effect of living in a household with both biological parents versus living in a household that does not have both biological parents.

The mean height of the entire sample was  $60.40 \pm 5.595$  inches and the mean weight was  $109.78 \pm 37.802$  pounds (Table 2). Mean height and mean weight were greater in the adolescent sample compared to the pre-adolescent sample. Likewise, mean height and mean weight are greater in males compared to females. The mean BMI of the entire sample was  $20.91 \pm 5.080$  (Table 2). The mean BMI of the adolescent sample was greater than that of the pre-adolescent sample, and the mean BMI of males was greater than that of females. BMI percentile was identified using the CDC growth charts, which was in turn used to determine overweight and at risk for overweight. The mean BMI percentile of the entire sample was  $64.61 \pm 28.696$  (Table 2). In contrast to BMI, the mean BMI percentile of the pre-adolescent sample was greater than that of the adolescent sample. The mean BMI percentile of the males was higher than that of the females. While the mean BMI percentiles were within the healthy weight range for all samples, the standard errors for these statistics indicate that the confidence intervals for each of the samples included BMI percentiles that are considered at risk for overweight. More than half of the children in the entire sample fell into the category of healthy weight (Table 3). Just below 20% of children were overweight and more than one-third of the children were either overweight or at risk for overweight. Similar patterns were observed in the other samples as well. More females were at a healthy weight compared to males, while a greater proportion of males than females were overweight or at risk for overweight.

The mean waist circumference of the entire sample was  $69.02 \pm 12.738$  cm (Table 3). The mean waist circumference of the pre-adolescent sample ( $65.41 \pm 11.213$

**TABLE 2** Sample size, mean, and standard error of height, weight, and BMI.

	Entire Sample	Pre-Adolescent (9-11 years)	Adolescent (13-15 years)	Male	Female
<b>Height (in)</b>					
N	303	161	142	155	148
Mean $\pm$ SE	60.40 $\pm$ 5.595	56.34 $\pm$ 3.494	64.99 $\pm$ 3.621	61.03 $\pm$ 6.194	59.73 $\pm$ 4.821
<b>Weight (lb)</b>					
N	303	161	142	155	148
Mean $\pm$ SE	109.78 $\pm$ 37.802	88.60 $\pm$ 24.771	133.80 $\pm$ 35.650	113.89 $\pm$ 37.902	105.48 $\pm$ 37.340
<b>BMI</b>					
N	303	161	142	155	148
Mean $\pm$ SE	20.91 $\pm$ 5.080	19.55 $\pm$ 4.380	22.45 $\pm$ 5.387	21.19 $\pm$ 5.168	20.62 $\pm$ 4.988
<b>BMI percentile</b>					
N	303	161	142	155	148
Mean $\pm$ SE	64.61 $\pm$ 28.696	65.05 $\pm$ 29.454	64.12 $\pm$ 27.907	66.68 $\pm$ 28.814	62.45 $\pm$ 25.508

cm) was smaller than that of the adolescent sample (72.92  $\pm$  13.049 cm). Males had a larger mean waist circumference (70.04  $\pm$  9.565 cm) than females (67.95  $\pm$  11.716 cm). Mean hip circumference of the entire sample was 93.75  $\pm$  13.764 cm (Table 3). Similar to mean waist circumference, the mean hip circumference of the pre-adolescent sample (77.97  $\pm$  10.859 cm) was smaller than that of the adolescent sample (90.31  $\pm$  13.797 cm). In contrast to mean waist circumference, males had a lower mean hip circumference (82.96  $\pm$  13.847 cm) than did females (84.58  $\pm$  13.674 cm). Waist and hip circumference were analyzed to determine waist/hip ratio, which has been used in research as a measure of central adiposity. The mean waist/hip ratio of the entire sample was 0.83  $\pm$  0.102 (Table 3). Pre-adolescents had a larger waist/hip ratio (0.84  $\pm$  0.071) compared to adolescents (0.81  $\pm$  0.128), as did males (0.84  $\pm$  0.073) compared to females (0.81  $\pm$  0.124).

**TABLE 3** Frequency (%) distributions for BMI category and waist and hip circumference.

<b>BMI category</b>	Entire Sample	Pre-Adolescent (9-11 years)	Adolescent (13-15 years)	Male	Female
N	303	161	142	155	148
1 = Underweight	3 (1.0)	1 (0.6)	2 (1.4)	2 (1.3)	1 (0.7)
2 = Healthy weight	188 (62.0)	99 (61.5)	89 (62.7)	89 (57.4)	99 (66.9)
3 = At risk for overweight	56 (18.5)	30 (18.6)	26 (18.3)	30 (19.4)	26 (17.6)
4 = Overweight	56 (18.5)	31 (19.3)	25 (17.6)	34 (21.9)	22 (14.9)
<b>Waist circumference (cm)</b>					
N	303	161	142	155	148
1 = Less than 50	6 (2.0)	3 (1.9)	3 (2.1)	5 (3.2)	1 (0.7)
2 = 50 to 59	53 (17.5)	51 (31.7)	2 (1.4)	23 (14.8)	30 (20.3)
3 = 60 to 69	124 (40.9)	61 (37.9)	63 (44.4)	55 (35.5)	69 (46.6)
4 = 70 to 79	69 (22.8)	28 (17.4)	41 (28.9)	39 (25.2)	30 (20.3)
5 = 80 to 89	31 (10.2)	13 (8.1)	18 (12.7)	22 (14.2)	9 (6.1)
6 = 90 to 99	14 (4.6)	4 (2.5)	10 (7.0)	8 (5.2)	6 (4.1)
7 = 100 or more	6 (2.0)	1 (0.6)	5 (3.5)	3 (1.9)	3 (2.0)
<b>Hip circumference (cm)</b>					
N	303	161	142	155	148
1 = Less than 50	6 (2.0)	2 (1.2)	4 (2.8)	4 (2.6)	2 (1.4)
2 = 50 to 59	0	0	0	0	0
3 = 60 to 69	36 (11.9)	35 (21.7)	1 (0.7)	21 (13.5)	15 (10.1)
4 = 70 to 79	76 (25.1)	60 (37.3)	16 (11.3)	40 (25.8)	36 (24.3)
5 = 80 to 89	88 (29.0)	42 (26.1)	46 (32.4)	41 (26.5)	47 (31.8)
6 = 90 to 99	67 (22.1)	17 (10.6)	50 (35.2)	34 (21.9)	33 (22.3)
7 = 100 or more	30 (9.9)	5 (3.1)	25 (17.6)	15 (9.7)	15 (10.1)
<b>Waist/hip ratio</b>					
N	303	161	142	155	148
1 = Less than 0.70	3 (1.0)	1 (0.6)	2 (1.4)	1 (0.6)	2 (1.4)
2 = 0.70 to 0.74	28 (9.2)	2 (1.2)	26 (18.3)	3 (1.9)	25 (16.9)
3 = 0.75 to 0.79	64 (21.1)	31 (19.3)	33 (23.2)	20 (12.9)	44 (29.7)
4 = 0.80 to 0.84	103 (34.0)	58 (36.0)	45 (31.7)	61 (39.4)	42 (28.4)
5 = 0.85 to 0.89	68 (22.4)	43 (26.7)	25 (17.6)	42 (27.1)	26 (17.6)
6 = 0.90 to 0.94	25 (8.3)	20 (12.4)	5 (3.5)	19 (12.3)	6 (4.1)
7 = 0.95 to 0.99	8 (2.6)	5 (3.1)	3 (2.1)	7 (4.5)	1 (0.7)
8 = 1.00 to 1.99	3 (1.0)	1 (0.6)	2 (1.4)	2 (1.3)	1 (0.7)
9 = 2.00 to 2.99	1 (0.3)	0	1 (0.7)	0	1 (0.7)



Triceps skinfold thickness and subscapular skinfold thickness may be used to estimate subcutaneous fat by using a skinfold caliper to measure skinfold thickness at respective locations on the body. The mean triceps skinfold of the entire sample was  $15.95 \pm 6.891$  mm (Table 4). Mean triceps skinfold measurement was slightly higher in pre-adolescents ( $16.13 \pm 6.532$  mm) than in adolescents ( $15.76 \pm 7.296$  mm). Females had a higher mean triceps skinfold measurement ( $17.33 \pm 6.284$  mm) compared to males ( $14.65 \pm 7.204$  mm). Reference values derived from the 1999-2002 NHANES report reference values for triceps skinfold measurements for children between the ages of 2 and 19 years (91). According to this report, triceps skinfold measurements between 21.4 mm and 22.3 mm and between 21.6 mm and 18.8 mm are reference values for males aged 9 to 11 years and 13 to 15 years, respectively, who are at risk for overweight. No reference values for triceps skinfold thickness were indicated for overweight males between the ages of 9 and 11 years or between 13 and 15 years. Triceps skinfold measurements between 21.7 mm and 24.8mm and between 26.7 mm and 26.1 mm are reference values for females aged 9 to 11 years and 13 to 15 years, respectively, who are at risk for overweight. A measurement of 33.4 was the only reference value indicated for triceps skinfold thickness for overweight females aged 13 years. The mean subscapular skinfold measurement of the entire sample was  $11.40 \pm 6.930$  mm (Table 4). The mean subscapular skinfold of adolescents (n=141,  $11.97 \pm 6.612$  mm) was slightly larger than that of pre-adolescents ( $10.90 \pm 7.181$ mm). Females had a slightly higher mean subscapular skinfold ( $11.85 \pm 7.251$  mm) than did males (n=154,  $10.97 \pm 6.602$  mm). According to the 1999-2002 NHANES report, subscapular skinfold

**TABLE 4** Frequency (%) distributions for skinfold thickness measurements.

<b>Triceps skinfold (mm)</b>	Entire Sample	Pre-Adolescent (9-11 years)	Adolescent (13-15 years)	Male	Female
N	303	161	142	155	148
1 = Less than 5	2 (0.7)	0	2 (1.4)	2 (1.3)	0
2 = 5 to 9	58 (19.1)	24 (14.9)	34 (23.9)	45 (29.0)	13 (8.8)
3 = 10 to 14	84 (27.7)	54 (33.5)	30 (21.1)	41 (26.5)	43 (29.1)
4 = 15 to 19	82 (27.1)	43 (26.7)	39 (27.5)	32 (20.6)	50 (33.8)
5 = 20 to 24	41 (13.5)	24 (14.9)	17 (12.0)	22 (14.2)	19 (12.8)
6 = 25 to 29	25 (8.3)	11 (6.8)	14 (9.9)	7 (4.5)	18 (12.2)
7 = 30 to 34	8 (2.6)	3 (1.9)	5 (3.5)	5 (3.2)	3 (2.0)
8 = 35 to 39	2 (0.7)	2 (1.2)	0	0	2 (1.4)
9 = 40 or more	1 (0.3)	0	1 (0.7)	1 (0.6)	0
<b>Subscapular Skinfold (mm)</b>					
N	302	161	141	154	148
1 = Less than 5	12 (4.0)	9 (5.6)	3 (2.1)	8 (5.2)	4 (2.7)
2 = 5 to 9	151 (50.0)	82 (50.9)	69 (48.9)	82 (53.2)	69 (46.6)
3 = 10 to 14	75 (24.8)	38 (23.6)	37 (26.2)	30 (19.5)	45 (30.4)
4 = 15 to 19	24 (7.9)	10 (6.2)	14 (9.9)	16 (10.4)	8 (5.4)
5 = 20 to 24	16 (5.3)	9 (5.6)	7 (5.0)	7 (4.5)	9 (6.1)
6 = 25 to 29	14 (4.6)	9 (5.6)	5 (3.5)	8 (5.2)	6 (4.1)
7 = 30 to 34	9 (3.0)	3 (1.9)	6 (4.3)	3 (1.9)	6 (4.1)
8 = 35 to 39	0	0	0	0	0
9 = 40 or more	1 (0.3)	1 (0.6)	0	0	1 (0.7)

measurements between 13.3 mm and 17.2 mm are reference values for males aged 9 to 11 years and 13 to 15 years, respectively, who are at risk for overweight (91). A measurement of 25.0 mm was the only reference value indicated for subscapular skinfold thickness for overweight males aged 13 years. Subscapular skinfold measurements between 15.4 mm and 17.9 mm and between 20.8 mm and 22.1 mm are reference values for females aged 9 to 11 years and 13 to 15 years, respectively, who are at risk for overweight. A measurement of 30.8 mm was the only reference value indicated for subscapular skinfold thickness for females aged 13 years.

Less than 4% of the children in the entire sample had been diagnosed with allergies (Table 5). Twice as many adolescents had been diagnosed with allergies compared to pre-adolescents. Males and females had similar rates of diagnosed allergies. Almost six times more children in the entire sample have been diagnosed with asthma compared to allergies (Table 5). More adolescent children were diagnosed with asthma compared to pre-adolescent children. Approximately 20% of both males and females were reported to be diagnosed with asthma. More children in the entire sample were reported to be taking prescription medication for allergies than were reported to be diagnosed with allergies (Table 5). Although more adolescent children than pre-adolescent children were diagnosed with allergies, more pre-adolescent children were reported to be taking prescription medication for allergies compared to

**TABLE 5** Frequency (%) distributions of allergy and asthma diagnoses and medication use.

	Entire Sample	Pre-Adolescent (9-11 years)	Adolescent (13-15 years)	Male	Female
<b>Allergies</b>					
N	299	159	140	152	147
0 = No	288 (96.3)	155 (97.5)	133 (95.0)	146 (96.1)	142 (96.1)
1 = Yes	11 (3.7)	4 (2.5)	7 (5.0)	6 (3.9)	5 (3.9)
<b>Allergy RX</b>					
N	299	159	140	152	147
0 = No	274 (91.6)	144 (90.6)	130 (92.9)	141 (92.8)	133 (90.5)
1 = Yes	25 (8.4)	15 (9.4)	10 (7.1)	11 (7.2)	14 (9.5)
<b>Asthma</b>					
N	299	159	140	152	147
0 = No	235 (78.6)	130 (81.8)	105 (75.0)	120 (78.9)	115 (78.2)
1 = Yes	64 (21.4)	29 (18.2)	35 (25.0)	32 (21.1)	32 (21.1)
<b>Asthma RX</b>					
N	299	159	140	152	147
0 = No	277 (92.6)	148 (93.1)	129 (92.1)	141 (92.8)	136 (92.5)
1 = Yes	22 (7.4)	11 (6.9)	11 (7.9)	11 (7.2)	11 (7.5)

adolescent children. Likewise, more females than males were reported to be taking prescription medication for allergies. Fewer children in the entire sample were reported to be taking prescription medication for asthma than for allergies despite a higher diagnosis rate for asthma (Table 5). More adolescent children compared to pre-adolescent children were reported to be taking prescription medication for asthma. Less than 10% of both males and females were reported to be taking prescription medication for asthma.

The mean dietary omega-3 fatty acid intake of the entire sample was  $0.51 \pm 0.366$  grams (Table 6). Both the mean intake of the pre-adolescent sample and the adolescent sample were similar to that of the entire sample. The mean intake of males was higher than that of females. According to intake, less than 5% of the children in the entire sample met the AI for dietary omega-3 fatty acid. A smaller proportion of children with asthma (3.3%) met the omega-3 fatty acid AI compared to children without asthma (4.9%). More pre-adolescent children than adolescent children met the AI for dietary omega-3 fatty acid. A smaller proportion of pre-adolescent children with asthma (3.6%) met the omega-3 fatty acid AI compared to pre-adolescent children without asthma (5.6%). A smaller proportion of adolescent children with asthma (3.0%) met the omega-3 AI compared to adolescent children without asthma (4.0%). More than twice as many females met the AI for dietary omega-3 fatty acid compared to males. None of the males with asthma met the omega-3 fatty acid AI. The proportion of females with asthma (6.5%) who met the omega-3 fatty acid AI was similar to that of females without asthma (6.2%). Only 1% of the children in the entire sample reported

**TABLE 6** Frequency (%) distributions of dietary intake and supplement use.

<b>Omega-3 fatty acid intake (g)</b>	Entire Sample	Pre-Adolescent (9-11 years)	Adolescent (13-15 years)	Male	Female
N	289	153	136	145	144
Mean	0.51	0.51	0.51	0.55	0.48
Standard error	0.366	0.399	0.327	0.382	0.347
<b>Met omega-3 fatty acid AI</b>					
N	289	153	136	145	144
0 = No	276 (95.5)	145 (94.8)	131 (96.3)	141 (97.2)	135 (93.8)
1 = Yes	13 (4.5)	8 (5.2)	5 (3.7)	4 (2.8)	9 (6.3)
<b>Omega-3 fatty acid supplement</b>					
N	303	161	142	155	148
0 = No	300 (99.0)	158 (98.1)	142 (100)	152 (98.1)	148 (100)
1 = Yes	3 (1.0)	3 (1.9)	0	3 (1.9)	0
<b>Vitamin C intake (mg)</b>					
N	289	153	136	145	144
Mean $\pm$ SE	73.67 $\pm$ 53.729	74.77 $\pm$ 52.878	72.44 $\pm$ 54.840	76.06 $\pm$ 55.074	71.27 $\pm$ 54.422
<b>Met vitamin C DRI</b>					
N	289	153	136	145	144
0 = No	137 (47.4)	58 (37.9)	79 (58.1)	71 (49.0)	66 (45.8)
1 = Yes	152 (52.6)	95 (62.1)	57 (44.1)	74 (51.0)	78 (54.2)
<b>Vitamin C supplement</b>					
N	303	161	142	155	148
0 = No	274 (90.4)	144 (89.4)	130 (91.5)	137 (88.4)	137 (92.6)
1 = Yes	29 (9.6)	17 (10.6)	12 (8.5)	18 (11.6)	11 (7.4)
<b>Multi-vitamin/mineral</b>					
N	303	161	142	155	148
0 = No	189 (62.4)	97 (60.2)	92 (64.8)	103 (66.5)	86 (58.1)
1 = Yes	114 (37.6)	64 (39.8)	50 (35.2)	52 (33.5)	62 (41.9)

taking an omega-3 fatty acid supplement (Table 6). Approximately 2% of both the pre-adolescents and males reported taking an omega-3 fatty acid supplement, while none of the adolescent children or females report taking such a supplement. The mean dietary vitamin C intake of the children of the entire sample was  $73.67 \pm 53.729$  mg (Table 6). The mean dietary vitamin C intake of pre-adolescent children was just higher than that of adolescent children. Like dietary omega-3 fatty acid intake, dietary vitamin C intake was higher in males than in females. According to this intake, just over half of the children in the entire sample met the DRI for dietary vitamin C (Table 6). A larger proportion of children with asthma (59.1%) met the vitamin C DRI compared to children without asthma (51.1%). More pre-adolescent children met the DRI for dietary vitamin C compared to adolescent children. A larger proportion of pre-adolescent children with asthma met the vitamin C DRI (67.9%) compared to pre-adolescent children without asthma (61.3%). A larger proportion of adolescent children with asthma (51.5%) met the vitamin C DRI compared to adolescent children without asthma (38.6%). A slightly higher proportion of females than males met the DRI for dietary vitamin C. A larger proportion of males with asthma (60.0%) met the vitamin C DRI compared to males without asthma (49.1%). A larger proportion of females with asthma (58.1%) met the vitamin C DRI compared to females without asthma (53.1%). Few children in the entire sample reported taking a vitamin C supplement (Table 6). More pre-adolescents than adolescents and more males than females reported taking such a supplement. Just over one-third of the children in the entire sample reported taking a multi-vitamin/mineral supplement (Table 6). A larger proportion of pre-adolescent children than adolescent

children reported taking a multi-vitamin/mineral supplement. More females than males also reported taking such a supplement.

### Binary Regression Analysis

Results of binary regression analysis performed on the entire sample (Table 7) revealed that children of ethnicities other than White were more likely to be diagnosed with asthma and were more likely to be using asthma medications than children of White ethnicity. Children who reported the use of vitamin C supplements were more likely to be diagnosed with allergies. Children who were using allergy medications were more likely to have asthma and to be using asthma medications compared to children who were not using allergy medications. Likewise, children who were using asthma medications or who had been diagnosed with asthma were more likely to be using allergy medications than children who were not using asthma medications. Neither vitamin C intake, omega-3 fatty acid intake, weight, BMI, nor any anthropometric measurements were found to be statistically related to asthma diagnosis.

**TABLE 7** Results of binary regression performed on the entire sample.

Dependent Variable	Covariate	Wald	Sig.	Odds Ratio
<b>Asthma</b>	Ethnicity	3.931	P<0.05	1.810
	Allergy RX	90.347	P<0.01	4.737
<b>Allergies</b>	Vitamin C Supplement	7.368	P<0.01	6.011
	Activity	4.573	P<0.05	3.148
	Hard exercise	4.463	P<0.05	0.575
<b>Asthma RX</b>	Ethnicity	7.801	P<0.01	3.515
	Allergy RX	5.653	P<0.05	3.779
<b>Allergy RX</b>	Asthma	13.123	P<0.01	4.737
	Asthma RX	5.653	P<0.05	3.779

The results of binary regression analysis performed on the pre-adolescents (Table 8) demonstrated correlations between asthma diagnosis and weight, BMI, BMI category, subscapular skinfold, and waist circumference. More specifically, children with larger subscapular skinfolds, children with larger waist circumferences, and children who were overweight were all more likely to be diagnosed with asthma compared to their counterparts. Several anthropometric measurements were found to be significantly correlated with the use of asthma medications as well. Children with higher weights, higher BMI's, larger subscapular skinfolds, and larger waist circumferences were more likely to be using asthma medications.

Results of binary logistic regression analysis of the adolescents (Table 9) found that the older children in this age group were less likely to be diagnosed with asthma and also less likely to be using asthma medications. In contrast to the results we saw regarding weight and asthma diagnosis in the previous sample, in this sample, children with higher weights were less likely to be diagnosed with asthma than children with lower weights. The odds of asthma diagnosis were very high for children using allergy medications. Children who were reported to be using allergy medications also had greater odds of being diagnosed with allergies and of using asthma medications. Children who were reported to be using asthma medications were more likely to be using allergy medications compared to children who were not using asthma medications. The odds of using asthma medications were greater for children who were not of White ethnicity. The odds of using asthma medications were also greater for children with asthma whose mothers were overweight. Vitamin C supplementation was associated



**TABLE 8** Results of binary regression performed on pre-adolescents.

Dependent Variable	Covariate	Wald	Sig.	Odds Ratio
<b>Asthma</b>	BMI category	4.893	P<0.05	1.709
	Subscapular skinfold	4.096	P<0.05	1.302
	Waist circumference	4.213	P<0.05	1.452
<b>Asthma RX</b>	Weight	4.602	P<0.05	1.022
	BMI	4.405	P<0.05	1.128
	BMI category	4.705	P<0.05	2.184
	Subscapular skinfold	5.228	P<0.05	1.481
	Waist circumference	4.124	P<0.05	1.683

**TABLE 9** Results of binary regression performed on adolescents.

Dependent Variable	Covariate	Wald	Sig.	Odds Ratio
<b>Asthma</b>	Age	9.505	P<0.01	0.445
	Weight	4.278	P<0.05	0.986
	Allergy RX	11.056	P<0.01	15.259
<b>Allergies</b>	Vitamin C Supplement	7.764	P<0.01	10.333
	Allergy RX	9.227	P<0.01	13.500
<b>Asthma RX</b>	Ethnicity	6.109	P<0.05	5.089
	Age	6.266	P<0.05	0.299
	Allergy RX	5.783	P<0.05	6.536
	Mom BMI category	4.639	P<0.05	2.253
<b>Allergy RX</b>	Allergies	9.227	P<0.01	13.500
	Asthma	11.056	P<0.01	15.259
	Asthma RX	5.783	P<0.05	6.536

with greater odds of allergy diagnosis. Children with an allergy diagnosis were more likely to be using allergy medications compared to children without allergies. Children with an asthma diagnosis were also more likely to be using allergy medications than children who did not have asthma. As in the results of the entire sample, neither BMI nor anthropometric measurements were found to be significantly related to asthma diagnosis.

Tables 10 and Table 11 list the results of binary logistic regression analysis performed on males and females, respectively. As in the pre-adolescent sample, the odds of asthma diagnosis were increased for overweight males. Allergy diagnosis and the use of allergy medications also increased the odds of asthma diagnosis in males. Asthma was not significantly related to any other variables in females. In males, the odds of allergy diagnosis were increased for children who were using vitamin C supplements, who had larger triceps and subscapular skinfolds, who were using asthma medications, and who were reported to be diagnosed with asthma. Neither vitamin C supplementation, subscapular skinfold, the use of asthma medications, nor asthma diagnosis were significantly correlated with the use of allergy medications in females. Females with larger triceps skinfolds were less likely to be diagnosed with allergies than females with smaller triceps skinfolds. Children who were not of White ethnicity and children who were using allergy medications were more likely to be using asthma medications. Neither of these variables was associated with the use of asthma medications in females. Instead, females with asthma whose mothers were overweight were more likely to be using asthma medications. The odds of using allergy medications

were increased by allergy diagnosis in both males and females. In males, these odds were also increased by asthma diagnosis and the use of asthma medications. In none of the samples discussed in this section were dietary intake, multi-vitamin/mineral or omega-3 fatty acids supplementation, guardian, waist/hip ratio, mom's smoking habit, or mom's education significantly related to any of the dependent variables that were analyzed, including asthma and allergy diagnosis and the use of asthma or allergy medications.

**TABLE 10** Results of binary regression performed on males.

Dependent Variable	Covariate	Wald	Sig.	Odds Ratio
<b>Asthma</b>	BMI category	4.173	P<0.05	1.605
	Allergies	5.721	P<0.05	8.429
	Allergy RX	9.935	P<0.01	8.120
<b>Allergies</b>	Vitamin C supplement	6.338	P<0.05	8.733
	Activity	4.730	P<0.05	4.927
	Hard exercise	6.742	P<0.01	0.294
	Triceps skinfold	6.316	P<0.05	1.887
	Subscapular skinfold	4.899	P<0.05	1.716
	Allergy RX	4.744	P<0.05	7.611
<b>Asthma RX</b>	Asthma	5.721	P<0.05	8.429
	Ethnicity	5.836	P<0.05	4.870
<b>Allergy RX</b>	Allergy Rx	5.668	P<0.05	6.234
	Activity	4.760	P<0.05	3.268
	Allergies	4.744	P<0.05	7.611
	Asthma	9.935	P<0.01	8.120
	Asthma RX	5.668	P<0.05	6.234

**TABLE 11** Results of binary regression performed on females.

Dependent Variable	Covariate	Wald	Sig.	Odds Ratio
<b>Allergies</b>	Triceps skinfold	7.191	P<0.01	0.114
	Allergy RX	4.229	P<0.05	7.222
<b>Asthma RX</b>	Mom BMI category	4.567	P<0.05	2.248
<b>Allergy RX</b>	Allergies	4.229	P<0.05	7.222

## CHAPTER V

### DISCUSSION

This chapter will discuss the variable frequency distributions and the outcomes of linear regression analysis and logistic regression analysis in more detail. It will also explain the significance and importance of these outcomes. Following these discussions, this chapter will provide information regarding limitations of this thesis project as well as provide suggestions for future research endeavors.

The result that adolescent children were taller and weighed more than pre-adolescent children was expected. Puberty, the factor separating these two samples is followed by a time of marked growth. In general, older children who have experienced puberty are taller and therefore weigh more than younger children who have not yet experienced puberty. Boys are usually taller than girls, therefore, the results that males were taller and weighed more than females was also expected. BMI, a measure of weight for height, is expected to be larger for taller children, who incidentally weigh more. This is consistent with the results of this analysis. Adolescent children, who were taller and weighed more than pre-adolescent children, had higher BMI's. Likewise, males, who were taller and weighed more than females, also had higher BMI's. Because children at different ages are experiencing different rates of growth and maturation, BMI percentile can determine an individual's level of growth and maturation in comparison to others of the same age. In this study, we found that pre-adolescents tend to have higher BMI percentile scores than adolescent children. This reveals that more pre-adolescent

children than adolescent children in this study had weights for height that exceed those of their peers. A possible explanation for this would be that, compared to their peers, the adolescents in this sample have not yet experienced a growth spurt. More males than females have weights for height that exceed those of their peers. Perhaps more males in this study, compared to other males have experienced a growth spurt, while fewer females in this study, compared to other females have not. Children with a BMI that lies above the 95<sup>th</sup> percentile, and perhaps even above the 85<sup>th</sup> percentile, are of concern because they may be at increased health risk. The finding that just fewer than 20% of children are above the 95<sup>th</sup> percentile is close to the national average of 17%, but slightly lower than average for the state of Texas. Results also showed that more than one-third of the children in this study were above the 85<sup>th</sup> percentile, and possibly at increased health risk. In particular, more males than females and slightly more pre-adolescents than adolescents fell into this category.

Children who were taller and who weighed more were expected to have larger waist and hip circumferences than children who were shorter and who weighed less. Results of this analysis confirm this. Adolescent children had larger waist and hip circumferences than pre-adolescent children, and males had larger waist and hip circumferences than females. Waist/hip ratio has been used by some researchers as an estimate of central adiposity. In order to include this measurement in analyses, waist and hip circumferences of the children in this study were compared. Under normal circumstances, hip circumference is larger than waist circumference. A higher ratio indicates an increasing waist circumference that is approaching the measure of the hip

circumference. In theory, individuals who become overweight accumulate fat in their abdomen, which results in an increasing waist circumference but little change in hip circumference. For this reason, waist/hip ratio has been used as a measure of central adiposity. We found that adolescents had higher waist/hip ratios than pre-adolescents. Likewise, males had higher waist/hip ratios compared to females. Interestingly, this corresponded with the results we discovered earlier that a larger proportion of both pre-adolescents and males were above the 85<sup>th</sup> BMI percentile. This finding supported the use as waist/hip ratio in this study as a possible indicator of central adiposity and suggested that more pre-adolescents and males were overweight. Overall body fat is often estimated through a series of skinfold measurements at specific locations on the body. The triceps and the subscapular area are two areas of the body which are targeted in this process. Analysis of triceps skinfold thickness and subscapular skinfold thickness revealed that pre-adolescents had thicker triceps skinfolds, while adolescents had thicker subscapular skinfolds. Distribution of body fat may differ between pre-adolescent and adolescent children because of differences in growth patterns. Females had both thicker triceps skinfolds and subscapular skinfolds compared to males. Males, in general, tend to have more lean body mass, which may explain why skinfolds were thicker in females. Differing body fat distributions between males and females may also partially explain these results.

Very few children in this study had received a diagnosis of allergies. Allergy prevalence in this study was much lower than the prevalence reported for the U.S (11%) and for the South (15%). Males and females had similar rates of diagnosis, but allergies

seemed to be more prevalent among adolescent children than among pre-adolescent children. One possible explanation for this difference is that older children have more autonomy and are more likely to explore their environment, thereby exposing themselves to more environmental allergens. They, therefore, are more likely than pre-adolescents to experience more allergy symptoms, which may then lead to a higher rate of allergy diagnosis. Many more children in this study were found to be diagnosed with asthma than to be diagnosed with allergies. Approximately 20% of children received a diagnosis of asthma, which is much higher than the average number of children in the U.S. who received an asthma diagnosis just two years ago. It is also higher than the prevalence rate in Texas, reported by Petronella et al. Similar to allergy diagnosis, males and females had similar rates of asthma diagnosis, but adolescents had higher rates of asthma diagnosis than pre-adolescents. This supports the idea that childhood asthma is more commonly allergic asthma, since more adolescents had allergies and asthma. Interestingly, the rate of allergy medication use was much higher than the rate of allergy diagnosis. This may be explained by under-reporting of allergy diagnosis or the misuse of allergy medications. Although more adolescent children had been diagnosed with allergies, more pre-adolescent children were reported to be using allergy medications. Perhaps pre-adolescent children had not received a physician diagnosis of allergies, mothers of pre-adolescents did not report allergy diagnosis to the interviewer, or allergy medications were being misused by pre-adolescents. Or, perhaps, adolescents experienced milder allergy symptoms that did not warrant the use of medications. More females than males were also reported to be using allergy medications despite similar

rates of allergy diagnosis. This could also be due to under-reported allergy diagnosis in females or the misuse of allergy medications by females. Females may have reported more symptoms of allergy and therefore were more likely to use medications, or males may have experienced milder allergy symptoms that did not require the use of medications. The use of asthma medications was less frequently reported than the use of allergy medications, despite a higher rate of asthma diagnosis. If the asthma experienced by the children in this study is allergic asthma, then perhaps the higher rate of allergy medication use reflects the attempt to control the allergy symptoms that trigger asthma. More adolescent children were reported to be using asthma medications, which was expected since more adolescent children were reported to have an asthma diagnosis. A similar rate of asthma medication use in males and females likely reflected the similar rates of asthma diagnoses in these two samples.

The mean dietary omega-3 fatty acid intake and the mean dietary vitamin C intake among the children in this study were both below recommendations for these nutrients. These nutrients provide protective effects against health risks as mentioned in a previous chapter; therefore, dietary intake of these nutrients is of concern. Vitamin C is found abundantly in fresh fruits and vegetables, consequently a low vitamin C intake likely reflects low fruit and vegetable consumption. Despite a higher dietary intake of vitamin C, fewer males are met the vitamin C DRI compared to females. The vitamin C DRI for males is higher than for females, which may explain why fewer males met recommendations. Likewise, more pre-adolescent than adolescent children met the DRI for vitamin C, probably due to lower recommendations. While half of the children in



this study were reported to be meeting the recommended intake for vitamin C, very few children were meeting recommended intake for omega-3 fatty acid. The AI for omega-3 fatty acid intake is lower for females and for pre-adolescents, which likely explains why more of these children met the recommendations for this nutrient compared to their counterparts. Few children in this study reported taking a vitamin C supplement, and even fewer reported taking an omega-3 fatty acid supplement. For both supplements, reports were higher among pre-adolescents compared to adolescents and among males compared to females. It may be that supplements were being used in conjunction with prescription medications to treat medical conditions, which may explain why pre-adolescents, who had a higher rate of allergy medication use, also had a higher rate of supplement use compared to adolescents. Or, perhaps supplements were used as an alternative to prescription medications, which may explain why the rate of allergy medication was lower in males, but the rate of supplement use was higher compared to females. If the low intakes of vitamin C reported here reflect low intakes of fruits and vegetables, it is likely that other vitamins and minerals may be lacking in the diet as well. Like the previously mentioned supplements, the use of a multi-vitamin/mineral supplement was reported to be higher among pre-adolescents compared to adolescents, perhaps reflecting their use in conjunction with prescription medications. In contrast to the previous example of supplement use, more females than males were reported to be taking a multi-vitamin/mineral supplement. Females may be more health-conscious and, therefore, more likely to use a multi-vitamin/mineral supplement.

Logistic regression analysis revealed that the use of allergy medications was positively correlated with asthma diagnosis. It has been suggested that allergic asthma is the most common type of asthma among children, which is supported by these results. BMI category was found to be positively correlated with asthma diagnosis in pre-adolescents and in males. Perhaps pre-adolescence is a vulnerable time during which asthma is adversely affected by overweight. A lower prevalence of overweight among females in this sample may have resulted in no correlation between BMI and asthma diagnosis. Waist circumference and subscapular skinfold were also found to be positively correlated with asthma diagnosis in pre-adolescents. BMI category was related to asthma diagnosis in pre-adolescents perhaps because this group of children had not yet experienced marked growth in height, so increases in weight would increase weight for height ratio. The relationship between asthma and waist circumference and between asthma and subscapular skinfold supports the idea that asthma may be related to adipose tissue. Age and weight appeared to be inversely related to asthma diagnosis in adolescents, suggesting that the younger children in this sample were more likely to be diagnosed with asthma. Perhaps asthma improved as age increases, or perhaps older children perceived their asthma to be better than it actually was. Weight may be a factor as a consequence of age. Younger children may not have experienced peak growth and consequently weigh less than older children.

Vitamin C supplementation was found to be positively correlated with allergy diagnosis among the children in this study, more particularly among adolescents and males. This may be due to the replacement of prescription medications by vitamin C for

the treatment of allergies. Adolescents had higher rates of asthma diagnosis but lower rates of medication use than pre-adolescents. Males had lower rates of allergy medication use compared to females, despite similar rates of allergy diagnosis.

The use of allergy medications was found to be positively correlated with the use of asthma medications. This supports the notion that the children in this study had allergic asthma, and were therefore treating their asthma directly through the use of asthma medications and indirectly through the use of allergy medications. BMI, BMI category and mother's BMI category were also found to be positively correlated with the use of asthma medications. If, in fact, overweight children are more likely to be diagnosed with asthma, then they would consequently be more likely to use asthma medications. If mothers are overweight, then children are more likely to be overweight, and therefore more likely to have asthma and use asthma medications. Age was inversely related to the use of asthma medications in adolescents. As mentioned previously, younger children were more likely to have asthma and as a result were more likely to use asthma medications. Asthma may have improved with age, thereby required less use of asthma medications. Because increasing waist circumference and subscapular skinfold thickness were found to increase the risk of asthma diagnosis, the finding that these factors were also associated with the use of asthma medications is not surprising. Diagnosis with allergies was found to increase the likelihood of allergy medication use, which was expected. Diagnosis with asthma and the use of asthma medications also increased the likelihood of allergy medication use. Again, this is most likely due to the presence of allergic asthma among the children in this study.

Positive correlations were found between ethnicity and asthma and between ethnicity and asthma medication use. This finding suggests that children of ethnicities other than White were more likely to be diagnosed with asthma and to be using asthma medications. This finding is consistent with the research that suggests asthma is more prevalent among minorities, such as Blacks and Hispanics. However, given the small sample size of minorities in this study, it would be difficult to specify which minority population was at higher risk for asthma.

## CHAPTER VI

### SUMMARY

In the present study, overweight children, as determined by BMI category, were at risk for asthma and the use of asthma medications. These children may have experienced more asthma symptoms which led them to seek medical care and therefore to receive an asthma diagnosis. Overweight children were also at risk for the use of allergy medications, which suggests that overweight children may have been taking multiple medications, which is consistent with research. Perhaps this is due to an inflammatory response initiated by adipose tissue, as discussed in an earlier chapter. Likewise children with large waist circumferences were at risk for asthma. Research has shown that waist circumference is associated with greater health risk among adults. More specifically, waist circumferences above 35 inches (88 cm) in women and 40 inches (102 cm) in men increase health risks (40). No such threshold values exist for children, making it difficult to establish that children with very large waist circumferences are at increased health risk, but the findings in this study suggest that they may be. Anthropometric measurements were also found to be positively correlated with asthma diagnosis, suggesting that children with excess body fat may be at risk for asthma. However, without having skinfold measurements from at least two additional sites on the body, it is difficult to determine total body fat composition. This makes it difficult to determine whether these correlations are actually due to body fat percentage or just due to differing fat distributions throughout the body. Children with asthma were

likely to be taking allergy medications despite the lack of an allergy diagnosis, suggesting that these children may have allergic asthma. They may have been prescribed allergy medications by their physicians in an attempt to treat the allergies that caused their asthma. Children who use vitamin C supplements were likely to have allergies, perhaps because these children were aware of their allergies and were using vitamin C to treat their symptoms. In this study, no significant correlations were found between asthma diagnosis and waist/hip ratio, dietary intake, guardian, smoking by the mother, or the mother's level of education, suggesting that these factors may not significantly contribute to asthma.

### **Limitations**

The findings in this study are not without limitations. The number of children who reported asthma and allergies was small, making it difficult to establish many correlations with their diagnoses. This was likely due to the fact that this survey was not designed specifically to obtain information on asthma or allergies. Very few of the many questions in this survey were asked regarding health conditions, and there were no questions regarding asthma symptoms and/or spirometry. This made it difficult to determine asthma severity and control, or to possibly identify undiagnosed asthma. Skin prick tests were not performed, which may have better identified children with allergies. Diagnosis of asthma in the child was defined as the report of asthma by the mother. This may lead to under-reporting or perhaps even over-reporting of medical conditions. Consulting medical records would be preferred but much more time consuming and invasive. On the other hand, physicians may not use the same criteria to diagnose

asthma (17, 33), which could also have affected the number of reported asthma cases in this study. Because this was a survey, there was no follow-up. Information at one point in time was obtained, making it impossible to determine if responses to questions change over time. If more children became overweight, would more develop asthma? As children grow older, do more grow out of their childhood asthma? The nature of this study also makes it difficult to determine a causal relationship between asthma and overweight in children. It is impossible to determine which occurred first. Finally, the regression analyses performed in this study were simple regression tests, which may not have accounted for possible confounding variables.

### **Future Research**

In order to assure that asthma and allergy are well represented, future studies should be designed to target these populations specifically. This will better predict possible risk factors for the development of both conditions. Preferably, these studies should be longitudinal in nature, allowing the establishment of the time of onset of asthma or allergies and their risk factors, such as overweight. Physicians, as well as researchers, should agree upon a definition of asthma and unanimous criteria should be established in order to diagnosis the condition correctly and consistently. Future analyses performed on asthma and its possible risk factors should be more specific, including possible confounding factors. This should be done so in order to eliminate false correlations.

## LITERATURE CITED

1. Lavoie KL, Bacon SL, Labrecque M, Cartier A, Ditto B. Higher BMI is associated with worse asthma control and quality of life but not asthma severity. *Respiratory Medicine*. 2006; 100: 648-657.
2. Akinbami LJ. The state of childhood asthma, United States, 1980-2005. Advance data from vital and health statistics; Hyattsville, MD: National Center for Health Statistics. 2006; 381.
3. Shapiro GG, Stout JW. Childhood asthma in the United States: urban issues. *Pediatric Pulmonology*. 2002; 33: 47-55.
4. U.S. Department of Health and Human Services and U.S. Department of Agriculture. *Dietary Guidelines for Americans, 2005*. 6<sup>th</sup> Edition, Washington, DC: U.S. Government Printing Office, January 2005.
5. National Center for Health Statistics. *Health, United States, 2005 with Chartbook on Trends in the Health of Americans*. Hyattsville, MD: 2005.
6. Lucas SR, Platts-Mills TAE. Paediatric asthma and obesity. *Paediatric Respiratory Reviews*. 2006; 7: 233-238.
7. Gold DR, Damokosh AI, Dockery DW, Berkey CS. Body-mass index as a predictor of incident asthma in a prospective cohort of children. *Pediatric Pulmonology*. 2003; 36: 514-521.
8. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*. 2006; 295(13): 1549-1555.
9. Fiore H, Travis S, Whalen A, Auinger P, Ryan S. Potentially protective factors associated with healthful body mass index in adolescents with obese and nonobese parents: a secondary data analysis of the third national health and nutrition examination survey, 1988-1994. *Journal of the American Dietetic Association*. 2006; 106: 55-64.
10. Romieu I, Mannino DM, Redd SC, McGeehin MA. Dietary intake, physical activity, body mass index, and childhood asthma in the third national health and nutrition survey (NHANES III). *Pediatric Pulmonology*. 2004; 38: 31-42.



11. Nava FV, Gonzalez AHS, Perales GM, Ochoa DL, Gomez MDB, Rodriguez EMV, Rodriguez CFV, Guzman FJB. Associations between family history of allergy, exposure to tobacco smoke, active smoking, obesity, and asthma in adolescents. *Archivos de Bronconeumologia*. 2006; 42(12): 621-626.
12. Raebel MA, Malone DC, Conner DA, Xu S, Porter JA, Lanty FA. Health services use and health care costs of obese and nonobese individuals. *Archives of Internal Medicine*. 2004; 164: 2135-2140.
13. Must A, Anderson SE. Effects of obesity on morbidity in children and adolescents. *Nutrition in Clinical Care*. 2003; 6(1): 4-12.
14. Tavasoli S, Heidarnazhad H, Kazemnejad A, Miri S. Association between asthma severity and obesity in two asthma clinics in Tehran. *Iranian Journal of Allergy, Asthma, and Immunology*. 2005; 4(4): 179-184.
15. Thomson CC, Clark S, Camargo CA Jr. Body mass index and asthma severity among adults presenting to the emergency department. *Chest*. 2003; 124: 795-802.
16. U.S. Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*. 2nd Edition. Washington, DC: U.S. Government Printing Office, November 2000.
17. Joseph CLM, Foxman B, Leickly FE, Peterson E, Ownby D. Prevalence of possible undiagnosed asthma and associated morbidity among urban schoolchildren. *Journal of Pediatrics*. 1996; 129(5): 735-742.
18. Lenney W. The burden of pediatric asthma. *Pediatric Pulmonology, Supplement*. 1997; 15: 13-16.
19. Deane S, Thomson A. Obesity and the pulmonologist. *Archives of Disease in Childhood*. 2006; 91: 188-191.
20. Expert Panel Report 2: Guidelines for the Diagnosis and Management of Asthma. National Institutes of Health. National Heart, Lung, and Blood Institute. 1997; 97-4057. <<http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm>> [Accessed February 2007]
21. Mannino DM, Mott J, Ferdinands JM, Camargo CA Jr, Friedman M, Greves HM, Redd SC. Boys with high body masses have an increased risk of developing asthma: findings from the national longitudinal survey of youth (NLSY). *International Journal of Obesity*. 2006; 30: 6-13.

22. Randolph C, Fraser B. Stressors and concerns in teen asthma. *Current Problems in Pediatrics*. 1999; 29: 82-93.
23. Fuhlbrigge AL, Adams RJ, Guilbert TW, Grant E, Lozano P, Janson SL, Martinez F, Weiss KB, Weiss ST. The burden of asthma in the United States; level and distribution are dependent on interpretation of the national asthma education and prevention program guidelines. *American Journal of Respiratory and Critical Care Medicine*. 2002; 166: 1044-1049.
24. Blaisdell CJ, LoCasale R, Gu A, Weiss, SR. Risk areas for pediatric acute care: asthma differs from upper and lower respiratory illness. *Health & Place*. 2007; 13: 404-416.
25. Ronmark E, Perzanowski M, Platts-Mills T, Lundback B. Incidence rates and risk factors for asthma among school children: a 2-year follow-up report from the obstructive lung disease in northern Sweden (OLIN) studies. *Respiratory Medicine*. 2002; 96: 1006-1013.
26. Chen Y, Dales R, Jiang Y. The association between obesity and asthma is stronger in non-allergic than allergic adults. *Chest*. 2006; 130: 890-895.
27. Kjellman B, Gustafsson PM. Asthma from childhood to adulthood: asthma severity, allergies, sensitization, living conditions, gender influence and social consequences. *Respiratory Medicine*. 2000; 94: 454-465.
28. Leech S. Applied physiology: understanding allergy. *Current Pediatrics*. 2002; 12: 376-381.
29. Lemanske RF, Busse WW. Asthma: factors underlying inception, exacerbation, and disease progression. *Journal of Allergy and Clinical Immunology*. 2006; 117: S456-S461.
30. Sazonov-Kocevar V, Laforest L, Travier N, Yin DD, Van Ganse E. Asthma and allergy medication use and costs among pediatric primary care patients on asthma controller therapy. *Pediatric Allergy and Immunology*. 2006; 17: 620-628.
31. Hong SJ, Lee MS, Lee SY, Ahn KM, Oh JW, Kim KE, Lee JS, Lee HB. High body mass index and dietary pattern are associated with childhood asthma. *Pediatric Pulmonology*. 2006; 41: 1118-1124.
32. Saint-Pierre P, Bourdin A, Chanez P, Daures JP, Godard P. Are overweight asthmatics more difficult to control? *Allergy*. 2006; 61: 79-84.

33. Gilliland FD, Berhane K, Islam T, McConnell R, Gauderman WJ, Gilliland SS, Avol E, Peters JM. Obesity and the risk of newly diagnosed asthma in school-age children. *American Journal of Epidemiology*. 2003; 158: 406-415.
34. Schachter LM, Peat JK, Salome CM. Asthma and atopy in overweight children. *Thorax*. 2003; 58: 1031-1035.
35. U.S. Department of Agriculture, Agricultural Research Service. Food and Nutrient Intakes by Children 1994-96, 1998. ARS Food Surveys Research Group. 1999. <[www.barc.usda.gov/bhnrc/foodsurvey/home.htm](http://www.barc.usda.gov/bhnrc/foodsurvey/home.htm)> [Accessed February 2007]
36. Wong KW. Clinical efficacy of n-3 fatty acid supplementation in patients with asthma. *Journal of the American Dietetic Association*. 2005; 105: 98-105.
37. Sidebotham HJ, Roche WR. Asthma deaths; persistent and preventable mortality. *Histopathology*. 2003; 43: 105-117.
38. Van Gent R, Van Der Ent CK, Rovers MM, Kimpen JLL, Van Essen-Zandvliet LEM, De Meer G. Excessive body weight is associated with additional loss of quality of life in children with asthma. *J Allergy Clin Immunol*. 2007. 119(3): 591-596.
39. Poulain M, Doucet M, Major GC, Drapeau V, Series F, Boulet LP, Tremblay A, Maltais F. The effect of obesity on chronic respiratory disease: pathophysiology and therapeutic strategies. *CMAJ*. 2006; 174(9): 1293-1299
40. Finer N. Medical consequences of obesity. *Medicine*. 2006; 34(12): 510-514.
41. Sood A, Dawson BK, Eid W, Eagleton LE, Henkle JQ, Hopkins-Price P. Obesity is associated with bronchial hyper-responsiveness in women. *Journal of Asthma*. 2005; 42: 847-852.
42. Clark NM, Brown R, Joseph CLM, Anderson EW, Liu M, Valerio M, Gong M. Issues in identifying asthma and estimating prevalence in an urban school population. *Journal of Clinical Epidemiology*. 2002; 55: 870-881.
43. McIntosh A, Davis G, Nayga R Jr., Anding J, Torres C, Kubena K, Perusquia E, Yeley G, You W. Parental time, role strain, and children's fat intake and obesity-related outcomes. Economic Research Service. US Department of Agriculture. Contractor and Cooperator Report Number 19. 2006. <<http://www.ers.usda.gov/Publications/ccr19/>> [Accessed March 2007]

44. Hoelscher DM, Day RS, Lee ES, Frankowski RF, Kelder SH, Ward JL, Scheurer ME. Measuring the prevalence of overweight in Texas schoolchildren. *American Journal of Public Health*. 2004; 94: 1002-1008.
45. U.S. Department of Health and Human Services. National Center for Health Statistics. Healthy People 2010. DATA2010...the Healthy People 2010 Database. <<http://www.cdc.gov/nchs/hphome.htm>> [Accessed February 2007]
46. Kilpelainen M, Terho EO, Helenius H, Koskenvuo M. Body mass index and physical activity in relation to asthma and atopic diseases in young adults. *Respiratory Medicine*. 2006; 100: 1518-1525.
47. McLachlan CR, Poulton R, Car G, Cowan J, Filsell S, Greene JM, Taylor DR, Welch D, Williamson A, Sears MR, Hancox RJ. Adiposity, asthma, and airway inflammation. *Journal of Allergy and Clinical Immunology*. 2007; 119(3): 634-639.
48. Ford ES. The epidemiology of obesity and asthma. *Journal of Allergy and Clinical Immunology*. 2005; 115: 897-909.
49. Kronander UN, Falkenberg M, Zetterstrom O. Prevalence and incidence of asthma related to waist circumference and BMI in a Swedish community sample. *Respiratory Medicine*. 2004; 98: 1108-1116.
50. CDC Growth Charts: United States. US Department of Health and Human Services. Centers for Disease Control and Prevention. National Center for Health Statistics. Hyattsville, MD 2007. Accessed February 2007. Available at <http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/charts.htm>
51. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics*. 1998; 101: 518-525.
52. Finkelstein EA, Fiebelkorn IC, Wang G. National medical spending attributable to overweight and obesity: how much, and who's paying? *Health Affairs Web Exclusive*. 2003; W3: 219-226.
53. Dixon AE, Shade DM, Cohen RI, Skloot GS, Holbrook JT, Smith LJ, Lima JJ, Allayee H, Irvin CG, Wise RA. Effect of obesity on clinical presentation and response to treatment in asthma. *Journal of Asthma*. 2006; 43: 553-558.
54. Schuh S, Johnson D, Stephens D, Callahan S, Canny G. Hospitalization patterns in severe acute asthma in children. *Pediatric Pulmonology*. 1997; 23: 184-192.

55. Petronella SA, Bricker SK, Perrotta D, Brown C, Brooks EG. Addressing asthma in Texas: development of a school-based asthma surveillance program for Texas elementary schools. *Journal of School Health*. 2006; 76(6): 227-234.
56. Joseph CLM, Havstad S, Anderson EW, Brown R, Johnson CC, Clark N. Effect of asthma intervention on children with undiagnosed asthma. *Journal of Pediatrics*. 2005; 146: 96-104.
57. Nathell L, Larsson K, Jensen I. Determinants of undiagnosed asthma. *Allergy*. 2002; 57: 687-693.
58. Quinn K, Shalowitz MU, Berry CA, Mijanovich T, Wolf RL. Racial and ethnic disparities in diagnosed and possible undiagnosed asthma among public-school children in Chicago. *American Journal of Public Health*. 2006; 96: 1599-1603.
59. Nolte H, Nepper-Christensen S, Backer V. Unawareness and undertreatment of asthma and allergic rhinitis in a general population. *Respiratory Medicine*. 2006; 100: 354-362.
60. Van Gent R, Van Essen LEM, Rovers MM, Kimpen JLL, Van Der Ent CK, De Meer G. Quality of life in children with undiagnosed and diagnosed asthma. *European Journal of Pediatrics*. Published online 2006.
61. Diamant Z, Boot JD, Virchow JC. Summing up 100 years of asthma. *Respiratory Medicine*. 2007; 101(3): 378-388.
62. Martinez FD. Asthma treatment and asthma prevention: a tale of 2 parallel pathways. *Journal of Allergy and Clinical Immunology*. 2007; 119: 30-33.
63. Mitchell DK, Adams SK, Murdock KK. Associations among risk factors, individual resources, and indices of school-related asthma morbidity in urban, school-aged children: a pilot study. *Journal of School Health*. 2005; 75(10): 375-383.
64. Debley JS, Redding GJ, Critchlow CW. Impact of adolescence and gender on asthma hospitalization: a population-based birth cohort study. *Pediatric Pulmonology*. 2004; 38: 443-450.
65. Bratton DL, Price M, Gavin L, Glenn K, Brenner M, Gelfand EW, Klinnert MD. Impact of a multidisciplinary day program on disease and healthcare costs in children and adolescents with severe asthma: a two-year follow up study. *Pediatric Pulmonology*. 2001; 31: 177-189.

66. Bloom B, Dey AN, Freeman G. Summary health statistics for U.S. children: national health interview survey, 2005. National Center for Health Statistics. *Vital Health Stat.* 2006; 10(231).
67. Baker JC, Ayres JG. Diet and asthma. *Respiratory Medicine.* 2000; 94: 925-934.
68. Ball SD, Kertesz D, Moyer-Mileur LJ. Dietary supplement use is prevalent among children with a chronic illness. *Journal of the American Dietetic Association.* 2005; 105: 78-84.
69. United States Department of Agriculture. MyPyramid.gov. <<http://www.mypyramid.gov/index.html>> [Accessed June 2007]
70. Flaherman V, Rutherford GW. A meta-analysis of the effect of high weight on asthma. *Archives of Disease in Childhood.* 2006; 91: 334-339.
71. Ulrik CS, Backer V, Dirksen A, Pedersen M, Koch C. Extrinsic and intrinsic asthma from childhood to adult age: a 10-yr follow-up. *Respiratory Medicine.* 1995; 89: 547-554.
72. Bacharier LB, Strunk RC, Mauger D, White D, Lemanske RF Jr, Sorkness CA. Classifying asthma severity in children; mismatch between symptoms, medication use, and lung function. *American Journal of Respiratory and Critical Care Medicine.* 2004; 170: 426-432.
73. Tollefsen E, Langhammer A, Bjermer L, Kurtze N, Holmen TL. Adolescents with wheeze have increased risk of additional health problems. The Young-HUNT study, Norway. *Preventive Medicine.* 2007; 44: 178-182.
74. Barreto ML, Cunha SS, Alcantara-Neves N, Carvalho LP, Cruz AA, Stein RT, Genser B, Cooper PJ, Rodrigues LC. Risk factors and immunological pathways for asthma and other allergic diseases in children: background and methodology of a longitudinal study in a large urban center in northeastern Brazil (Salvador-SCAALA study). *BMC Pulmonary Medicine.* 2006; 6(15).
75. Gibson PG, Ram FSF, Powell H. Asthma education. *Respiratory Medicine.* 2003; 97: 1036-1044.
76. Schatz M, Sorkness CA, Li JT, Marcus P, Murray JJ, Nathan RA, Kosinski M, Pendergraft TB, Jhingran P. Asthma control test: reliability, validity, and responsiveness in patients not previously followed by asthma specialists. *Journal of Allergy and Clinical Immunology.* 2006; 117: 549-556.

77. Bisgaard H, Szeffler S. Understanding mild persistent asthma in children: the next frontier. *Journal of Allergy and Clinical Immunology*. 2005; 115: 708-713.
78. Hallstrand TS, Curtis JR, Aitken ML, Sullivan SD. Quality of life in adolescents with mild asthma. *Pediatric Pulmonology*. 2003; 36: 536-543.
79. King MT, Hall J, Lancsar E, Fiebig D, Hossain I, Louviere J, Reddel H, Jenkins CR. Patient preferences for managing asthma: results from a discrete choice experiment. *Health Economics*. Published online 2006.
80. Sullivan SD, Rasouliyan L, Russo PA, Kamath T, Chipps BE. Extent, patterns, and burden of uncontrolled disease in severe or difficult-to-treat asthma. *Allergy*. 2007; 62: 126-133.
81. Kanter RK, Moran JR. Recent trends in pediatric hospitalization in New York state. *Journal of Pediatrics*. 2006; 148: 637-641.
82. Frank TL, Cropper JA, Hazell ML, Frank PI, Hannaford PC. Childhood asthma: healthcare resource utilization in those with and without a diagnosis of the condition. *Respiratory Medicine*. 2004; 98: 454-463.
83. Eneli IU, Karmaus WK, Davis S, Kuehr J. Airway hyperresponsiveness and body mass index: the child health and environment cohort study in Hesse, Germany. *Pediatric Pulmonology*. 2006; 41: 530-537.
84. Sherry B. Food behaviors and other strategies to prevent and treat pediatric overweight. *International Journal of Obesity*. 2005; 29: S116-S126.
85. Lin BH, Morrison RM. Higher fruit consumption linked with lower body mass index. *Food Reviews*. 2002; 25(3): 28-32.
86. Belamarich PF, Luder E, Kattan M, Mitchell H, Islam S, Lynn H, Crain EF. Do obese inner-city children with asthma have more symptoms than nonobese children with asthma? *Pediatrics*. 2000; 106: 1436-1441.
87. Hess MA. Portion sizes of popular foods. University of Wisconsin-Stout: American Dietetic Association and Center for Nutrition Education; 1997.
88. ESHA Research. The food processor SQL nutrition analysis and fitness software. Salem, OR: ESHA Research; 2000-2003.
89. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics; 1988.

90. Food and Nutrition Information Center. National Agricultural Library. United States Department of Agriculture. <<http://fnic.nal.usda.gov>> [Accessed February 2007]
91. McDowell MA, Fryar CD, Hirsch R, Ogden CL. Anthropometric reference data of children and adults: U.S. population, 1999-2002. Advance data from vital and health statistics; Hyattsville, MD: National Center for Health Statistics. 2005.



**VITA**

Name: Jennifer Leah Furman

Address: Intercollegiate Faculty of Nutrition, 218 Kleberg Center, 2253 TAMU,  
College Station, TX 77843-2253

Email Address: [jlgtierrez@tamu.edu](mailto:jlgtierrez@tamu.edu)

Education: B.A., Biology, Texas A&M University, 2000  
M.S., Nutrition, Texas A&M University, 2007